Childhood traumatic experiences and addiction-like eating behaviors: the mediating roles of attachment, mentalization, and emotional eating

Alessandro Alberto Rossi[1,2]* and Stefania Mannarini[1,2]

Abstract

Introduction Childhood traumatic experiences (CTEs) represent a significant vulnerability factor for addiction-like eating behaviors, yet the underlying developmental pathways remain poorly understood. According to infancy research and attachment theory, this study tested a comprehensive mediation model examining how CTEs contribute to addiction-like eating patterns (i.e., appetite drive and low diet control) through attachment insecurity, impaired reflective functioning, and emotional eating.

Method A cross-sectional study was conducted on a conventional non-clinical sample 1,014 Italian adults. Participants completed a set of validated and standardized scales. Structural equation modeling with latent variables and bootstrap resampling (10,000 iterations) was employed to test the hypothesized model.

Results The model demonstrated adequate fit and supported all hypotheses. CTEs significantly predicted both attachment anxiety, attachment avoidance, and impaired reflective functioning. Attachment anxiety, but not avoidance, mediated the relationship between CTEs and impaired reflective functioning. Also, impaired reflective functioning significantly predicted emotional eating, which in turn predicted both appetite drive and low dietary control. The complete mediation pathway was statistically significant, explaining 53.5% of variance in appetite drive and 20.6% in dietary control.

Discussion These findings provide the first empirical support for a trauma-based developmental model of addiction-like eating behaviors. The results highlight the central role of attachment anxiety and impaired reflective functioning in linking early relational trauma to emotional eating and food addiction-like patterns. Clinical implications suggest that mentalization-based interventions targeting attachment insecurity and emotion regulation may be particularly beneficial for individuals with trauma histories and problematic eating behaviors.

Plain English Summary
People who experienced difficult or harmful relationships with caregivers during childhood, such as abuse or neglect, are more likely to develop unhealthy eating patterns that resemble addiction. This study explored how

*Correspondence:
Alessandro Alberto Rossi
a.rossi@unipd.it

Full list of author information is available at the end of the article

this happens. We surveyed over 1,000 Italian adults and found that childhood trauma creates a chain reaction. First, these early negative experiences often lead people to feel anxious in their relationships and struggle to understand their own emotions and those of others. When people cannot recognize or manage their feelings well, they may turn to food for comfort, eating to cope with stress rather than hunger. Over time, this emotional eating can develop into addiction-like behaviors, including intense cravings for certain foods and difficulty controlling how much they eat. Our findings suggest that treating problematic eating patterns requires addressing more than just food and diet. Therapies that help people better understand their emotions, improve their relationships, and develop healthier ways to manage stress may be especially helpful for those who experienced childhood trauma. Understanding these connections can lead to more effective support for people struggling with food-related difficulties.

Keywords Food addiction, Addiction-like eating behaviors, Childhood traumatic experiences, Attachment anxiety, Mentalization, Low diet control, Eating addiction

Introduction

Disordered eating behaviors related to excessive food intake and overeating include a range of maladaptive patterns triggered by dysfunctional responses to food-related stimuli [1]. These behaviors have garnered increasing clinical interest because of their strong links with overweight and obesity [2], which are chronic conditions shaped by complex medical, environmental, and psychological influences [3]. Studies have described different forms of excessive food consumption and overeating, from compulsive grazing to binge eating episodes [4–6]. These patterns often share transdiagnostic features [7] and symptoms suggestive of food-related addictive processes, which may underlie and perpetuate maladaptive (over)eating behaviors [8].

These disordered eating behaviors are frequently associated with the repeated overconsumption (i.e., consuming food in quantities beyond physiological needs or intended amounts) of ultra-processed foods (UPFs). These foods are highly palatable and can trigger addiction-like eating responses and symptoms in certain individuals—particularly those with vulnerabilities related to reward sensitivity, emotion dysregulation, or early adverse experiences—which is commonly referred to as food addiction (FA) [9–12]. UPFs rich in sugar, salt, and fat [13–15] have been shown to contribute to the onset and persistence of overweight and obesity, partly because of their addictive properties [16–18] and their capacity to engage neural reward systems [19–21] and activate brain circuits [22] involved in addictive behavior and substance-related and addictive disorders (SRADs).

