The Role of Binge Eating in a Sequential Mediation Model of Stress, Emotional Eating, and BMI

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Article

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Abstract

Background/Objectives: Chronic stress contributes to obesity through maladaptive eating behaviors, including emotional eating (eating due to negative emotions) and binge eating (consuming large amounts of food with a loss of control). A theoretical model suggests that emotional eating can escalate to binge eating along a severity continuum, but this sequential pathway from stress to higher body mass index (BMI) has remained empirically untested. This study therefore examined a serial mediation model in which perceived stress predicts BMI sequentially through emotional eating and then binge eating. **Methods:** In this cross-sectional study, 272 Korean adults completed the Perceived Stress Scale, the Dutch Eating Behavior Questionnaire (emotional eating subscale), and the Binge Eating Scale. The serial mediation model was tested using PROCESS macro model 6, with age, gender, and education as covariates. **Results:** The serial mediation pathway (stress → emotional eating → binge eating → BMI) was statistically significant (Indirect effect B = 0.071, 95% CI [0.041, 0.112]). A separate simple mediation path through binge eating alone was also significant (B = 0.056, 95% CI [0.018, 0.102]), whereas the path through emotional eating alone was not significant. The total indirect effect (B = 0.108, 95% CI [0.052, 0.172]) was significant, indicating that the influence of stress on BMI was fully mediated by the eating behaviors modeled. **Conclusions:** This study provides the first empirical evidence supporting a sequential pathway from stress to elevated BMI via the progression from emotional to binge eating. The findings support the overeating continuum model and highlight binge eating as a pivotal mediator. This behavioral progression suggests that emotional and binge eating are distinct stages, offering crucial opportunities for tailored prevention and intervention.

Keywords: stress; emotional eating; binge eating; obesity; mediation; overeating continuum

1. Introduction

Chronic stress contributes to obesity through multiple interconnected eating behavior changes [1]. Stress activates the hypothalamic-pituitary-adrenal (HPA) axis, leading to elevated cortisol levels that stimulate hunger and increase preference for energy-dense "comfort foods" high in fat, sugar, and calories [1–3]. These hormonal changes are accompanied by alterations in appetite-regulating hormones, including increased ghrelin secretion and reduced sensitivity to leptin and insulin, which collectively promote hyperphagia and disrupt normal satiety signaling [1,2,4]. Meta-analytic evidence from 54 studies (N=119,820) demonstrates that stress systematically increases intake of unhealthy, energy-dense foods (Hedges' g = 0.116) while significantly decreasing consumption of nutritious options (Hedges' g = -0.111) [5]. Importantly, consuming highly palatable foods provides temporary relief by suppressing stress responses and activating reward systems through negative feedback mechanisms that dampen HPA axis activity [1,6]. Stress also enhances brain reward pathways, particularly sensitizing the nucleus accumbens and increasing dopamine-mediated food seeking, while simultaneously impairing executive function and self-regulatory capacity [1]. This process creates a reinforcing cycle in which stress-induced eating becomes a learned coping strategy, with repeated consumption of comfort foods strengthening these maladaptive behavioral patterns [2,6]. Therefore, it is crucial to understand how stress-related changes lead to distinct maladaptive

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eating behaviors—especially emotional eating and binge eating—that differ in their psychological mechanisms, clinical significance, and metabolic consequences.

Among the maladaptive eating patterns, emotional eating and binge eating are recognized as critical pathways linking stress to obesity. Emotional eating is characterized by consuming food in response to emotional states rather than physiological hunger, serving as a coping mechanism to regulate negative affect such as stress, anxiety, or depression [7,8]. In contrast, binge eating involves consuming objectively large amounts of food within a discrete time period (e.g., in 2 hours) accompanied by a subjective sense of loss of control over eating [9]. While emotional eating typically involves frequent episodes of modest food intake to cope with distress, binge eating reflects a more severe and dysregulated pattern of intake, often followed by intense guilt or shame [9]. Importantly, binge eating is not only more behaviorally extreme but also constitutes a core diagnostic symptom of eating disorders such as binge eating disorder (BED). These behaviors differ not only in their severity and expression but also in their neurobiological underpinnings: emotional eating is primarily associated with limbic system activation for emotion regulation, whereas binge eating is linked to prefrontal disinhibition and impaired executive control. [1,9].

