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RESEARCH ARTICLE

Affective factors predicting binge eating disorder: The role of emotion regulation, impulsivity, and cognitive distortions

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ABSTRACT

Objective: Binge eating disorder (BED) is characterized by a marked loss of control over food intake, leading to episodes of excessive eating that are often followed by feelings of guilt, sadness, and disgust. These negative emotional experiences may be influenced by underlying cognitive distortions. Moreover, difficulties in utilizing effective coping mechanisms are associated with emotion dysregulation. This study aimed to investigate the complex interrelationships among BED symptoms, emotion dysregulation, impulsivity, and cognitive distortions within an integrated model.

Method: A total of 460 healthy female participants were recruited for this study. Data were collected using the Binge Eating Disorder Scale, the Difficulties in Emotion Regulation Scale-Short Form, the Barratt Impulsivity Scale-Short Form, and the Cognitive Distortions Questionnaire.

Results: The findings indicate that higher levels of emotion dysregulation, cognitive distortions, and impulsivity significantly predict BED symptoms. Furthermore, emotion dysregulation and impulsivity were found to mediate the relationship between cognitive distortions and BED symptoms. This suggests a compounded effect, wherein elevated emotion dysregulation, impulsivity, and cognitive distortions are associated with an increase in BED symptoms.

Conclusion: The integrated model proposed in this study offers new insights into the diagnostic and therapeutic approaches for BED symptoms, highlighting the importance of addressing the interconnected psychological factors involved.

Keywords: Binge eating disorder, emotion dysregulation, impulsivity, cognitive distortions

INTRODUCTION

Eating disorders are complex psychiatric conditions influenced by a combination of psychological, biological, and sociocultural factors. When left untreated, these disorders can lead to serious health complications and even mortality. They are marked by significant disturbances in eating behaviors (1). According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), eating disorders include Anorexia Nervosa (AN), Pica, Avoidant/Restrictive Food Intake Disorder, Rumination Disorder, Bulimia Nervosa (BN), and Binge Eating Disorder (BED) (1).

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BED is characterized by recurrent episodes of uncontrollable overeating within a short period. Individuals with BED symptoms often eat alone to avoid feelings of shame and subsequently experience emotions such as disgust, guilt, or sadness. Research suggests that individuals with eating disorders often struggle with coping mechanisms for managing negative emotions, leading to emotion dysregulation (2, 3). Difficulty regulating emotions can disrupt eating behaviors (3). Binge eating may serve as a temporary means of alleviating psychological distress (2), and is frequently associated with rumination, a transdiagnostic cognitive process involving repetitive, self-critical thoughts. Empirical evidence suggests that negative emotions can trigger binge eating episodes, as individuals with BED often struggle with deficits in emotion regulation and resort to binge eating as a maladaptive coping mechanism (4). While this supports the emotion regulation model of BED, findings remain heterogeneous. Some studies indicate that impulsivity may further intensify this mechanism by impairing inhibitory control and decision-making under distress (4, 5). Recent research also suggests that emotion dysregulation plays a central role in the psychopathology of BED, particularly through its interaction with impulsivity and cognitive distortions (5). Individuals with BED frequently exhibit impaired emotional awareness and difficulty controlling impulsive behaviors, which may reinforce maladaptive eating patterns. These findings highlight that binge eating is not merely a behavioral issue but is deeply connected to underlying difficulties in emotion regulation and cognitive processing. Furthermore, this tendency may impair problem-solving abilities and contribute to ambivalence in individuals exhibiting BED symptoms (6).

In addition to emotion dysregulation, dysfunctional thought patterns, known as core beliefs, may exacerbate BED symptoms. These core beliefs, often formed early in life, shape an individual's self-concept and worldview, and they frequently go unchallenged. Beyond influencing emotional responses, cognitive distortions can also affect impulsive tendencies. Such maladaptive thought patterns may heighten impulsivity by encouraging short-term, reward-seeking behaviors and weakening self-control (7, 8). As a result, individuals with BED symptoms often struggle to delay gratification, leading to impulsive food consumption in response to distressing emotions (9, 10). While previous research has primarily linked cognitive distortions to BED symptoms through emotion dysregulation, the role of impulsivity in this relationship remains underexplored. Emerging studies suggest that cognitive distortions may contribute to cognitive rigidity, reducing one's ability to evaluate consequences and increasing vulnerability to impulsive actions (7, 11). Specifically, catastrophizing and dichotomous thinking can intensify distress and prompt urgency-driven decisions, thereby reinforcing impulsive eating behaviors (10). Furthermore, impaired cognitive flexibility, often observed in individuals with high levels of cognitive distortions, may reduce their ability to regulate impulsive urges, leading to recurrent binge eating episodes (7, 8).

