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Impulsivity, ınternalizing symptoms, and disordered eating as predictors of adolescents' body mass ındex: a structural equation modeling study in adolescents with overweight and obesity and their mothers

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Abstract

Obesity in children and adolescents, a global health concern with serious cardiometabolic and psychosocial impacts, requires understanding BMI risk factors to inform effective prevention and intervention strategies. This study aimed to compare the sociodemographic and clinical characteristics of adolescents with normal weight, overweight, and obesity to identify the main predictors of BMI. Methods: This crosssectional study included adolescents with normal weight (n = 100), overweight (n = 100), or obesity (n = 100) and their mothers. Adolescents were evaluated through semi-structured clinical interviews, and only those without comorbid psychiatric or medical conditions were included. Both adolescents and their mothers completed selfreport measures assessing disordered eating behaviors, impulsivity, and comorbid psychiatric symptoms. Structural equation modeling was used to examine the factors associated with adolescents' BMI. Adolescents with overweight and obesity and their mothers exhibited higher levels of comorbid anxiety and depressive symptoms, greater engagement in restrictive, emotional, and external eating behaviors, and increased impulsivity compared to those with normal weight ($p < .05$). The analysis shows that adolescents' BMI is directly predicted by adolescents' impulsivity (β = .842) and gender (β = .073). Maternal impulsivity indirectly influences adolescents' BMI through multiple pathways, including both maternal (β = .360) and adolescent (β = .629) disordered eating behaviors and adolescents' impulsivity (β = .120). Additionally, maternal BMI, maternal and adolescent disordered eating behaviors further contribute to these indirect effects. This study demonstrates the dynamic relationship between adolescent and maternal factors in predicting adolescent BMI. The findings emphasize the importance of targeting both adolescent and maternal factors, particularly impulsivity, to develop effective obesity prevention and intervention strategies.

Keywords Impulsivity · Internalizing symptoms · Disordered eating · Adolescent · Obesity · Maternal

Obesity is a major global health problem associated with serious cardiometabolic and psychosocial consequences, including increased risk of early mortality in children and adolescents. The Centers for Disease Control and Prevention (CDC) define overweight (OW) in youth as a body mass index (BMI) between the 85th and 95th percentiles, and obesity as a BMI at or above the 95th percentiles based on age- and sex-specific growth charts (CDC, 2000). Over the past five decades, obesity rates among children and adolescents have risen sharply worldwide (Jebeile et al., 2022). One study reported that from 1975 to 2016, the age-standardized prevalence rates of obesity increased from 0.7% to 5.6% in girls and from 0.9% to 7.8% in boys (NCD-RisC, 2017). The German Health Interview and Examination Survey for Children and Adolescents (KiGGS) found that OW and obesity rates tend to increase with age (Mauz et al., 2020). Beyond their negative effects on physical health, overweight and obesity are associated with several psychosocial outcomes, including lower self-esteem, higher internalizing

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symptoms, reduced quality of life, and disordered eating behaviors (Jebeile et al., 2022). While these psychosocial outcomes are well-documented, the relationships between impulsivity, internalizing symptoms, and disordered eating in shaping BMI trajectories remain underexplored.

Adolescence represents a critical period marked by significant physiological, behavioral, and psychosocial changes that may increase vulnerability to obesity. From a physiological perspective, hormonal alterations lead to an increase in the number and size of adipocytes, particularly in females, and greater body fat contributes to reduced insulin sensitivity. Common changes in eating behaviors—such as increased consumption of processed and fast foods, larger portion sizes, and more frequent eating out—along with prolonged screen time and reduced physical activity, further exacerbate the risk of obesity during this stage (Alberga et al., 2012; Jebeile et al., 2022). Among the psychosocial changes, the growing prominence of physical appearance as a determinant of social acceptance and self-esteem is particularly noteworthy (Smink et al., 2018). Furthermore, emerging emotion regulation difficulties may lead adolescents to use eating behavior as a coping mechanism to alleviate negative emotions. Considering these factors, understanding the potential causes of obesity at this vulnerable developmental stage is important for the development of preventive public health strategies and tailored intervention programs in pediatric populations.

The potential role of impulsivity in the onset and maintenance of obesity has become an important area of research. Impulsivity is typically defined as the tendency to act without considering possible negative consequences, manifesting as increased novelty seeking, disadvantageous decision-making, and poor behavioral/cognitive control (Romer et al., 2017). A meta-analysis on the role of impulsivity in obesity reported a moderate effect size in the relationship between general impulsivity and obesity in the pediatric population (Thamotharan et al., 2013). Although impulsivity is generally associated with externalizing behaviors, it is also considered a risk factor for internalizing symptoms due to increased sensitivity to negative emotional stimuli, emotion regulation difficulties, and impulsive reactivity (Peters et al., 2015). Internalizing symptoms, on the other hand, not only interact with impulsivity but also represent an independent risk factor for obesity (Uysal et al., 2024). Psychosocial factors such as peer victimization, social stigma, and body dissatisfaction contribute to higher rates of internalizing symptoms in adolescents with obesity. In addition, internalizing symptoms may play a role in the bidirectional relationship with obesity through mechanisms such as emotional dysregulation, disrupted sleep–wake cycle, and poor dietary and physical activity behaviors (Wojcik et al., 2023). Given their central role in the onset, maintenance, and exacerbation of obesity, understanding the relationships between impulsivity, internalizing symptoms, and BMI could improve both psychological well-being and clinical outcomes in adolescents with higher weight status.

Adolescents cannot be considered in isolation from the family system. The role of mothers is particularly important, as they are still the primary caregivers in most households. Therefore, it is important to assess maternal factors as well as individual risk factors in adolescents with obesity. Numerous studies have consistently associated parental eating behaviors and weight status with children's weight outcomes (Mahmood et al., 2021). A meta-analysis examining the association between maternal pre-pregnancy BMI and childhood obesity found that maternal pre-pregnancy obesity was associated with a 3.64-fold increase in the odds of childhood (Heslehurst et al., 2019). However, studies particularly investigating the relationship between maternal impulsivity, maternal internalizing symptoms, and children's BMI remain scarce. One study reported a statistically significant positive relationship between parental impulsivity and children's BMI—particularly in girls—but found no mediating effect of parent-reported child impulsivity (Sleddens et al., 2016). Another study involving a sample of 258 mothers of obese and non-obese children found that mothers of children with obesity scored higher on all subscales of the Minnesota Multiphasic Personality Inventory than mothers of children without obesity (Precenzano et al., 2021).

Building upon the findings presented, the primary aim of this study was to compare adolescents with normal weight (NW), OW, and obesity and their mothers in terms of internalizing symptoms, impulsivity, and disordered eating behaviors. In addition, the bidirectional effects of the adolescents' and their mothers' levels of impulsivity, internalizing symptoms, and disordered eating behaviors on the adolescents' BMI were aimed to be assessed. The final aim of the study was to examine the predictors of adolescents' BMI. Our main hypotheses in this study are (1) Adolescents with OW and obesity and their mothers would have higher levels of internalizing symptoms, impulsivity, and disordered eating behaviors compared to those of NW. (2) Adolescents with OW and obesity and their mothers would have higher levels of impulsivity and disordered eating behaviors, even after controlling for internalizing symptoms. (3) Adolescents' impulsivity would increase their percentile BMI indirectly via disordered eating behaviors, while maternal impulsivity indirectly influences adolescents' BMI by increasing their impulsivity levels. (4) Adolescents' impulsivity, internalizing symptoms, gender, and maternal impulsivity would be important predictors of BMI percentile values in adolescents.