Emotional eating serves as a well-established mediator of the relationship between perceived stress and obesity-related outcomes [8]. For instance, in a large-scale cross-sectional study of U.S. adults, stress-related eating was found to fully mediate the link between stressful life events and key obesity markers like waist circumference and BMI [10]. This finding is consistent across different populations, with a study of multinational university students similarly demonstrating that emotional eating mediated the pathway from perceived stress to higher BMI [11]. The mediating role of emotional eating is also evident in high-stress populations, such as U.S. Army Soldiers, where it connected perceived stress to both increased BMI and body composition failure [12]. Furthermore, this mechanism appears to influence not just weight but also dietary choices, as emotional eating partially mediated the relationship between perceived stress and poorer dietary quality in a study of Hispanic/Latino adolescents [13].

Binge eating, likewise, has been identified as another critical pathway in the stress–obesity link, with some evidence suggesting it may be a more potent mediator than emotional eating. For example, Chao and colleagues reported that although both behaviors were linked to stress, only binge eating showed a significant relationship with increased waist circumference in a sample of adult women. [14]. The robustness of this pathway is strengthened by longitudinal evidence; a study of sexual-minority women demonstrated that binge eating mediated the relationship between baseline post-traumatic stress symptoms and higher BMI two years later [15]. The clinical significance of this link is further underscored in behavioral weight loss intervention studies, where eating disorder pathology mediated the effect of stress on weight loss specifically within the subgroup of patients with regular binge eating [16]. Finally, mechanistic studies using moderated mediation have begun to clarify how this occurs, showing that stress is associated with increased likelihood of binge eating through maladaptive coping strategies [17].

Despite their distinctions, a growing body of theoretical and empirical evidence suggests that emotional eating and binge eating may lie on a single severity continuum, wherein emotional eating can progress to clinically significant binge eating [9,18]. This progression is theoretically grounded in Davis's 'overeating continuum model' [18], which posits that overeating behaviors initially used to 'self-medicate' negative affect—a core functional definition of emotional eating—can escalate in severity and compulsiveness toward full-blown binge eating. This theoretical model is bolstered by converging lines of empirical evidence. For instance, a longitudinal study with adolescent girls has shown that baseline emotional eating significantly predicts the onset of binge-eating symptoms over a two-year period (hazard ratio ≈ 1.8) [19]. This progression is also supported by a cross-sectional study of overweight adults, which found that emotional eating scores increase progressively from non-binge eaters to individuals with full Binge Eating Disorder (BED) [20]. Furthermore, mechanistic insight from a study using ecological momentary assessment demonstrates that a failure to down-regulate negative affect after an emotional eating episode can create a self-reinforcing loop,

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precipitating full binge episodes. [21]. Finally, mediation studies in adults with trauma exposure have confirmed that emotional eating is a key pathway linking post-traumatic stress to binge eating [22]. Collectively, this evidence aligns with the overeating continuum model and points to a potential sequential pathway from stress to obesity, where emotional eating develops into binge eating.

Prior research has established that both emotional eating and binge eating are independent mediators in the stress-obesity relationship. However, the sequential pathway suggested by the 'overeating continuum model'—wherein emotional eating escalates into binge eating—has yet to be empirically tested within a single, integrated framework. To address this critical gap, this study tested a serial mediation model hypothesizing that perceived stress increases BMI sequentially, first through emotional eating and then through binge eating. The present cross-sectional analysis tests a statistical model representing this hypothesized pathway, providing initial evidence. The findings are expected to help clarify the distinct roles of emotional and binge eating, potentially informing the development of more precise intervention targets.

2. Materials and Methods

2.1. Participants

This study was approved by the Institutional Review Boards of Pusan National University (PNU IRB 2023-18-HR, PNU IRB 2024-148-HR), and all participants provided informed consent in the present study. Participants were recruited from the Korean adult population aged 19 to 59 years. Pregnant individuals were excluded due to potential effects on body weight and dietary behaviors. Recruitment mostly occurred online through advertisements posted on university internet boards and popular mobile and messaging apps, while offline recruitment was limited to poster placements on local university campuses.

Demographic information including gender, age, years of education, height, and weight was collected via self-report. Of the 289 individuals initially screened, 17 participants with a BMI below 18.5 kg/m^2 were excluded from the analysis. The final analytic sample thus comprised 272 individuals, consisting of 96 normal-weight, 39 pre-obese, and 137 obese individuals according to the diagnostic criteria in the Clinical Practice Guidelines of the Korean Society for the Study of Obesity [23]. Among these 272 participants, 115 were male (42.3%). The age distribution included 156 participants aged 29 years or younger (57.4%), 108 participants in their 30s (39.7%), and 8 participants in their 40s (2.9%). Additional demographic information is presented in Table 1.