Research has linked emotional deprivation, social isolation, and inadequate self-control with BED (12). Waller identified that social isolation, feelings of vulnerability, inadequacy, high standards, and justification beliefs significantly contribute to BED (13). These maladaptive thoughts may underlie the emotion dysregulation commonly observed in the disorder. Additionally, impulsivity, frequently driven by emotion dysregulation, is a key factor in BED (9). As a multidimensional personality trait, impulsivity influences both the development and maintenance of BED due to its association with uncontrolled, excessive food intake (14). However, the specific facets of impulsivity most affected in BED are still unclear. The reward system, central to impulsivity, is believed to play a significant role in BED (14). Among the various subtypes of impulsivity, motor impulsivity has been particularly implicated in BED, as it reflects difficulties in inhibiting automatic responses, such as uncontrolled eating behaviors. Previous studies suggest that individuals with BED exhibit heightened reward sensitivity and impaired impulse control, especially in behavioral tasks measuring motor impulsivity (15). In line with these findings, the current study focuses on motor impulsivity as a key predictor of BED symptoms. Meta-analytic evidence further indicates that individuals with obesity, both with and without BED, demonstrate deficits in inhibitory control, particularly in tasks assessing response inhibition (16). These self-regulatory impairments have been associated with dysfunction in the prefrontal cortex, a brain region critical for inhibitory control and decision-making (16). However, the extent to which these deficits are specific to BED, rather than obesity more broadly, remains an open question, warranting further investigation. Gray (17) highlighted the connection between the brain's reward center, which regulates the behavioral inhibition and activation system, and impulsivity in

BED. Voon's study (18) emphasized the importance of emotion and behavior regulation processes, along with impulsivity, in hedonic eating and BED.

While existing literature has explored eating disorders in relation to stress, thinking styles, emotion regulation processes, and impulsivity (2, 6, 14, 19), there is a lack of research examining BED within the context of core beliefs alongside these variables. Given the complexity of BED, a comprehensive model is essential. Research indicates that BED is significantly more prevalent among women than men, with studies reporting that approximately 60-70% of individuals diagnosed with BED symptoms are female (5, 20). This gender disparity may be attributed to hormonal influences, sociocultural pressures, and differences in emotional regulation strategies (21–23). Women are more likely to engage in binge eating as a maladaptive coping mechanism for emotional distress, whereas men with BED symptoms often exhibit higher levels of externalizing behaviors, such as substance use (24). In line with these findings, the current study focuses exclusively on women to minimize potential confounding variables related to gender differences in BED symptomatology.

Therefore, the primary objective of this study is to examine the mechanisms through which cognitive distortions contribute to BED symptoms, focusing on the mediating roles of emotion dysregulation and impulsivity. While previous research has documented the role of emotion dysregulation in BED symptoms, the contribution of impulsivity as a parallel mediator remains less explored. Therefore, this study integrates emotion regulation difficulties and impulsive tendencies into a comprehensive model to clarify their distinct yet interconnected roles in BED symptoms (Fig. 1). Given that both emotion dysregulation and impulsivity contribute to the development and maintenance of BED, this study aims to examine their simultaneous mediating roles in the relationship between cognitive distortions and BED symptoms. Furthermore, BED has been consistently associated with higher Body Mass Index (BMI), with evidence suggesting a bidirectional relationship between the two conditions (25, 26). This relationship indicates that while elevated BMI may contribute to the onset or worsening of BED symptoms, binge eating behaviors also promote excessive weight gain over time (26). One potential explanation for this link involves metabolic and hormonal dysregulation, particularly alterations in ghrelin and leptin levels, which may reinforce compulsive eating behaviors and impair appetite regulation (27). Additionally, heightened reward sensitivity and impaired inhibitory control in individuals with BED may lead to a preference for highly palatable, calorie-dense foods, further contributing to weight gain and obesity (14). These findings underscore the importance of considering BMI in the context of BED, particularly in relation to cognitive distortions, impulsivity, and difficulties in emotion regulation. By incorporating these variables into a comprehensive model, this research seeks to clarify how distorted cognitions contribute to binge eating through both difficulties in emotion regulation and increased impulsivity. Specifically, impulsivity may serve as a key pathway through which cognitive distortions lead to BED symptoms. Distorted thinking patterns, such as catastrophizing and dichotomous thinking, can impair self-regulation, heighten sensitivity to immediate rewards, and weaken response inhibition, making individuals more prone to impulsive eating behaviors (7, 28). While emotion dysregulation contributes to binge eating by intensifying negative affect, impulsivity may accelerate the transition from distress to action, prompting individuals to engage in binge eating without considering long-term consequences (9). This distinction highlights the mediating roles of these variables in the development of BED. Accordingly, this study aims to examine the distinct yet interconnected roles of emotion dysregulation and impulsivity in shaping BED symptoms. By analyzing these variables, the study seeks to identify key predictors of BED symptoms and provide insights into the roles of emotion regulation, reward systems, decision-making, impulsivity, and core beliefs. It proposes a novel, integrated model of BED symptoms, elucidating the interplay between core beliefs, emotion dysregulation, and impulsivity in driving binge eating behavior. Based on this framework, we hypothesize that higher levels of cognitive distortions will be positively associated