Material and methods

Participants and procedure

This case–control study recruited adolescents aged 12 to 18 years, together with their mothers (only mothers were included, as all primary caregivers were mothers). Participants were initially selected from adolescents who presented to the pediatric outpatient clinic of the Training and Research Hospital for several reasons (e.g., cold, stomachache, constipation, routine follow-up) between December 1, 2024, and February 1, 2025. The adolescents were referred to the child and adolescent psychiatry outpatient clinic after their routine assessment at the pediatric outpatient clinic. Adolescents were eligible for inclusion if they had no psychiatric diagnosis, were not using psychiatric medication, had no chronic conditions such as diabetes mellitus, hypertension, asthma, or neurological or cardiological diseases, and provided written consent. Participants were divided into three groups based on their BMI percentile values: 5%−84%, normal weight (NW); 85%−94%, overweight (OW); and 95% and above, with obesity. The final sample therefore represents a specific clinical subgroup derived from a general pediatric outpatient population.

Participants' age, gender, and anthropometric measurements were recorded by the researchers. All participants underwent a semi-structured diagnostic interview using the Kiddie-Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version DSM-V (K-SADS-PL) to ensure the absence of psychiatric comorbidities. These interviews were conducted face-to-face by a child and adolescent psychiatrist. Both the adolescents and their mothers completed the Dutch Eating Behavior Questionnaire (DEBQ) and the Barratt Impulsiveness Scale-Brief (BIS-Brief). Furthermore, adolescents individually completed the Revised Children's Anxiety and Depression Scale—Child Version (RCADS-C), while mothers filled out the Revised Children's Anxiety and Depression Scale—Parent Version (RCADS-P) and the Brief Symptom Inventory (BSI). Figure 1 provides information on the initial number of participants assessed, the evaluation procedure, the number of participants excluded along with their reasons for exclusion, and the final sample size.

The ethical committee of Kastamonu University approved the protocol (November 20, 2024/No: 2024- KAEK- 110). A written informed consent form was obtained from each of the participants and their families. Participants were thoroughly informed about the purpose and design of the study, the timeline, expectations from participants, the potential risks and benefits, assurances about data confidentiality, and their right to withdraw at any time without facing any consequences.

Measures

The Kiddie-Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version DSM-V (K-SADS-PL) is a semi-structured interview form that has demonstrated validity and reliability in a Turkish sample (Unal et al., 2019). The K-SADS-PL was administered to adolescents and their mothers by a child and adolescent psychiatrist to exclude adolescents with any psychiatric disorders from the initial cohort.

The Revised Children's Anxiety and Depression Scale (RCADS) is a 47-item self-report instrument designed to evaluate symptoms of anxiety and depression in children and adolescents, with total scores ranging from 0 to 141. It includes both a child version (RCADS-C) and a parent version (RCADS-P), which are used together to provide a comprehensive clinical assessment. Raw scores from its subscales are transformed into T-scores, with T-scores of 65 or higher indicating clinical significance. Alongside the subscale scores, a total internalizing score is derived by summing all subscales, offering an overall evaluation of the severity of the internalizing symptoms. The total internalizing score was used primarily in statistical analyses. The Turkish adaptations of the RCADS-C and RCADS-P have demonstrated reliable and valid psychometric properties (Görmez et al., 2017a, 2017b). In this study, Cronbach's alpha was 0.882.

The Brief Symptom Inventory (BSI), which was developed to assess various psychiatric symptoms, includes 9 subscales and 3 global indices. In its Turkish adaptation, five main factors were identified: anxiety, depression, negative self-image, somatization, and hostility. The global indices consist of the Global Severity Index (GSI), which assesses the overall level of distress; the Positive Symptom Total (PST), representing the total number of symptoms reported; and the Positive Symptom Distress Index (PSDI), reflecting the average intensity of distress experienced (Hisli Şahin & Durak, 1994). The PSDI was used primarily in statistical analyses. In this study, Cronbach's alpha was 0.899.

The Dutch Eating Behavior Questionnaire (DEBQ) is a 33-item self-report instrument consisting of three subscales: external eating, emotional eating, and restrictive eating. It is scored on a 5-point Likert scale, with higher scores indicating an increase in disordered eating behaviors. DEBQ is a valid and reliable psychometric instrument for Turkish samples (Bozan et al., 2011). In this study, Cronbach's alpha was 0.987 for adolescents and 0.915 for mothers.

The Barratt Impulsiveness Scale-Brief (BIS-Brief) is an 8-item self-report instrument designed to assess impulsivity in both clinical and non-clinical populations. It comprised two distinct dimensions: poor self-regulation and impulsive behavior. The BIS-Brief is a valid and reliable psychometric

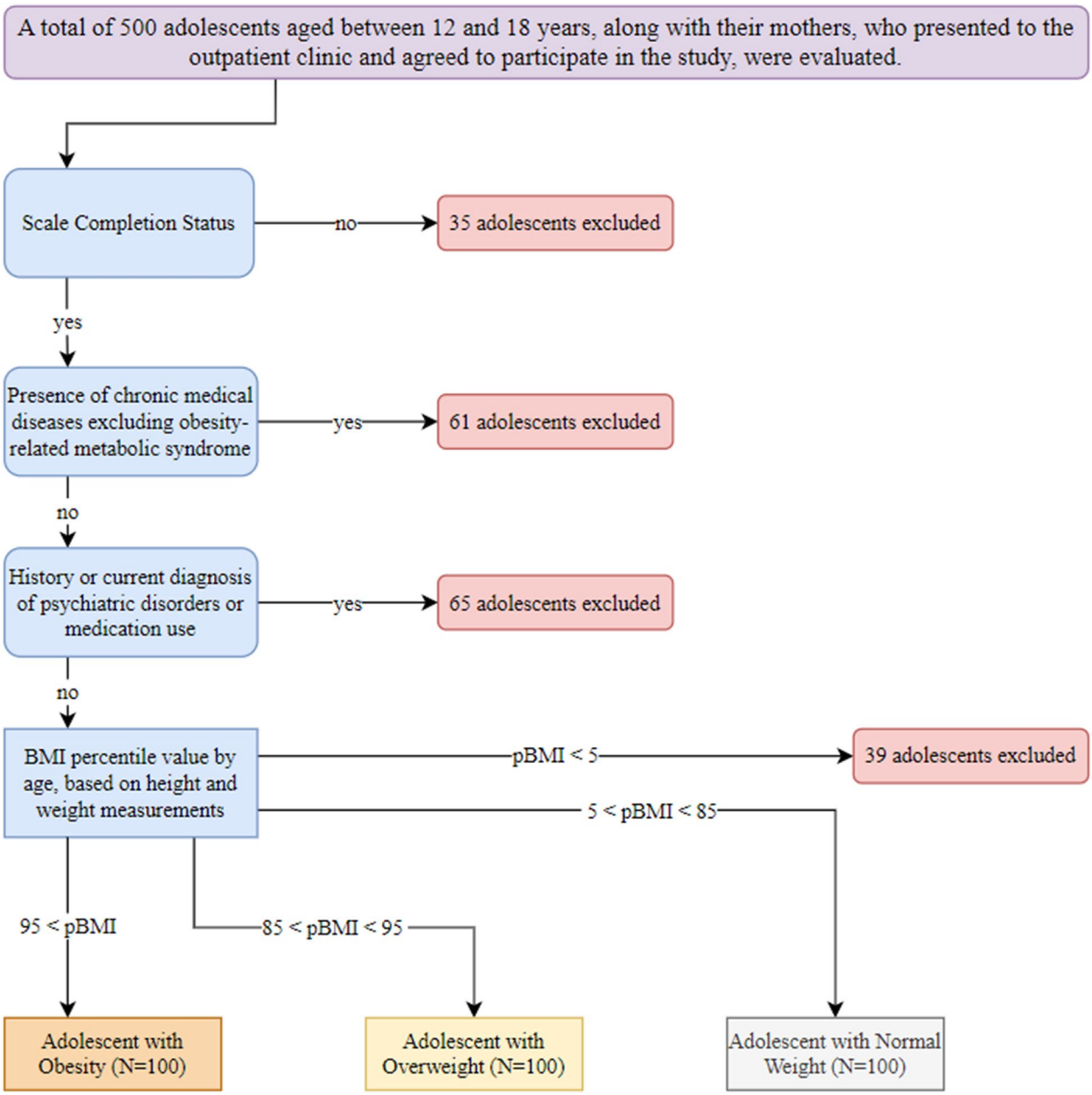

Fig. 1 Flow diagram

tool for the Turkish sample (Benk Durmuş et al., 2022). In this study, Cronbach's alpha was 0.876 for adolescents and 0.924 for mothers.