2.2. Clinical Questionnaires

2.2.1. Perceived Stress

Perceived stress levels were measured using the 10-item Perceived Stress Scale (PSS) [24], which employs a 5-point Likert-type response format ranging from 0 to 4. Higher total scores indicate greater perceived stress. The Korean validation of the PSS yielded satisfactory internal consistency, with Cronbach's alpha coefficients of .82 [25], and the present study estimated Cronbach's alpha as .85.

2.2.2. Emotional Eating

Emotional eating was assessed with the Dutch Eating Behavior Questionnaire (DEBQ), which comprises three subscales: emotional eating, external eating, and restrained eating [26]. Responses were recorded on a 5-point Likert-type scale from 1 ('never') to 5 ('very often'), with higher scores reflecting a greater tendency toward each eating behavior. This study focused solely on the emotional eating subscale, which consists of 13 items. The Korean version of the DEBQ emotional eating subscale showed good internal consistency, with Cronbach's alpha values of .92 in prior research [27] and .93 in this study.

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2.2.3. Binge Eating

Binge eating severity was evaluated using the Binge Eating Scale (BES), a widely employed 16-item self-report instrument [28]. Items are scored on either a 0–2 or 0–3 scale depending on the item. Total scores are interpreted as follows: less than 18 indicates no binge eating symptoms; 18 to 26 indicates moderate binge eating; and 27 or above indicates severe binge eating behavior. The Korean version of the BES demonstrated good internal consistency, with Cronbach's alpha of .84 in previous studies [29] and .90 in the present study.

2.3. Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics version 29.0. escriptive statistics (means and standard deviations) and Pearson correlations were calculated for all study variables in the full sample (see Table 1). Gender was coded as a binary variable (0 = female, 1 = male) for correlation analysis.

A serial mediation model was tested using Model 6 of the PROCESS macro version 4.2 for IBM SPSS Statistics [30]. Perceived stress (PSS) was entered as the independent variable (X); emotional eating (DEBQ) and binge eating (BES) were specified as mediators (M1 and M2); and BMI (kg/m²) was the dependent variable (Y). Gender (1 = male, 0 = female), chronological age (years), and years of education were included as covariates in the model. Indirect effects were estimated using 10,000 bias-corrected bootstrap resamples, and 95% percentile bootstrap confidence intervals were interpreted; an effect was considered statistically significant if its confidence interval did not include zero. Unstandardized regression coefficients (B), standard errors (SE), confidence intervals, and standardized regression coefficients (β) were reported. All analyses were conducted using the full sample (N = 272).

3. Results

3.1. Descriptive Statistics

The study analyzed 272 participants, including 137 individuals classified as obese. Table 2 presents the descriptive statistics and correlations for all study variables in the full sample (N = 272). Bivariate Pearson correlation analyses showed that BMI was positively correlated with emotional eating ($r = 0.182$, $p < 0.01$), binge eating ($r = 0.383$, $p < 0.001$), and age ($r = 0.214$, $p < 0.001$). Perceived stress was positively correlated with both emotional eating ($r = 0.426$, $p < 0.001$) and binge eating ($r = 0.397$, $p < 0.001$). Emotional eating and binge eating were also strongly positively correlated ($r = 0.621$, $p < 0.001$). Male gender was associated with higher perceived stress, emotional eating, and binge eating (all $p < 0.01$).

Table 1. Descriptive statistics in study variables (N = 272).

		Pearson correlation r						
Variables	**Mean (SD) or n (%)**	1	2	3	4	5	6	7
1. Gender (female=0, male=1)	115 (42.3%) [a]	-						
2. Age	28.75 (5.85)	-.035	-					
3. Education	15.43 (2.52)	.015	.277**	-				
4. BMI	25.39 (4.83)	-.077	.214**	-.025	-			
5. Perceived stress (PSS)	17.75 (6.13)	.183**	-.015	-.021	.098	-		
6. Emotional eating (DEBQ)	33.39 (6.21)	.254**	.107	.004	.182**	.426**	-	
7. Binge eating (BES)	13.40 (8.50)	.253**	.143*	-.058	.383**	.397**	.621**	-

[a] Number and percentage of male participants were provided for gender. *: $p < .05$, **: $p < .01$ (two-tailed test).