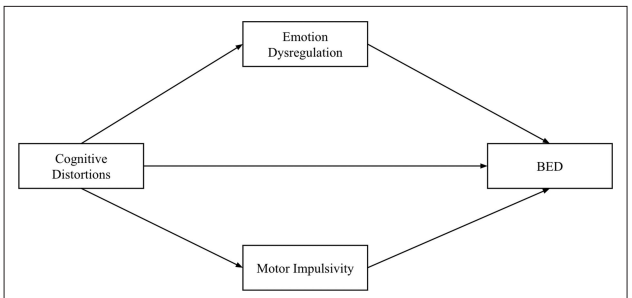

Figure 1. Research model of hypothesized relationships.

BED: Binge eating disorder.

Table 1: Demographic characteristics of the study sample

Variables	Frequency	%	M	SD
Age			25.21	6.34
BMI			22.40	3.77
Education level				
Primary school	1	0.2		
Middle school	4	0.9		
High school	157	34.1		
Bachelor's degree	221	48		
Master's degree	65	14.1		
Ph.D. degree	12	2.6		
Employment status				
Employed	161	35		
Unemployed	299	65		
Exercise frequency*			2.62	2.4
Do you ever lose control while eating?				
Yes	256	55.7		
No	204	44.3		

*: Participants were asked how many times they exercised per week (including light physical activities such as walking). M: Mean; SD: Standard deviation.

with BED symptoms. Furthermore, we expect emotion dysregulation to mediate the relationship between cognitive distortions and BED symptoms, while impulsivity also serves as a mediator. Lastly, we propose that higher BMI will be associated with increased BED symptoms, potentially influencing the severity of binge eating episodes.

METHODS

Sample

The study included 460 female participants from various provinces of Turkiye. Participation was voluntary. Inclusion criteria required participants to be over 18 years of age, not undergoing psychological treatment, not taking psychological treatment, and having no current alcohol or nicotine dependence. Additionally, participants were excluded if they had any current or past psychiatric diagnoses (e.g., depressive or anxiety disorders), a history of severe head injury or concussion, or any history of psychological treatment. Recruitment was conducted via an invitation flyer outlining the eligibility requirements. Participants' Body Mass Index ranged from 15.82 to 40.53 (M=22.40; SD=3.77), and their ages ranged from 18 to 57 years (M=25.21; SD=6.34). The demographic characteristics of the participants are presented in Table 1.

Data Collection Tools

Sociodemographic Information Form

This form, developed by the researchers, collected information on participants' age, weight, height, education level, income, occupation, psychiatric/psychological history, and dietary and exercise habits.

Binge Eating Disorder Scale (BEDS)

Developed by Gormally et al. (29) and adapted into Turkish by Tosyali and Harma (30), this 16-item scale measures the severity of binge eating, including both cognitive and behavioral dimensions. Items are rated on a 4-point Likert scale, with higher scores reflecting greater binge eating severity. Each response option is assigned a weight from 0 to 3, with higher values indicating increased binge eating severity. Confirmatory factor analysis (CFA) was conducted to assess the scale's construct validity. The results indicated an acceptable model fit: χ^2 (103)=188.656, p<0.001, Comparative Fit Index (CFI)=0.911, Root Mean Square Error of Approximation (RMSEA)=0.053, Standardized Root Mean Square Residual (SRMR)=0.048. Factor loadings ranged from 0.30 (Item 1) to 0.65 (Item 10). The scale has previously demonstrated good reliability (α=0.83) and showed high internal consistency in the current study (α=0.92).