Statistical analysis

We conducted all statistical analyses using SPSS version 28.0 and STATA version 14.2. The significance level was set at $p < 0.05$ for all tests. Descriptive statistics were used to summarize demographic and anthropometric characteristics, and group differences were evaluated using one-way analysis of variance (ANOVA) and chi-square (χ^2) tests, as appropriate. Post-hoc comparisons for significant ANOVA results were conducted using Tukey's HSD test. Analysis of covariance (ANCOVA) was employed to examine differences in impulsivity (BIS-Brief Total), and eating behaviors (DEBQ-Total) across weight status groups, controlling for relevant covariates. For adolescents, the RCADS-C Total Internalizing score was included as the covariate, while for mothers, the BSI-PSDI score was included as the covariate. Partial correlation analyses were conducted to investigate the direct relationships between adolescents' percentile BMI (pBMI) and measures of impulsivity and eating behaviors in both adolescents and mothers. These analyses controlled for

maternal BMI, adolescents' gender, adolescents' total internalizing scores (RCADS-C), and maternal psychological distress (BSI-PSDI). Linear regression analyses were performed to identify independent predictors of adolescents' pBMI. Predictor variables included adolescents' gender, impulsivity (BIS-Brief Total), eating behaviors (DEBQ-Total), and total internalizing scores (RCADS-C). Maternal variables such as BMI, impulsivity (BIS-Brief Total), eating behaviors (DEBQ-Total), and psychological distress (BSI-PSDI) were also included in the regression models.

Structural equation modeling (SEM) was conducted using STATA to test the hypothesized relationships between maternal and adolescent impulsivity, eating behaviors, BMI, and adolescent pBMI. Model fit was evaluated using multiple indices, including the chi-square test, root mean square error of approximation (RMSEA), comparative fit index (CFI), Tucker-Lewis index (TLI), and standardized root mean square residual (SRMR). The coefficient of determination (CD) was reported to assess the explanatory power of the model. To facilitate interpretation of the model fit indices, brief explanations and commonly accepted thresholds are provided as follows: RMSEA (Root Mean Square Error of Approximation) estimates the degree of error in the model's approximation of the population covariance matrix—lower values indicate better fit, with values <0.06 considered excellent. CFI (Comparative Fit Index) and TLI (Tucker-Lewis Index) assess model fit relative to a null model, accounting for sample size and model complexity—values >0.95 are generally interpreted as indicative of excellent fit. SRMR (Standardized Root Mean Square Residual) represents the standardized difference between observed and predicted correlations, with values <0.08 suggesting a good fit. These indices collectively help determine how well the hypothesized structural model captures the observed data patterns. Figure 2 depicts the hypothesized model examining the interconnected relationships between maternal and adolescent impulsivity (measured by BIS-Brief Total), disordered eating behaviors (assessed by DEBQ-Total), maternal BMI, and adolescent pBMI. The model proposes that both maternal and adolescent impulsivity influence disordered eating behaviors, which in turn affect BMI in mothers and pBMI in adolescents. In addition, direct pathways between impulsivity and BMI measures are hypothesized to capture potential direct effects independent of disordered eating behaviors. Although SEM allows for the modeling of latent constructs, in this study we used total scores for BIS-Brief and DEBQ due to their strong psychometric properties and frequent use as composite indicators in previous research. Furthermore, the short length of the BIS-Brief precluded reliable latent variable modeling. This model aims to elucidate the complex dynamics underlying shared behavioral and physiological traits within mother-adolescent dyads. This comprehensive approach allowed for a detailed investigation of the relationships between maternal and adolescent characteristics, highlighting direct and indirect pathways influencing adolescent pBMI.

Results

Table 1 presents the demographic, anthropometric and clinical characteristics of participants based on weight status categories (O, OW, and NW). The mean age did not differ significantly between the groups ($F(2)=0.30$, $p=0.738$). Gender distribution was also comparable across groups, with no statistically significant differences observed ($\chi^2(2)=2.670$, $p=0.263$). As expected, participants' pBMI differed significantly across the groups ($F(2)=385.34$, $p<0.001$). Mothers' mean age was not significantly different between the groups ($F(2)=1.83$, $p=0.162$). However, mothers' BMI showed a statistically significant difference between groups ($F(2)=4.22$, $p=0.016$). Post-hoc

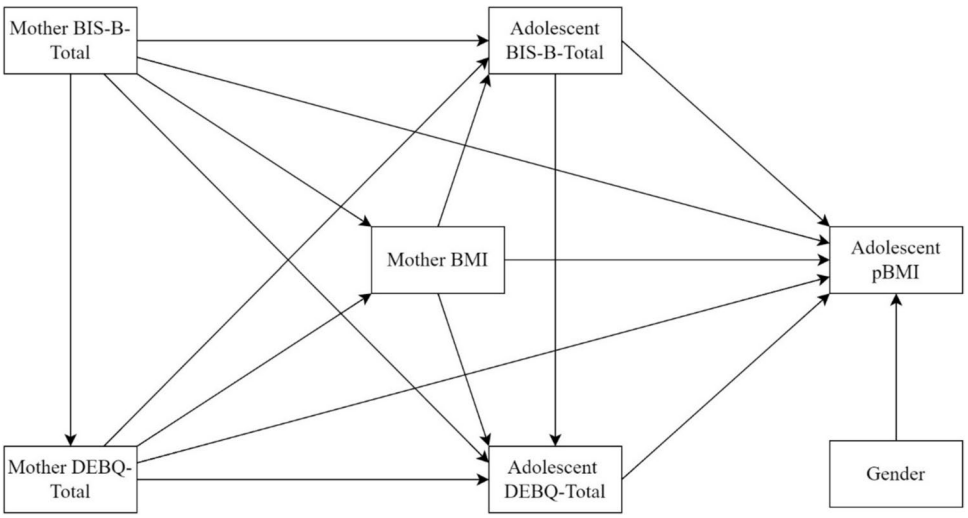

Fig. 2 Hypothesized model of relationships between maternal and adolescent ımpulsivity, disordered eating behaviors, BMI, and adolescent pBMI

Table 1 Demographic, anthropometric, and clinical characteristics of participants