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3.2. Mediation Analysis

A serial multiple mediation analysis using PROCESS macro Model 6 was performed to examine whether emotional eating and binge eating sequentially mediate the effect of perceived stress on BMI, controlling for age, years of education, and gender. Regression coefficients are presented in Figure 1 and Table 2. Perceived stress was positively associated with emotional eating (B = 0.743, SE = 0.103, p < .001), and emotional eating significantly predicted binge eating (B = 0.379, SE = 0.039, p < .001). Perceived stress also had a direct positive effect on binge eating (B = 0.219, SE = 0.071, p = .002). In the final model predicting BMI, only binge eating showed a significant positive association with BMI (B = 0.253, SE = 0.041, p < .001). The effects of perceived stress (B = -0.016, p = .738) and emotional eating (B = -0.026, p = .393) on BMI were not significant, indicating full mediation.

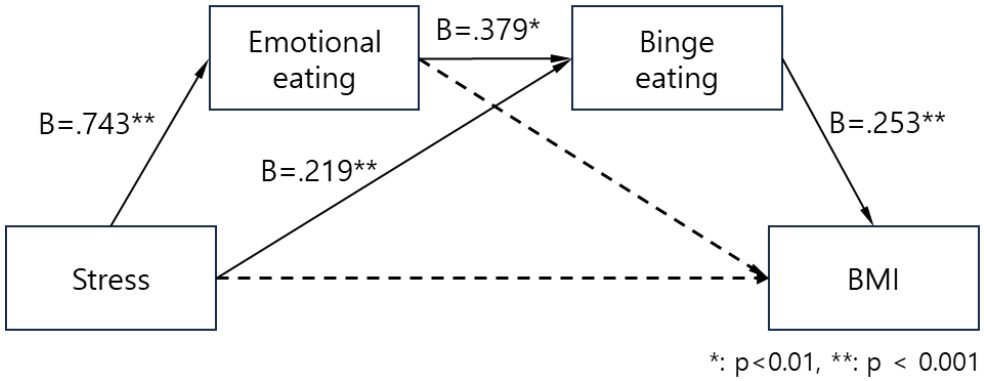

Figure 1. Regression coefficients in the serial mediation model (PROCESS model 6). Only significant coefficients in each path were depicted. Dashed lines represent non-significant paths.

Table 2. Regression coefficients for serial mediation model (PROCESS model 6).

Outcome	Predictor	B	SE	β	t	p-value	95% CI of B
Emotional eating	Perceived stress	.743	.103	.393	7.192	<.001	[.540, .946]
	Age	.251	.111	.127	2.270	.024	[.033, .470]
	Education	-.121	.257	-.026	-.470	.639	[-.626, .385]
	Gender: Male	4.378	1.281	.187	3.418	.001	[1.856, 6.899]
Binge eating	Perceived stress	.219	.071	.158	3.079	.002	[.079, .360]
	Emotional eating	.379	.039	.517	9.811	<.001	[.303, .455]
	Age	.172	.071	.119	2.439	.015	[.033, .311]
	Education	-.308	.162	-.091	-1.898	.059	[-.628, .011]
	Gender: Male	1.688	.826	.098	2.042	.042	[.061, 3.315]
Body Mass Index	Perceived stress	-.016	.049	-.021	-.335	.738	[-.112, .080]
	Emotional eating	-.026	.030	-.062	-.855	.393	[-.086, .034]
	Binge eating	.253	.041	.445	6.136	.000	[.172, .334]
	Age	.135	.048	.163	2.800	.005	[.040, .229]
	Education	-.080	.110	-.042	-.724	.470	[-.296, .137]
	Gender: Male	-1.597	.560	-.164	-2.850	.005	[-2.700, -.494]

The total effect of perceived stress on BMI was marginally significant (B = 0.091, SE = 0.047, p = .055). However, the direct effect became non-significant after inclusion of the mediators (B = -0.016, SE = 0.049, p = .738), indicating the presence of indirect effects.

Bootstrapping with 10,000 samples revealed a statistically significant total indirect effect of perceived stress on BMI (B = 0.108, Boot SE = 0.031, 95% CI [0.052, 0.172]; β = 0.137), as shown in Table 4. Among the three indirect pathways, the serial mediation path through emotional eating and binge eating was significant (B = 0.071, Boot SE = 0.018, 95% CI [0.041, 0.112]; β = 0.091), and the simple mediation path through binge eating alone (Stress → Binge eating → BMI) was also significant (B =

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0.056, Boot SE = 0.022, 95% CI [0.018, 0.102]; β = 0.071). The mediation path through emotional eating alone (Stress → Emotional eating → BMI) was not significant (B = -0.019, Boot SE = 0.023, 95% CI [-0.068, 0.025]; β = -0.024). These results indicate that perceived stress indirectly contributes to increased BMI via its effects on binge eating, both directly and through emotional eating.