Difficulties in Emotion Regulation Scale-Short Form (DERS-16)

Originally developed by Gratz and Roemer (31) and adapted into Turkish by Yigit and Guzey Yigit (32), this 16-item scale assesses difficulties in emotion regulation across five sub-dimensions: clarity, goals, impulse, strategies, and non-acceptance. Items are rated on a 5-point Likert scale ranging from 1 (almost never) to 5 (almost always), with higher scores indicating greater difficulties in emotion regulation. CFA was conducted to evaluate the construct validity of the scale. The results indicated a good model fit: χ^2 (94, N=316)=258.250, χ^2/df=2.75, CFI=0.94, Goodness of Fit Index (GFI)=0.91, Tucker-Lewis Index (TLI)=0.93, RMSEA=0.07. The internal consistency coefficient for the overall DERS-16 was reported as 0.92, further supporting its reliability. In the current study, the scale demonstrated excellent reliability, with a Cronbach's alpha of 0.94. To assess predictive validity, previous research examined correlations between DERS-16 scores and measures of psychological distress using the subscales of the Brief Symptom Inventory (BSI). The results indicated strong, positive correlations between all BSI subscales and DERS-16 scores, with correlation coefficients ranging from 0.28 to 0.69, confirming the scale's ability to effectively predict psychological distress.

Barratt Impulsiveness Scale-11 Short Form (BIS-11-SF)

Originally developed by Barratt (33) and revised in 1995, this scale measures impulsivity. The Turkish version, adapted by Tamam et al. (34), consists of 15 items across three subscales: attention, motor impulsivity, and non-planning. Items are rated on a 4-point Likert scale (1=rarely/never, 2=sometimes, 3=often, 4=almost always/always), with higher scores indicating greater impulsivity. BIS-11-SF scores demonstrated strong convergent validity, showing a high correlation with the original BIS-11 (r=0.94, p<0.001) and with the remaining items (r=0.65, p<0.001). The correlation between the residual items and the full BIS-11 was also high (r=0.87, p<0.001). Internal consistency, as measured by Cronbach's alpha, was reported at 0.82 for the total scale, with subscale values ranging from 0.64 to 0.80. In the current study, the internal consistency of the BIS-11-SF was found to be acceptable, with a Cronbach's alpha of 0.75. The scale underwent exploratory factor analysis to assess its validity, and its correlations with the Frontal Systems Behavior Scale were also examined.

Cognitive Distortions Scale (CDS)

Developed by Briere (35) and adapted into Turkish by Batmaz et al. (36), the Cognitive Distortions Scale measures the frequency and severity of 15 cognitive distortions, such as dichotomous thinking, fortune-telling, and magnification/minimization. The scale uses a 4-point frequency scale (i.e., never, occasionally, most of the time, almost always) and a 4-point intensity scale (i.e., not at all, a little, much, very much). Frequency and intensity scores are combined into a 4×4 matrix, with each cell assigned a score from 0 to 5, allowing for the calculation of frequency, intensity, and total (composite) scores. The total score, ranging from 0 to 75, is obtained by summing all item scores. Batmaz et al. (36) found that the CDS total score correlated highly with its subscales (r=0.95 for frequency, r=0.96 for intensity), confirming the scale's internal consistency. The scale also demonstrated strong concurrent validity, as the CD-Quest total score was significantly correlated with the CDS total score (r=0.75, p<0.001) and with related constructs such as mood severity and negatively biased thinking. Discriminant validity analyses further supported the measure's effectiveness in distinguishing between individuals with high and low levels of cognitive distortions. The authors also reported excellent internal consistency for the scale (Cronbach's alpha=0.93). In the current study, only the total score was computed, and the internal consistency of the CDS was high (Cronbach's alpha=0.90), indicating excellent reliability.

The descriptive statistics for the measurement tools are presented in Table 2.

Procedure

Ethical approval was obtained from the Dokuz Eylul University Scientific Research and Publication Ethics Committee (Approval Date: 03.01.2023, Number: 13-467244). Participants were recruited through online announcements shared via social media and the university's information-sharing system. Data collection was conducted online, with participants completing a battery of self-report measures after providing informed consent.

Statistical Analysis

Data were analyzed using SPSS 29 and PROCESS v3.5 (37). The initial data screening involved assessing missing values, identifying outliers, evaluating normality, and testing homogeneity of variance. No missing values were detected, so no imputation was necessary. Outliers were assessed using standardized Z-scores, and none exceeded the conventional cutoffs