	O (N = 100)	OW (N = 100)	NW (N = 100)	Statistics*		Post-hoc diff
	M (*SD*) or n (%)	*M* (*SD*) or n (%)	*M* (*SD*) or n (%)	*F* or χ^2	*p*	
Age *(years)*	14.89 (2.04)	14.89 (2.09)	14.70 (1.83)	$F(2) = .30$	.738	-
Gender						
Female	45 (45%)	55 (55%)	55 (55%)	$\chi^2(2) = 2.67$	.263	-
Male	55 (55%)	45 (45%)	45 (45%)			
pBMI	96.98 (1.42)	89.67 (2.88)	46.23 (23.99)	$F(2) = 385.34$	**<.001**	NW < OW < O
Mothers age *(years)*	43.10 (5.78)	42.98 (5.23)	44.25 (4.44)	$F(2) = 1.83$	.162	-
Mothers BMI *(kg/m²)*	27.02 (4.73)	27.03 (4.43)	25.47 (3.87)	$F(2) = 4.22$	.016	NW < O = OW
RCADS-C						
Total Anxiety & Depression	68.64 (10.56)	69.74 (9.86)	58.57 (13.16)	$F(2) = 29.75$	**<.001**	NW < O = OW
BSI						
GSI	1.43 (.96)	1.79 (.91)	.56 (.51)	$F(2) = 60.13$	**<.001**	NW < O < OW
PST	76.01 (50.88)	94.75 (48.12)	29.50 (26.94)	$F(2) = 60.15$	**<.001**	NW < O < OW
PSDI	2.05 (.69)	2.32 (.68)	1.29 (.44)	$F(2) = 75.75$	**<.001**	NW < O < OW

M Mean; *SD* Standard Deviation; *pBMI* Body Mass Index Percentile; *BMI* Body Mass Index; O Adolescent with Obesity; OW Adolescent with Overweight; NW Adolescent with Normal Weight; *RCADS-C* Revised Children's Anxiety and Depression Scale—Child Version; *BSI* Brief Symptom Inventory; *GSI* Global Severity Index; *PST* Positive Symptom Total; *PSDI* Positive Symptom Distress Index. Comparative analyses of categorical variables were conducted using Pearson's chi-square test, and continuous variables were analyzed using one-way ANOVA followed by Tukey post hoc tests

Table 2 Correlation matrix of adolescent pBMI, gender, BIS-total B, DEBQ-total, RCADS-C-total, and maternal BMI, BIS-B-total, DEBQ-total, and BSI-PSDI scores

		ADOLESCENT					MOTHER			
		pBMI	Gender	BIS-B-Total	DEBQ-Total	RCADS-C-Total	mBMI	BIS-B-Total	DEBQ-Total	BSI-PDSI
ADOLESCENT	pBMI	1								
	Gender	-.14*	1							
	BIS-B-Total	.84**	-.08	1						
	DEBQ-Total	.58**	-.03	.69**	1					
	RCADS-C-Total	.36**	-.02	.40**	.44**	1				
MOTHER	mBMI	.11	-.06	.16**	.26**	.33**	1			
	BIS-B-Total	.81**	-.08	.94**	.69**	.38**	.17**	1		
	DEBQ-Total	.35**	-.08	.36**	.39**	.31**	.24**	.36**	1	
	BSI-PDSI	.42**	-.00	.51**	.44**	.24**	.08	.50**	.16**	1

RCADS-C-Total The Revised Children's Anxiety and Depression Scale—Child Version Total Anxiety and Depression Scores; *BSI* Brief Symptom Inventory; *PSDI* Positive Symptom Distress Index, *pBMI* Body Mass Index Percentile; *mBMI* Maternal Body Mass Index; *BIS-B* Barratt Impulsiveness Scale-Brief; *DEBQ* Dutch Eating Behavior Questionnaire

*Pearson correlations were used. *$p < .05$, **$p < .01$. Gender was coded as 0 = female and 1 = male

comparisons indicated that mothers of NW participants had a significantly lower BMI compared to mothers of O and OW participants, with no significant difference between the latter two groups (NW < O = OW). The ANOVA results indicate significant group differences in internalizing symptoms and psychological distress. For the RCADS-C Total Anxiety and Depression score, NW participants reported significantly lower levels of anxiety and depression compared to both O and OW participants ($F(2) = 29.75$, $p < 0.001$). Similarly, significant differences were found across all BSI global scores. The GSI, PST, and PSDI scores were lowest in NW participants and highest in OW participants, with intermediate values for O participants (GSI: $F(2) = 60.13$, $p < 0.001$; PST: $F(2) = 60.15$, $p < 0.001$; PSDI: $F(2) = 75.75$, $p < 0.001$). Group differences for the DEBQ subscales and the RCADS-C and RCADS-P subscales are provided in Supplementary Table S1.

Table 2 shows the correlation matrix among adolescents' percentile BMI, gender, BIS-B-Total, DEBQ-Total, and RCADS-C-Total, as well as corresponding maternal variables including maternal BMI, BIS-B-Total, DEBQ-Total, and BSI-PSDI. Adolescent pBMI was significantly positively correlated with BIS-B-Total ($r = 0.84$, $p < 0.01$), DEBQ-Total ($r = 0.58$, $p < 0.01$), and RCADS-C-Total ($r = 0.36$, $p < 0.01$). It was also significantly associated with maternal BIS-B-Total ($r = 0.81$, $p < 0.01$), maternal DEBQ-Total ($r = 0.35$, $p < 0.01$), and maternal BSI-PSDI ($r = 0.42$, $p < 0.01$). Adolescent gender showed a small but significant negative correlation with pBMI (r—0.14, $p < 0.05$), indicating higher pBMI among females. Additionally, strong

positive correlations were observed between adolescent and maternal BIS-B-Total ($r=0.94$, $p<0.01$), and between adolescent and maternal DEBQ-Total ($r=0.39$, $p<0.01$). Maternal BSI-PSDI was also significantly correlated with both adolescent and maternal BIS-B-Total and DEBQ-Total.

Table 3 presents the results of the ANCOVA analyses comparing adolescents' impulsivity (BIS-Brief Total) and eating behavior (DEBQ-Total) scores, with the covariate being adolescents' self-reported total internalizing scores measured by the RCADS-C. Similarly, for mothers, impulsivity (BIS-Brief Total) and eating behavior (DEBQ-Total) scores were compared, with the covariate being mothers' PSDI scores from the BSI. For adolescents, significant differences were observed across weight status groups in both impulsivity and eating behavior scores ($p<0.001$ for all). Post-hoc analyses indicated that adolescents with NW had significantly lower scores on both BIS-Brief Total and DEBQ-Total compared to adolescents with OW and O, while the latter two groups did not differ significantly (NW < O = OW). Furthermore, subscale analyses of eating behavior (restrained eating, emotional eating, and external eating) revealed similar patterns, with adolescents with NW consistently scoring lower than their OW and O peers, and significant differences between OW and O groups being observed for restrained and emotional eating scores (NW < OW < O). For mothers, significant differences were also found across weight status groups in impulsivity and eating behavior scores ($p<0.001$ for all). Mothers of adolescents with NW scored significantly lower on both BIS-Brief Total and DEBQ-Total compared to mothers of adolescents with OW and O, with no significant differences between the latter two groups (NW < O = OW). Subscale analyses of eating behavior revealed consistent trends, with mothers of adolescents with NW scoring the lowest on restrained eating, emotional eating, and external eating subscales. For external eating, scores showed a graded increase across weight groups, with NW mothers scoring lower than OW mothers, who scored lower than O mothers (NW < OW < O).