Table 3. Indirect effects of perceived stress on BMI via emotional and binge eating (Bootstrp = 10,000).

Indirect Path	Effect (B)	Boot SE	Boot LL	Boot UL
Perceived stress → Emotional eating → BMI	-.019	.023	-.068	.025
Perceived stress → Binge eating → BMI	.056	.022	.018	.102
Perceived stress → Emotional eating → Binge eating → BMI	.071	.018	.041	.112
Total Indirect Effect	.108	.031	.052	.172

4. Discussion

4.1. Main Findings and Theoretical Implications

The present study offers the first empirical test of a serial mediation model linking perceived stress to BMI through a sequential pathway involving emotional eating and binge eating. The findings highlight two key patterns: (1) a significant serial mediation pathway (Perceived stress → Emotional eating → Binge eating → BMI) that supports the overeating continuum hypothesis; and (2) an additional direct pathway from stress through binge eating alone to BMI. Notably, emotional eating did not independently mediate BMI when controlling for binge eating, suggesting that its influence on weight may primarily operate through progression to more severe dysregulated eating behaviors. This pattern is consistent with theoretical models positing emotional eating as an earlier, less severe stage on a continuum of problematic eating behaviors escalating toward clinically significant binge eating episodes [9,18].

These findings provide important empirical support for Davis's overeating continuum model [18], which conceptualizes eating behaviors along a spectrum of severity—from occasional overeating to compulsive binge eating. The significant serial mediation observed here indicates that emotional eating may serve as a developmental precursor to binge eating. This progression aligns with affect regulation models, where repeated use of food to manage negative emotions can lead to more severe loss-of-control episodes over time. The present results extend previous longitudinal findings by Stice et al. [19], who demonstrated that emotional eating prospectively predicted binge eating onset, by providing direct statistical evidence within a mediation framework. Furthermore, our findings complement cross-sectional observations by Ricca et al. [20], which demonstrate stepped increases in emotional eating scores from non-binge eaters to subthreshold and full BED groups, illustrating how this progression may contribute to weight-related outcomes.

These findings provide crucial empirical support for Davis's overeating continuum model [18], which conceptualizes eating behaviors as existing along a severity spectrum from occasional overeating to compulsive binge eating. The significant serial mediation pathway observed here suggests that emotional eating may indeed serve as a developmental precursor to binge eating, as theoretically proposed. This progression is consistent with affect regulation models of eating pathology, where repeated use of food to manage negative emotions may lead to more severe loss-of-control episodes over time. The current results extend previous longitudinal findings by Stice et al. [19], who demonstrated that emotional eating prospectively predicted binge eating onset, by providing direct statistical evidence for this sequential relationship within a mediation framework. Furthermore, our findings complement cross-sectional observations by Ricca et al. [20] showing stepped increases in emotional eating scores across non-binge eaters, subthreshold BED, and full BED groups, by demonstrating how this progression may contribute to weight-related outcomes.

The identification of a significant direct mediation pathway from stress through binge eating to BMI, independent of the serial pathway, carries important theoretical and clinical implications. This

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finding indicates that binge eating may develop in response to stress via multiple mechanisms, not solely through the escalation of emotional eating. For example, some individuals might bypass a distinct emotional eating phase and develop binge eating directly in reaction to stress, potentially due to factors such as genetic predisposition, pre-existing impulse control difficulties, or exposure to particularly severe stressors [15,31–33]. This dual-pathway model aligns with Chao et al. [14], who reported that although both emotional eating and binge eating were linked to stress, only binge eating demonstrated a significant association with adverse metabolic outcomes. The simultaneous existence of both serial and direct pathways underscores binge eating as a critical intervention target, as it functions as the final common pathway for stress-related weight gain, regardless of whether individuals progress through emotional eating or develop binge eating independently.

4.2. Clinical and Practical Implications

The serial mediation results offer key guidance for prevention and intervention. First, recognizing emotional eating as a precursor to binge eating reveals a critical window for early prevention.