Table 2: Descriptive statistics of study variables

	Min	Max	M	SD
BEDS	12	58	24.02	9.14
Clarity	2	10	4.56	1.69
Goals	3	15	9.12	3.02
Impulse	3	15	6.33	2.93
Strategies	5	25	11.42	4.71
Non-acceptance	3	15	6.25	2.95
DERS Total	16	75	37.68	12.90
Non-planning	7	17	13.16	1.86
Motor impulsivity	6	20	10.44	1.93
Attention	7	20	11.83	1.72
BIS-11-SF Total	26	49	35.43	3.06
CDS	0	67	16.77	13.25
Dichotomous thinking	0	5	1.09	1.37
Fortune telling	0	5	1.11	1.33
Discounting the positive	0	5	1.04	1.41
Emotional reasoning	0	5	1.27	1.40
Labeling	0	5	1.13	1.43
Magnification/minimization	0	5	1.01	1.41
Selective abstraction	0	5	1.15	1.44
Mind reading	0	5	1.48	1.47
Overgeneralization	0	5	0.87	1.23
Personalization	0	5	0.88	1.32
Should statements	0	5	1.40	1.47
Jumping to conclusions	0	5	0.89	1.24
Blaming others or oneself	0	5	1.00	1.42
"What if...?" statements	0	5	1.35	1.48
Unfair comparisons	0	5	1.10	1.36

BED: Binge Eating Disorder Scale total score; DERS: Difficulties in Emotion Regulation Scale-Short Form total score; BIS-11-SF: Barratt Impulsiveness Scale-11 Short Form total score; CDS: Cognitive Distortions Scale total score.

(Z>±3.29), indicating no extreme values required exclusion. Normality was evaluated through skewness and kurtosis values, all within the acceptable range of ±1.5, and confirmed with standardized Z-scores (38). Homogeneity of variance was tested using Levene's test, which indicated that group variances were equal.

A one-way analysis of variance (ANOVA) was conducted to examine whether participants' BED symptoms differed by BMI category. Since homogeneity of variances was confirmed, Tukey's Honestly Significant Difference (HSD) test was used for post-hoc comparisons. Additionally, an independent samples t-test was performed to investigate differences in BED symptoms based on whether participants reported a loss of control over eating. Relationships between variables were analyzed using Pearson correlation.

Mediation analyses were conducted using PROCESS v3.5 to determine whether emotion regulation difficulties and impulsivity mediated the relationship between cognitive distortions and BED symptoms. Preliminary correlation analyses indicated that only motor impulsivity was significantly associated with BED symptoms ($r=250$, $p<0.001$), while attentional ($r=0.005$, $p>0.05$) and non-planning impulsivity ($r=0.140$, $p>0.05$) were not. As a result, motor impulsivity was selected as the primary impulsivity variable for this study. Before conducting the mediation analysis, assumptions of linearity, multicollinearity, and homoscedasticity were tested. Variance inflation factors (VIFs) were examined to ensure that multicollinearity was not a concern. Homoscedasticity was assessed through inspection of residual plots. Mediation models were tested using bootstrapping with bias-corrected confidence intervals (5,000 resamples) to obtain robust estimates of indirect effects. The absence of collinearity further validated the mediation paths.

RESULTS

To examine how BED symptoms vary across BMI categories, participants' BMI values were classified into four groups based on World Health Organization (WHO) standards: underweight, normal weight, overweight, and obese. A one-way ANOVA revealed significant differences in BED symptoms across these groups, $F(3, 457)=36.1$, $p<0.000$, $\eta^2=0.29$. A Tukey HSD post-hoc test was conducted to further explore group differences. Underweight individuals (N=247, M=17.39, SD=4.54) exhibited significantly lower BED symptom levels compared to those in the normal weight (N=165, M=22.75, SD=8.10), overweight (N=35, M=29.38, SD=9.22), and obese (N=13, M=35.00, SD=11.27) categories ($p<0.001$). Normal weight individuals also had significantly higher BED symptoms than underweight participants but significantly lower BED symptoms than obese individuals ($p<0.001$). Finally, the Tukey HSD results indicated that obese individuals showed significantly higher BED symptoms than both underweight and normal weight individuals ($p<0.001$).

An independent samples t-test was conducted to examine differences in BED symptoms based on participants' reported loss of control over eating behavior. Results showed that individuals who reported a loss of control (N=257, M=28.75, SD=9.27) had significantly higher BED symptoms than those

Table 3: Pearson correlations among study variables

	BMI	Emotion dysregulation	Motor impulsivity	Cognitive distortions
BED symptoms	0.651*	0.412*	0.250*	0.409*
BMI	–	0.160*	0.172*	0.008
Emotion dysregulation		–	0.285*	0.584*
Motor impulsivity			–	0.280*

*: p<0.001. BED: Binge Eating Disorder; BMI: Body Mass Index.

who did not (N=203, M=18.09, SD=4.17), t (458)=-15.23, p<0.000, d=0.15.