Table 4 presents the results of a linear regression analysis investigating potential independent variables predicting adolescents' pBMI across the entire sample. The model was statistically significant ($F(8)=94.84$, $p<0.001$), with an R^2 value of 0.72, indicating that 72.3% of the variance in pBMI was explained by the predictors included in the model. The adjusted R^2 value was 0.72. Among adolescents, gender was a significant predictor of pBMI ($\beta=-0.07$, $t(292)=-2.31$, $p=0.021$), with males showing lower pBMI values compared to females. Impulsivity, as measured by BIS-Brief Total, also emerged as a strong positive predictor ($\beta=0.68$, $t(292)=7.19$, $p<0.001$), indicating that higher impulsivity scores were associated with higher pBMI. However, DEBQ-Total (eating behaviors) and RCADS-C Total Internalizing Scores were not significant predictors of pBMI. For mothers, none of the predictors—BMI, BIS-Brief Total (impulsivity), DEBQ-Total (eating behaviors), or BSI-PSDI (psychological distress)—significantly predicted adolescents' pBMI. The results of the linear regression analyses examining the predictive effects of RCADS-C subscales on restrictive, emotional, and external eating behaviors in adolescents within each group (O, OW, NW) are provided in the supplementary material (Tables S5-S14). Table S15 also shows the results of partial correlation analyses examining the direct relationships between adolescents' pBMI and measures of impulsivity and eating behaviors in both adolescents and their mothers in the supplementary material.

We first tested the initial model, which was theoretically constructed based on previous research findings and our clinical observations (see Fig. 2). This model assumed that adolescents' eating behaviors would have a direct or indirect effect on pBMI. However, the results indicated that eating behaviors did not significantly predict pBMI (see Fig. 3). In light of this finding, we considered the possibility that a mediator variable may have been overlooked in the initial model and subsequently developed an alternative model. This revised model assumed that adolescents' impulsivity increased eating behavior, which in turn influenced pBMI. This alternative model (see Fig. 4) showed better fit indices than the initial model and was also more theoretically sound. It was therefore accepted as the final model.

Figure 3 presents the SEM results of the first model initially hypothesized and tested in Fig. 2, with all specified pathways included. In this model, mother BIS-B-Total significantly predicted mother DEBQ-Total ($\beta=0.36$, $p<0.001$), adolescent DEBQ-Total ($\beta=0.36$, $p=0.003$), and adolescent BIS-B-Total ($\beta=0.94$, $p<0.001$). Mother DEBQ-Total was positively associated with both adolescent DEBQ-Total ($\beta=0.13$, $p=0.004$) and mother BMI ($\beta=0.21$, $p<0.001$), while mother BMI significantly predicted only adolescent DEBQ-Total ($\beta=0.12$, $p=0.003$). Additionally, adolescent BIS-B-Total positively predicted both adolescent DEBQ-Total ($\beta=0.28$, $p=0.019$) and adolescent pBMI ($\beta=0.69$, $p<0.001$). Gender significantly predicted adolescent pBMI ($\beta=$–0.07, $p=0.019$). However, the path from adolescent DEBQ-Total to adolescent pBMI was not statistically significant ($\beta<$–0.01, $p=0.987$), nor were several other pathways (e.g., from Mother BIS-B-Total to Mother BMI, $\beta=0.10$, $p=0.095$; from Mother BIS-B-Total to Adolescent pBMI, $\beta=0.15$, $p=0.114$; from Mother BMI to Adolescent BIS-B-Total, $\beta=$–0.01, $p=0.757$; from Mother BMI to Adolescent pBMI, $\beta=$–0.05, $p=0.987$; from Mother DEBQ-Total to Adolescent pBMI, $\beta=0.06$, $p=0.104$; from Mother DEBQ-Total to Adolescent BIS-B-Total, $\beta=0.02$, $p=0.362$). After removing all non-significant pathways, a more parsimonious model was generated, as shown in the

Table 3 Results of ANCOVA comparing impulsivity and eating behaviors in adolescents and mothers, with R-CADS-C-total internalizing score and BSI-PSDI as covariates

		O (N = 100)		OW (N = 100)		NW (N = 100)		Statistics*		Part. Eta Sq	Cohen d (O vs OW) (O vs NW) (OW vs NW)	Post-hoc diff
		M (*SD*)	Adj. *M* (*SE*)	*M* (*SD*)	Adj. *M* (*SE*)	*M* (*SD*)	Adj. *M* (*SE*)	*F(2)*	*p*			
ADOLESCENT	BIS-B-TOTAL	25.49 (1.29)	25.48 (.15)	25.41 (1.42)	25.40 (.15)	12.73 (1.69)	12.75 (.16)	2046.0	<.001	.933	.06 8.49 8.12	NW < O = OW
	Poor Self-regulation	9.00 (.83)	8.99 (.08)	8.95 (.82)	8.93 (.08)	4.87 (.82)	4.91 (.09)	671.6	<.001	.820	.06 5.01 4.98	NW < O = OW
	Impulsive Behavior	16.49 (1.11)	16.50 (.13)	16.46 (1.15)	16.47 (.13)	7.86 (1.44)	7.84 (.13)	1344.1	<.001	.901	.03 6.71 6.60	NW < O = OW
	DEBQ-TOTAL	131.51 (17.58)	129.77 (2.65)	121.07 (30.66)	118.69 (2.68)	67.94 (30.63)	72.06 (2.79)	115.2	<.001	.438	.42 2.55 1.73	NW < OW < O
	Restrained Eating Behavior	52.60 (9.49)	51.79 (1.28)	47.29 (14.71)	46.18 (1.30)	25.41 (14.26)	27.34 (1.35)	86.4	<.001	.369	.43 2.24 1.51	NW < OW < O
	Emotional Eating Behavior	40.26 (7.46)	39.81 (.91)	36.32 (9.84)	35.71 (.92)	21.04 (9.81)	22.10 (.96)	90.9	<.001	.381	.45 2.21 1.56	NW < OW < O
	External Eating Behavior	38.65 (7.06)	38.17 (.85)	37.46 (9.57)	36.81 (.86)	21.49 (9.02)	22.62 (.90)	87.2	<.001	.371	.14 2.12 1.72	NW < O = OW

Table 3 (continued)

		O (N = 100)		OW (N = 100)		NW (N = 100)		Statistics*		Part. Eta Sq	Cohen d (O vs OW) (O vs NW) (OW vs NW)	Post-hoc diff
		M (*SD*)	Adj. *M* (*SE*)	*M* (*SD*)	Adj. *M* (*SE*)	*M* (*SD*)	Adj. *M* (*SE*)	*F(2)*	*p*			
MOTHER	BIS-B-TOTAL	20.91 (1.14)	20.92 (.13)	21.07 (1.24)	21.10 (.14)	8.46 (1.37)	8.42 (.14)	2290.2	<.001	.939	-.13 9.88 9.65	NW<O=OW
	Poor Self-regulation	6.99 (.82)	6.99 (.08)	7.08 (.84)	7.07 (.09)	2.92 (.82)	2.93 (.10)	552.8	<.001	.789	-.11 4.96 5.01	NW<O=OW
	Impulsive Behavior	13.92 (.79)	13.93 (.09)	13.99 (.83)	14.03 (.10)	5.54 (1.12)	5.49 (.11)	1907.6	<.001	.928	-.09 8.65 8.57	NW<O=OW
	DEBQ-TOTAL	92.28 (47.58)	92.17 (4.16)	81.59 (46.63)	81.30 (4.44)	52.90 (24.63)	53.30 (4.72)	17.8	<.001	.107	.23 1.04 .77	NW<O=OW
	Restrained Eating Behavior	35.31 (20.07)	35.23 (1.77)	31.05 (19.87)	30.85 (1.89)	19.09 (10.68)	19.37 (2.01)	16.4	<.001	.100	.21 1.01 .76	NW<O=OW
	Emotional Eating Behavior	28.24 (14.06)	28.19 (1.25)	25.34 (13.95)	25.20 (1.33)	16.79 (7.79)	16.98 (1.41)	16.5	<.001	.100	.21 1.01 .74	NW<O=OW
	External Eating Behavior	28.73 (14.58)	26.75 (1.25)	25.20 (13.67)	25.26 (1.34)	17.02 (7.58)	16.94 (1.42)	18.1	<.001	.109	.25 1.01	NW<OW<O