The serial mediation findings have significant implications for prevention and intervention strategies. The identification of emotional eating as a precursor to binge eating [19,20,22] suggests a critical window for early intervention (or prevention). Early interventions targeting emotion regulation skills, stress management, and adaptive coping strategies may prevent the escalation from emotional eating to binge eating [34,35]. Second, these results underscore the need to tailor weight management interventions to behavior severity. Individuals exhibiting binge eating may benefit from intensive, specialized treatments, whereas those with predominant emotional eating could respond well to lower-intensity programs. For those with established binge eating, interventions must target both emotional triggers and loss-of-control episodes. Integrating dialectical behavior therapy, mindfulness techniques, and cognitive-behavioral strategies can effectively address both emotion regulation deficits and binge eating behaviors [36–38].

4.3. Limitations

Several limitations should be acknowledged. First, the cross-sectional design precludes causal inferences about the ordering of stress, emotional eating, and binge eating pathways; multi-wave longitudinal data are needed to confirm causality. Second, the relatively small sample (N = 272) drawn from a single cultural context—predominantly young Korean adults—limits external validity to older, non-Korean, or clinical populations. Third, reliance on self-report questionnaires introduces potential response bias and measurement error; objective assessments (e.g., ecological momentary assessment, clinician-administered interviews) should be incorporated in future studies. Fourth, only one serial mediation model based on the overeating continuum framework was tested; alternative or parallel pathways (e.g., involving sleep disturbance, physical activity, or dietary quality) remain unexamined and warrant comparison using model-comparison approaches.

4.4. Future Directions

Future research should pursue the following directions:

- Longitudinal, multi-wave designs are essential to establish temporal precedence and examine how stress-related changes in eating behaviors predict weight gain trajectories over time.
- Expanding recruitment to larger, more diverse samples—including varied ages, ethnicities, and clinical populations with diagnosed BED—will enhance generalizability and clarify subgroup differences.
- Incorporating objective measures such as laboratory-based stress and eating assessments, wearable monitoring of physical activity, and biological markers (e.g., cortisol) can mitigate self-report biases and provide mechanistic insights.

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- Testing of competing mediation models and potential moderators (e.g., personality traits, genetic polymorphisms, environmental stressors) should delineate alternative pathways and identify individual vulnerability factors.
- Prospective or retrospective studies in clinical eating disorder cohorts can pinpoint transition points from emotional eating to BED, elucidate risk and protective factors, and inform targeted prevention and intervention strategies.

5. Conclusions

This study offers the first empirical support for a serial mediation model linking perceived stress to BMI via sequential pathways of emotional eating and binge eating. These results validate theoretical frameworks of an overeating continuum, where emotional eating precedes more severe binge episodes. Importantly, the presence of both serial and direct pathways underscores binge eating as a critical intervention target for obesity prevention and treatment. Clinically, emotional eating may serve as an early indicator of risk for escalating dysregulated eating and weight gain, warranting timely emotion-regulation interventions. Individuals with established binge eating may benefit from specialized, intensive treatments. Future studies should employ longitudinal, multi-wave designs, recruit larger and culturally diverse samples, and incorporate objective assessments to further elucidate and confirm the stress–eating–obesity nexus.

This study provides the first empirical evidence for a serial mediation model linking perceived stress to BMI through sequential pathways involving emotional eating and binge eating. The findings support theoretical models proposing that these eating behaviors exist on a severity continuum, with emotional eating representing an earlier stage that can progress to more severe binge eating patterns. The identification of both serial and direct pathways to binge eating highlights the clinical importance of this behavior as a key target for obesity prevention and treatment. From a practical standpoint, the findings suggest that emotional eating may serve as an early warning sign for individuals at risk of developing more severe eating problems and weight gain. Early identification and intervention targeting emotion regulation skills may prevent progression to binge eating, while individuals already experiencing binge eating may require more intensive, specialized interventions. Future studies should employ longitudinal, multi-wave designs, recruit larger and culturally diverse samples, and incorporate objective assessments to further clarify and confirm the stress–eating–obesity pathway.

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Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board of Pusan National University (PNU IRB/2023_18_HR, approved on 10 February 2023; 2024_148_HR, approved on 23 October 2024).

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The raw data supporting the conclusions of this article will be made available by the author on request.

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Abbreviations

The following abbreviations are used in this manuscript:

BED	**Binge eating disorder**
BES	Binge Eating Scale
BMI	Body mass index
DEBQ	Dutch Eating Behavior Questionnaires
HPA	Hypothalamic-pituitary-adrenal
PSS	Perceived Stress Scale
SD	Standard deviation

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