Pearson correlation analysis was conducted to examine the relationships among the study variables (Table 3). BED symptoms were positively correlated with BMI (r=0.651, p<0.001), emotion dysregulation (r=0.412, p<0.001), motor impulsivity (r=0.250, p<0.001), and cognitive distortions (r=0.409, p<0.001). BMI was also positively correlated with emotion dysregulation (r=0.160, p<0.001) and motor impulsivity (r=0.172, p<0.001), but not with cognitive distortions (p>0.05). Significant positive correlations were also found between emotion dysregulation and motor impulsivity (r=0.285, p<0.001), as well as between emotion dysregulation and cognitive distortions (r=0.584, p<0.001). Additionally, motor impulsivity was positively correlated with cognitive distortions (r=0.280, p<0.001).

A parallel mediation model was tested to examine whether emotion dysregulation and motor impulsivity mediate the relationship between cognitive distortions and BED symptoms. The model is presented in Figure 2. Analysis revealed that cognitive distortions significantly predicted emotional dysregulation (b=0.60, SE=0.04, t=15.41, p<0.001) and motor impulsivity (b=0.28, SE=0.04, t=6.24, p<0.001). Cognitive distortions also directly predicted BED symptoms (b=0.15, SE=0.04, t=4.13, p<0.001). Furthermore, both emotion dysregulation (b=0.21, SE=0.04, t=5.64, p<0.001) and motor impulsivity (b=0.52, SE=0.21, t=2.54, p=0.011) were significant predictors of BED symptoms.

The total effect of cognitive distortions on BED symptoms was significant (b=0.30, SE=0.03, t=9.58, p<0.001, 95% confidence interval (CI) [0.24, 0.36]). When the mediators were included in the model, the direct effect remained significant but was reduced (b=0.15, SE=0.04, t=4.13, p<0.001, 95% CI [0.08, 0.23]), indicating partial mediation. The total indirect effect was also significant (b=0.15, SE=0.03, 95% CI [0.09, 0.21]), suggesting that cognitive distortions influenced BED symptoms through both emotion dysregulation and motor impulsivity. Specifically, emotion dysregulation mediated the relationship (b=0.12, SE=0.03, 95% CI [0.07, 0.18]), as did motor impulsivity (b=0.02, SE=0.01, 95% CI [0.003, 0.05]). These findings indicate that cognitive distortions contribute to BED symptoms both directly and indirectly via emotion dysregulation and motor impulsivity. However, emotion dysregulation accounted for a larger portion of the indirect effect, highlighting its stronger mediating role.

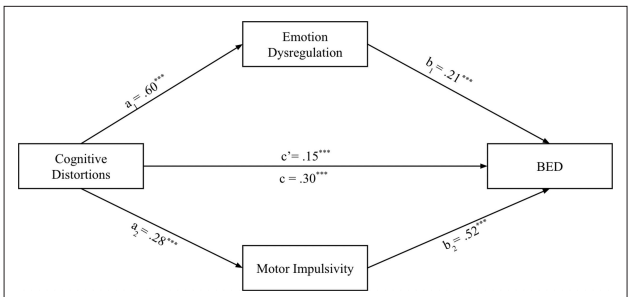

Figure 2. Parallel mediation model examining the roles of emotion dysregulation and motor impulsivity in the relationship between cognitive distortions and BED symptoms.

BED: Binge eating disorder.

Additionally, a separate mediation analysis was conducted to examine whether emotion dysregulation alone mediated the relationship between cognitive distortions and BED symptoms. The results showed that cognitive distortions significantly predicted emotion dysregulation (b=0.60, SE=0.04, t=15.41, p<0.001, 95% CI [0.52, 0.67]). Cognitive distortions also significantly predicted BED symptoms (b=0.17, SE=0.04, t=4.52, p<0.001, 95% CI [0.09, 0.24]), and emotion dysregulation remained a significant predictor in the model (b=0.22, SE=0.04, t=6.08, p<0.001, 95% CI [0.15, 0.29]).

The total effect of cognitive distortions on BED symptoms was significant (b=0.30, SE=0.03, t=9.58, p<0.001, 95% CI [0.24, 0.36]). When emotion dysregulation was included as a mediator, the direct effect of cognitive distortions on BED symptoms was reduced but remained significant (b=0.17, SE=0.04,

t=4.52, p<0.001, 95% CI [0.09, 0.24]), indicating partial mediation. The indirect effect of cognitive distortions on BED symptoms via emotion dysregulation was also statistically significant (b=0.13, SE=0.03, 95% CI [0.08, 0.19]), confirming that emotion dysregulation serves as a mediator in this relationship.