M mean; *SD* standard deviation; *SE* standard error; *Adj.* Adjusted, *p* Significance – *p* value; *O* Adolescent with Obesity; *OW* Adolescent with Overweight; *NW* Adolescent with Normal Weight, *Post-hoc diff.* Post-hoc differences, *RCADS-C* The Revised Children's Anxiety and Depression Scale—Child Version; *BSI* Brief Symptom Inventory; *PSDI* Positive Symptom Distress Index; *BIS-B* Barratt Impulsiveness Scale-Brief; *DEBQ* Dutch Eating Behavior Questionnaire; *Part. Eta Sq.* Partial Eta Squared

*In the comparison (ANCOVA) of adolescents' scales across groups, the internalizing score R-CADS-C Total was used as a covariate. For the comparison of maternal impulsivity and eating behaviors across groups, BSI-PSDI was used as a covariate

Table 4 Linear regression analysis of factors predicting adolescents' percentile BMI (pBMI)

		Unstandardized Coefficients		Standardized Coefficients			95% Confidence Interval for *B*	
		B	*SE*	*β*	*t*	*p*	Lower Bound	Upper Bound
	(constant)	−8.300E-16	.03	-	.00	1.000	-.06	.06
ADOLESCENT	Gender	-.07	.03	-.07	−2.31	**.021**	-.13	-.01
	BIS-B-TOTAL	.68	.10	.68	7.19	**<.001**	.50	.87
	DEBQ-TOTAL	-.01	.05	-.01	-.12	.903	-.10	.09
	RCADS-C-Total	.04	.04	.04	1.05	.295	-.03	.11
MOTHER	BMI	-.06	.03	-.06	−1.70	.091	-.12	.01
	BIS-B-TOTAL	.15	.10	.15	1.62	.105	-.03	.34
	DEBQ-TOTAL	.05	.04	.05	1.43	.154	-.05	.12
	BSI-PSDI	-.02	.04	-.02	-.57	.572	-.09	.05

SE standard error; *β* Beta; *p* Significance– *p* value; *Adj.* Adjusted; *RCADS-C* The Revised Children's Anxiety and Depression Scale—Child Version; *BSI* Brief Symptom Inventory; *PSDI* Positive Symptom Distress Index; *BIS-B* Barratt Impulsiveness Scale-Brief; *DEBQ*=Dutch Eating Behavior Questionnaire

*dependent variable=pBMI, N=300, $F(8, 292)=94.84$, $p<.001$, $R^2=.72$, Adj. $R^2=.72$. Gender was coded as 0=female and 1=male

lower panel of Fig. 3. This refined model demonstrated excellent fit to the data, with fit indices of RMSEA=0.024, SRMR=0.032, CFI=0.999, TLI=0.997, and CD=0.896.

Figure 4 presents the final SEM tested in this study, based on iterative model comparisons and theoretical refinement. This model was developed following the alternative models depicted in Figs. 2 and 3 and demonstrated improved model fit. All pathways shown are statistically significant, except for the path between Adolescent DEBQ-Total and Adolescent pBMI, which remained non-significant and was therefore excluded from the final model. In this model, mother BIS-B-Total significantly predicted both mother DEBQ-Total ($\beta=0.36$, $p<0.001$) and adolescent BIS-B-Total ($\beta=0.90$, $p<0.001$). Mother DEBQ-Total was a significant predictor of both adolescent DEBQ-Total ($\beta=0.13$, $p=0.004$) and mother BMI ($\beta=0.24$, $p<0.001$), and mother BMI significantly predicted only adolescent DEBQ-Total ($\beta=0.12$, $p=0.004$). Additionally, Adolescent DEBQ-Total significantly predicted Adolescent BIS-B-Total ($\beta=0.06$, $p=0.017$). Adolescent BIS-B-Total significantly predicted adolescent pBMI ($\beta=0.84$, $p<0.001$). Gender also emerged as a significant predictor of adolescent pBMI ($\beta=$—0.07, $p=0.019$). The model exhibited excellent fit to the data, with the following fit indices: RMSEA=0.022, SRMR=0.030, CFI=0.999, TLI=0.998, and CD=0.896. Notably, these fit statistics indicate a slightly better model fit compared to the alternative model presented in Fig. 3, which reported RMSEA=0.024, SRMR=0.032, CFI=0.999, and TLI=0.997. This improvement further supports the final model's robustness and parsimony.

Discussion

To our knowledge, this is the first study that not only compares adolescents with NW, OW, and obesity and their mothers in terms of impulsivity, internalizing symptoms, and disordered eating behaviors but also employs SEM with a relatively large sample size to determine the directionality of these relationships and their impact on adolescents' BMI. We hypothesized that adolescents' impulsivity would indirectly increase their BMI through disordered eating behaviors. However, contrary to our expectations, adolescents' impulsivity was directly associated with BMI, which constitutes one of the main findings of our study. Although the impact of general impulsivity on body weight has been widely recognized (Thamotharan et al., 2013), studies in pediatric samples report inconsistent findings regarding whether this relationship is direct or mediated by other factors (Nederkoorn et al., 2007; Scholten et al., 2014). Neurodevelopmental features associated with heightened impulsivity—such as delayed myelination and extensive synaptic pruning in the prefrontal cortex—are typical characteristics of adolescence (Romer et al., 2017). However, these changes may manifest more prominently at the behavioral level in adolescents with higher weight status, potentially contributing to the heightened impulsivity found in this subgroup. In this context, impulsivity may be linked to weight gain not only through disordered eating behaviors but also through factors such as irregular eating patterns, prolonged screen time, difficulty maintaining regular physical activity, and easier adaptation to peer groups with unhealthy eating habits. In addition, disordered eating behaviors were defined as external eating, restricted eating, and emotional eating in our study. However, disordered eating behaviors are conceptualized on a spectrum, from passive overeating to binge eating and food addiction in the