A separate mediation analysis was conducted to examine whether motor impulsivity mediated the relationship between cognitive distortions and BED symptoms. The model showed that cognitive distortions significantly predicted motor impulsivity (b=0.28, SE=0.04, t=6.24, p<0.001, 95% CI [0.03, 0.06]). The overall model predicting BED symptoms was also significant: cognitive distortions significantly predicted BED symptoms (b=0.27, SE=0.03, t=8.36, p<0.001, 95% CI [0.21, 0.33]), and motor impulsivity emerged as a significant predictor as well (b=0.70, SE=0.21, t=3.35, p<0.001, 95% CI [0.29, 1.11]).

The total effect of cognitive distortions on BED symptoms was significant (b=0.30, SE=0.03, t=9.58, p<0.001, 95% CI [0.24, 0.36]). When motor impulsivity was included as a mediator, the direct effect of cognitive distortions on BED symptoms remained significant but slightly decreased (b=0.27, SE=0.03, t=8.36, p<0.001, 95% CI [0.21, 0.33]), indicating partial mediation. The indirect effect of cognitive distortions on BED symptoms via motor impulsivity was also statistically significant (b=0.03, SE=0.01, 95% CI [0.01, 0.06]), confirming that motor impulsivity serves as a mediator in this relationship.

DISCUSSION

The primary objective of this study was to identify predictors of BED symptoms within a comprehensive model. The results indicate that higher BMI values—particularly in the overweight and obese categories—are associated with increased BED symptoms, suggesting that elevated BMI is a risk factor for BED. This finding aligns with previous research linking BED with overweight and obesity (39). The bidirectional relationship between BED and BMI suggests that a higher BMI may contribute to the onset or exacerbation of BED symptoms, while binge eating behaviors can also lead to further weight gain (39, 40). Additionally, women with high BMI and BED symptoms have been shown to experience elevated levels of ghrelin, an appetite-regulating hormone, particularly under stress (41). This finding suggests that stress is a significant risk factor for BED, influencing decision-making mechanisms and being closely associated with elevated BMI.

Another key finding is that loss of control over eating is a core feature of BED, with individuals reporting this loss exhibiting higher levels of BED symptoms (1). This supports the notion that a pronounced loss of control increases the risk for both the onset and persistence of BED (42).

The parallel mediation analysis revealed that emotion dysregulation, impulsivity, and cognitive distortions are significant predictors of BED symptoms. This integrated approach is novel, as previous studies have typically examined these factors in isolation rather than within a unified model. For instance, prior research has linked BED to impulsivity and emotion dysregulation; however, this study is the first to simultaneously examine their interrelations within the framework of cognitive distortions (43, 44). This comprehensive perspective offers a more nuanced understanding of how cognitive distortions interact with psychological mechanisms to drive BED symptoms. The findings indicate that these variables influence BED both directly and indirectly. Specifically, individuals with high levels of cognitive distortions are more likely to experience emotion dysregulation and impulsivity, which, in turn, exacerbate BED symptoms. This supports existing literature linking BED to difficulties in managing negative emotions and higher levels of emotion dysregulation (10). These difficulties often include challenges in identifying emotions and a tendency to interpret ambiguous situations negatively (10).

Research has shown that cognitive distortions, such as "all-or-nothing" thinking, overgeneralization, and catastrophizing, are associated with increased emotion dysregulation (45, 46). These distortions are also linked to body dissatisfaction, weight fluctuations, regret, and shame following episodes of overeating in individuals with eating disorders, further exacerbating difficulties in emotion regulation (45).

Consistent with the present findings, Dingemans et al. (44) reported that individuals with BED exhibit more negative affect and greater emotion dysregulation than healthy controls. This heightened dysregulation is likely influenced by underlying cognitive distortions. Although their study, like similar research, focused on clinical populations, our findings extend these observations to a non-clinical sample. Indeed, our results suggest that cognitive distortions not only correlate with BED symptoms but also exert their influence through emotion dysregulation and impulsivity. This indicates that while emotion dysregulation plays a central role in

BED, its effects may be partially driven by underlying cognitive distortions—an area that has not been fully explored in previous research. Notably, clinical studies have also reported that higher levels of emotion dysregulation are associated with increased cognitive distortions (47). Given that our sample consists of individuals from the general population rather than a clinical BED group, these findings suggest that cognitive distortions and emotion dysregulation may play significant roles even at subclinical levels of BED. Addressing cognitive distortions is critical in the treatment of BED, as they are associated with poorer treatment outcomes (47). Therefore, both emotion dysregulation and cognitive distortions likely serve as mediating factors in the development, maintenance, and treatment outcomes of BED.

The study also found that impulsivity mediates the relationship between cognitive distortions and BED symptoms. Individuals with high levels of cognitive distortions often exhibit increased motor impulsivity, which, in turn, heightens BED symptoms. Impulsive behaviors, characterized by a loss of control, can make it especially difficult to resist binge eating episodes (48).

Cognitive distortions related to body image, weight, and food are significant contributors to the risk of binge eating episodes and the negative emotions associated with eating-related psychopathology. This study empirically supports previous theoretical assumptions by demonstrating that cognitive distortions significantly contribute to BED symptoms. The mediating role of impulsivity suggests that individuals with cognitive distortions may engage in impulsive eating behaviors as a maladaptive coping strategy. Furthermore, the mediating role of emotion dysregulation confirms that cognitive distortions can intensify emotional distress, which in turn exacerbates BED symptoms. These findings validate prior assumptions that distorted cognitive patterns—such as dichotomous thinking, overgeneralization, and catastrophizing—serve as a psychological foundation for BED-related behaviors (43). Volery et al. (43) found that individuals with BED exhibited higher levels of cognitive distortions compared to both obese and healthy control groups. These distortions, often related to body image, weight, and food, have been consistently reported across multiple studies. Previous research also suggests that individuals with high levels of impulsivity are more prone to cognitive distortions such as "all-or-nothing" thinking, overgeneralization, and catastrophizing, which are commonly observed in various eating disorders, including BED (47). The findings of the current study support the hypothesis that impulsive behaviors, emotion dysregulation, and cognitive distortions are significant predictors of BED symptoms. The results provide strong empirical support for the proposed model. Specifically, they confirm that both emotion dysregulation and impulsivity mediate the relationship between cognitive distortions and BED symptoms, indicating that these psychological mechanisms function in parallel rather than sequentially. This distinction is critical, as it suggests that interventions targeting either emotion regulation or impulsivity may reduce BED symptoms by addressing the underlying cognitive distortions.

Limitations and Future Directions

Although the present findings offer new insights into the diagnosis and treatment of BED, several limitations warrant consideration. First, while the mediation analyses revealed significant indirect effects of emotion dysregulation and impulsivity, the persistence of a direct effect suggests that cognitive distortions alone do not fully explain BED symptoms. This implies that other factors—such as genetic predispositions, early-life experiences, and environmental stressors—may also contribute. Future research should explore a broader range of psychological and biological variables to better elucidate the complex mechanisms underlying BED symptoms.

Second, this study focused exclusively on female participants. While this enhances the relevance of the findings for women, it limits generalizability to males, who may exhibit different patterns of BED symptomatology. Third, the study relied entirely on self-report measures, which introduces the potential for response bias. Specifically, motor impulsivity was assessed using a self-report scale, which may lead to subjective interpretations of impulsive behavior. Although self-report tools are widely used in impulsivity research, they may not fully capture behavioral aspects of impulsivity, which are often better evaluated through reaction time-based tasks (e.g., Go/No-Go, Stop-Signal Task) or clinical interviews. Future research should consider incorporating such objective measures to provide a more comprehensive understanding of the role of impulsivity in BED symptoms.

Another limitation is the absence of a standardized screening tool to exclude participants with other eating disorders. Although the study aimed to examine BED symptoms in a non-clinical sample, the inclusion of structured diagnostic assessments or validated screening instruments for additional eating disorders would have strengthened the methodology by reducing potential confounding variables.

Despite these limitations, the study's integrated model offers a novel perspective on BED symptoms that can inform intervention, diagnosis, and treatment. Given the strong association between cognitive distortions and BED symptoms, cognitive therapies—such as Cognitive Behavioral Therapy (CBT) and schema therapy—may be particularly effective. Previous research has shown that CBT reduces binge eating episodes by targeting maladaptive thought patterns and enhancing emotion regulation strategies (44, 49). Similarly, schema-focused interventions have demonstrated promise in addressing core maladaptive beliefs among individuals with persistent BED symptoms (13). Future studies should explore the comparative efficacy of these cognitive therapies in treating BED and investigate whether modifying cognitive distortions also improves impulsivity and emotion regulation, ultimately leading to a reduction in BED severity.

CONCLUSION

The present study found that higher BMI and loss of control over eating behavior are associated with increased BED symptoms, reinforcing prior evidence that elevated weight status and impaired self-regulation are key contributors to binge eating. Additionally, emotion dysregulation, impulsivity, and cognitive distortions were all significantly correlated with BED symptoms. Importantly, the integrated model proposed in this study underscores the mediating roles of emotion dysregulation and impulsivity in the relationship between cognitive distortions and BED symptoms. This offers a novel perspective for both diagnosis and treatment. By targeting these interconnected psychological factors, future interventions may more effectively address the cognitive, emotional, and behavioral underpinnings of BED, ultimately leading to improved clinical outcomes.

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