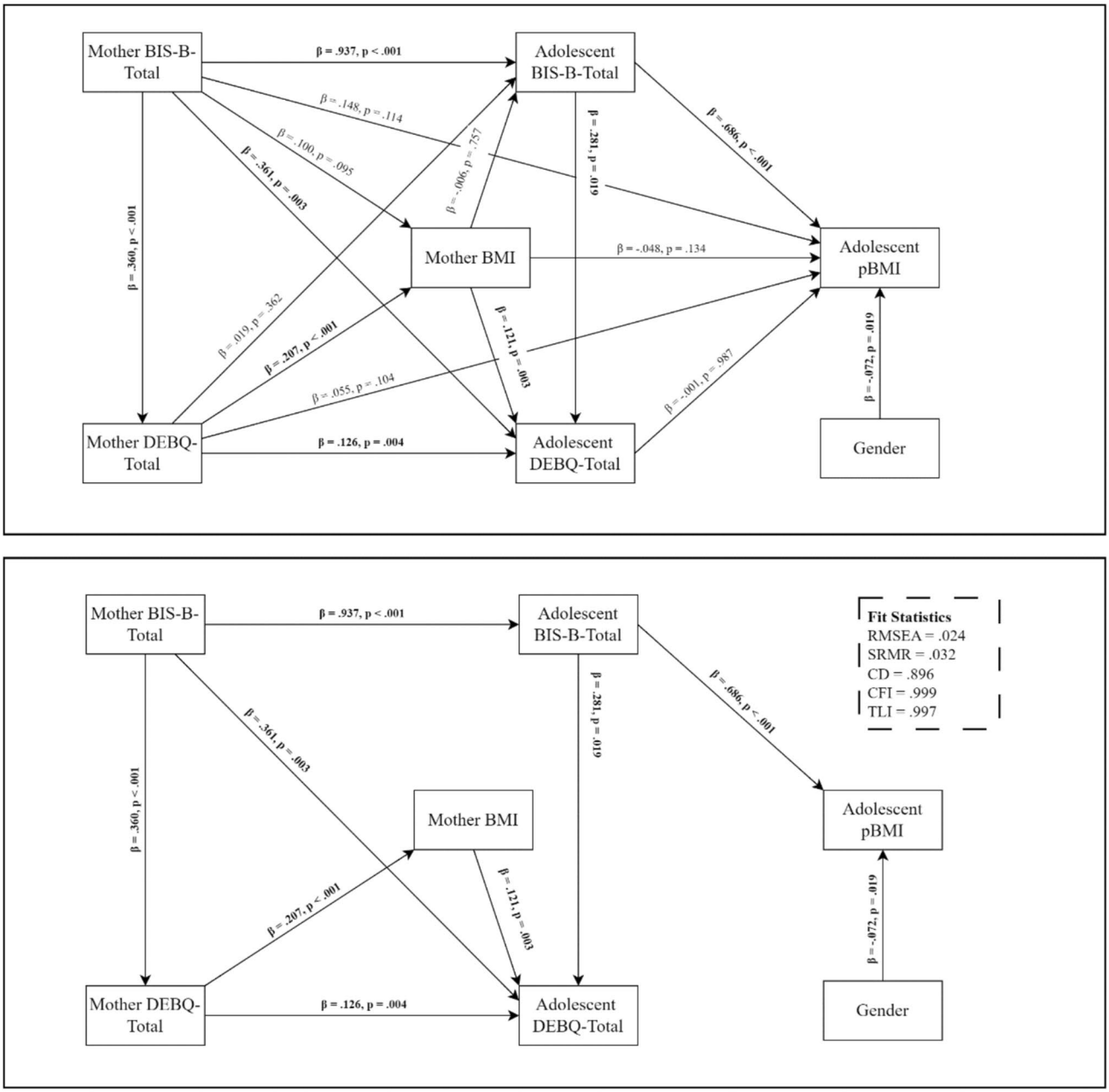

Fig. 3 SEM results before and after removing non-significant paths. Note: The upper panel represents the tested version of the hypothesized model presented in Figure 2, including all specified paths. The lower panel presents the model after removing non-significant paths, along with model fit indices

literature (Davis, 2013). Impulsive people also have a neurobiologically high reward sensitivity, leading to increased motivation toward rewarding stimuli. Consequently, they may be more likely to consume palatable foods regardless of physiological hunger—a pattern strongly associated with binge eating, food cravings, and food addiction (Kidd & Loxton, 2021). This may support another possible explanation for the direct association between impulsivity and BMI. It is also important to emphasize that our findings do not suggest that impulsivity is a universal trait among adolescents but rather a tendency observed specifically in those with higher weight status. Given the multidimensional effect of impulsivity on weight status, future research that examines impulsivity-related factors more comprehensively will contribute to a better understanding of the nature of the relationship between impulsivity and BMI in adolescents with OW and obesity.

We hypothesized that maternal impulsivity would indirectly influence adolescents' BMI by increasing their impulsivity levels. Contrary to our hypothesis, maternal BMI and

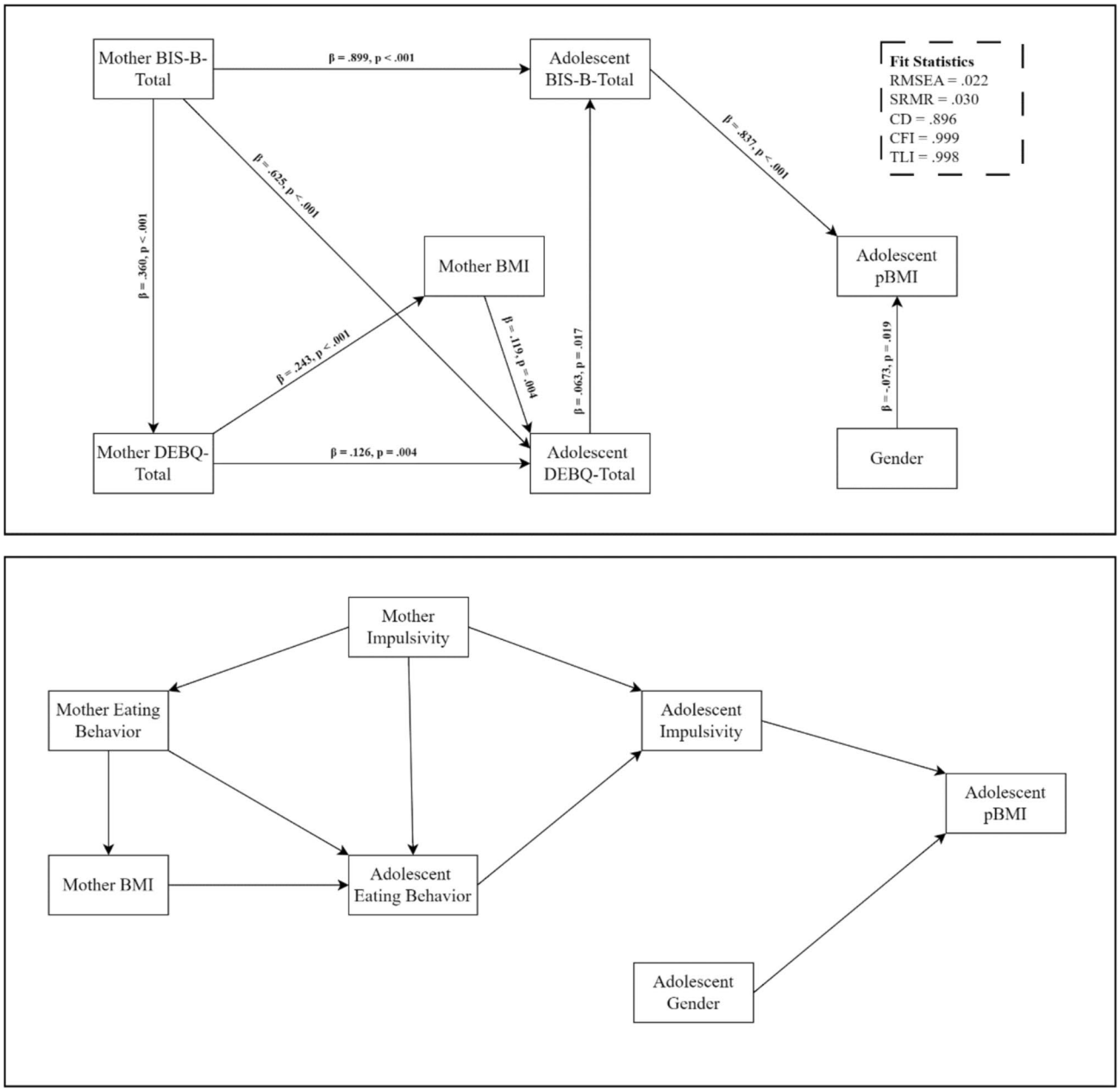

Fig. 4 SEM results: alternative and final model testing the ındirect pathway between adolescent DEBQ-total and pBMI. Note: The upper panel presents an alternative version of the model depicted in Fig. 3, testing the presence of an indirect pathway between Adolescent DEBQ-Total and pBMI, in place of the non-significant direct association. Model fit indices are provided, indicating an excellent model fit, with slightly improved indices compared to the model in Fig. 3. The lower panel illustrates final model, summarizing the relationships between maternal and adolescent eating behaviors, impulsivity, and BMI

impulsivity were indirectly associated with adolescents' BMI through adolescents' disordered eating behaviors, representing another important finding of our study. Numerous studies have shown that maternal obesity is associated with an increased risk of OW and obesity in children and adolescents (Bahreynian et al., 2017; Heslehurst et al., 2019; Svensson et al., 2011). This association is largely attributed to epigenetic mechanisms, as those with a family history of obesity are more likely to inherit genetic predispositions that increase vulnerability to excessive weight gain. In addition to genetic factors, parents with OW and obesity may also contribute to an obesogenic environment, characterized by high-calorie, low-nutrient food consumption, irregular mealtimes, and low levels of physical activity, affecting not only themselves but also their children (Brophy et al., 2009; Mahmood et al., 2021). This association may be more pronounced in cultures with traditional family structures, such as ours, where mothers shape the home food environment

and food holds cultural significance beyond nutrition (e.g., leaving food on the plate may be seen as disrespectful). This dynamic may promote disordered eating behaviors in adolescents through modeling and learned behaviors, potentially increasing the likelihood of OW and obesity.

Maternal impulsivity, along with maternal BMI, may be associated with disordered eating behaviors in adolescents through several mechanisms. Impulsive decision-making may lead to inappropriate parenting practices such as shaming, yelling, or physical discipline, which may increase family stress (Friedman et al., 2016). This negative emotional climate may impair adolescents' emotion regulation (Rabinowitz et al., 2018), making them more likely to engage in eating as a maladaptive coping strategy (Esen Öksüzoğlu et al., 2024). In addition, impulsive mothers may exhibit inconsistent parenting styles, alternating between strict control and excessive permissiveness. This unpredictability may create confusion about eating rules at home, disrupt adolescents' eating patterns, and promote disordered eating behaviors (Chen & Johnston, 2007). According to our findings, maternal impulsivity also indirectly affects maternal BMI through the mediating role of maternal disordered eating. These results highlight that mothers may indirectly affect their children's weight status through their own eating behavior, weight, and impulsivity. In this context, a better understanding of the role of mothers in adolescent eating behavior may be important for the development of parental awareness and regulatory programs.

We hypothesized that adolescents' impulsivity, internalizing symptoms, gender, and maternal impulsivity would be important predictors of adolescents' BMI. Consistent with our hypothesis, both adolescents' impulsivity and gender emerged as significant predictors. The literature consistently shows that females are at higher risk for obesity. Physiologically, females tend to have more total body fat, primarily stored in subcutaneous tissue with low metabolic activity due to estrogen, which makes fat loss more difficult. In addition, leptin resistance—despite higher leptin levels—may increase vulnerability to weight gain. In contrast, optimal levels of testosterone appear to play a protective role against obesity by reducing visceral fat accumulation and supporting insulin sensitivity in males (Saad et al., 2012). Sociocultural factors also play an important role. While female body image is mostly associated with thinness, increasing weight gain during adolescence can exacerbate the gap between perceived and ideal body image, leading to body dissatisfaction. Furthermore, social pressure, appearance-related criticism, and body norms shaped by social media—often directed at females—may trigger disordered eating behaviors. In contrast, males tend to face less direct pressure in this context. This difference may be partly explained by cultural norms that associate masculinity more closely with strength and functionality than with adherence to aesthetic standards (Koceva et al., 2024). Considering these factors, particularly female adolescents, require targeted interventions that address both metabolic and psychological aspects of weight regulation.

We hypothesized that adolescents with OW and obesity and their mothers would have higher levels of internalizing symptoms, impulsivity, and disordered eating behaviors compared to those in the NW group. As we expected, in addition to the previous findings on impulsivity, adolescents with OW and obesity and their mothers also had higher levels of disordered eating behaviors and internalizing symptoms compared to those in the NW group. This is particularly relevant in the context of social cognitive theory, which highlights the role of parents—especially mothers—as key models for gender-specific behaviors. Mothers' thoughts, attitudes, and behaviors regarding food, body image, and weight influence adolescents through both observational learning and explicit/implicit communication (Bandura et al., 1963). In support of this, one study found that adolescent females who perceived maternal pressure to be thin reported greater desires for thinness and more restrictive eating behaviors (Field et al., 2005). Another study concluded that mothers' verbal comments and behavioral cues were shown to play a significant role in the intergenerational transmission of eating disorders (Arroyo et al., 2017). These findings highlight the importance of increasing parental awareness to promote healthy eating habits and body image among adolescents.

This study also found that adolescents with higher weight status and their mothers reported higher levels of internalizing symptoms than the NW group, despite the absence of a clinical psychiatric diagnosis. Psychosocial stressors related to obesity (e.g., peer victimization, body dissatisfaction) may contribute to emotional distress at a subclinical level in adolescents (Pryor et al., 2016). Similarly, mothers may experience emotional distress due to concerns about their child's health, social judgment, or family dynamics. At the same time, maternal psychological status may also influence the adolescent's emotional well-being (Dimitratos et al., 2022). Given the cross-sectional nature of this study, it is not possible to determine the directionality of this relationship; however, the findings suggest a potential bidirectional relationship that warrants further investigation. In this context, adolescents with higher weight status and their mothers may be considered a more psychologically vulnerable group.

Despite the important findings of the study, several limitations should be considered. The cross-sectional design prevents the establishment of causal relationships, even with the use of advanced statistical analysis (SEM). Therefore, any directional interpretations regarding the relationships

between variables should be made with caution. Future longitudinal research is needed to better understand how these variables influence each other over time. In addition, the selection of the sample from a specific geographical region may limit the generalizability of the findings. Despite highlighting the effect of impulsivity on adolescent weight status, it should be acknowledged that other unmeasured variables — such as dietary habits, physical activity levels, sleep patterns, and socioeconomic status — may also contribute to adolescents' weight outcomes. Only mothers were included in the study, as they represented the majority of accompanying caregivers during data collection. This may partly reflect the fact that mothers are still commonly seen as the primary caregivers in our cultural context. Future studies should consider the potential influence of fathers or other primary caregivers on adolescents' weight-related outcomes. Finally, as impulsivity and disordered eating behaviors are multidimensional constructs, the self-report measurements used in the study may not have adequately represented all subcomponents of these constructs. Future studies that address these limitations are expected to provide deeper insights into the complex nature of overweight and obesity in adolescents.

Conclusion and ımplications for future research

This study aimed to identify factors predicting adolescent BMI by comparing impulsivity, internalizing symptoms, and disordered eating behaviors in adolescents with NW, OW, and obesity and their mothers. The results showed that adolescents' impulsivity directly predicted their BMI, while mothers' own weight status and impulsivity were indirectly associated with adolescents' disordered eating behaviors. In addition, adolescents' impulsivity and gender were found to be the most important predictors of their BMI. These findings highlight that individual risk factors, in addition to parental eating behaviors and psychological characteristics, play an important role in the adolescents with obesity. In this context, involving family members in the clinical management would be an important step to improve the effectiveness of treatment and prevention interventions in adolescents with higher weight status. Furthermore, given the direct impact of impulsivity on body weight status, intervention programs aimed at improving cognitive control and promoting self-regulation skills will not only help to regulate eating behavior but also support long-term weight management. Ultimately, female adolescents may require targeted interventions that address both metabolic and psychological aspects of weight regulation.

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Data availability The datasets analyzed during the current study are available from the corresponding author upon reasonable request.

Declarations

Conflict of interest The authors declare no conflict of interest.

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