Thus, FA not only overlaps with transdiagnostic core features of disordered eating behaviors and eating disorders (EDs) but also exhibits traits common in SRADs, creating a self-reinforcing and cyclical pattern [23, 24].

Indeed, individuals with FA develop addiction-like symptoms that are strongly polarized toward food. They experience tolerance, whereby increasing quantities of food—particularly UPFs and palatable ones—are needed to achieve the same level of satisfaction [25] or emotional relief [4, 23]. Additionally, individuals with FA may experience withdrawal-like symptoms, such as irritability, restlessness, and intense food cravings [25, 26]. These symptoms can trigger intrusive thoughts and compulsive eating behaviors (i.e., emotional eating) [8] aimed at restoring the pleasurable sensations associated with food consumption [4, 5], often resulting in uncontrolled eating episodes [8, 27], such as grazing or binge eating. Indeed, core features of FA include heightened appetite drive—characterized by increased responsiveness to food-related cues and tendencies toward overeating—as well as reduced dietary control, marked by difficulty regulating food intake and adhering to structured eating patterns [4, 5, 27, 28]. These manifestations reflect the persistent cognitive and behavioral dysregulation around food [8] that characterizes FA. These manifestations are not limited to binge eating but span a spectrum of maladaptive eating behaviors [23, 24] that compromise self-regulation [27, 29, 30]. Furthermore, FA symptoms are frequently observed across different ED diagnoses [23, 31, 32], suggesting that FA may operate as a transdiagnostic construct [33–36]. This finding reinforces the complex and dual clinical nature of FA: on the one hand, it involves neurobiological and behavioral features akin to those of SRADs [22]; on the other hand, it encompasses the psychological and emotional processes commonly associated with EDs [8, 27].

Consequently, given the dual nature of FA, it is plausible that FA may share etiological pathways with both SRADs and EDs [27, 37]. Although various risk factors and triggering cues have been identified in the literature [38, 39], their specific roles and the way they interact are still not entirely clear. Moreover, among the factors implicated in both conditions, childhood traumatic experiences (CTEs)—including emotional, physical, and sexual abuse, as well as emotional and physical neglect occurring within the caregiving context [40]—have emerged as a significant and shared vulnerability [41–48].

Within the theoretical framework of infancy research and attachment theory, CTEs (particularly emotional

maltreatment) have been consistently linked to disordered eating behaviors [27, 48–52]. The literature indicates that such early traumatic adversities are consistently associated with increased vulnerability to dysfunctional eating patterns [27, 49, 53], as well as to FA [27, 54, 55]. Although the body of research is still developing, current findings point to a relationship between CTEs, especially emotional maltreatment, and the emergence of FA symptoms [56–61] and addiction-like eating behaviors [27], with more severe trauma often linked to greater symptom intensity [62].

Consistent with attachment theory, CTEs are known to undermine the development of secure attachment patterns [49, 63, 64], often leading to relational insecurities (e.g., attachment anxiety and attachment avoidance) and increased vulnerability to psychopathologies, such as SRADs and EDs [44, 49, 65]. According to attachment theory, CTEs can interfere with the development of the attachment system by undermining the caregiver's role as a secure base and disrupting the formation of secure attachment bonds [42, 66], potentially leading to difficulties in self-regulation and a fragmented self-concept [30, 67]. These outcomes are often associated with emotionally unresponsive or misattuned caregiving, which impairs the child's ability to use the caregiver as a secure base—a core component of attachment security [30, 68]. Traumatic interactions with caregivers during childhood shape internal working models—mental representations of the self, others, and interpersonal relationships—that influence emotional regulation, self-perception, and behavior across the lifespan, including into adulthood [49, 69]. In adulthood, attachment is typically conceptualized along two dimensions: anxiety and avoidance [70, 71]. Attachment anxiety reflects a negative view of the self and a heightened need for closeness, often accompanied by a fear of rejection. In contrast, attachment avoidance involves a positive self-view paired with mistrust of others and discomfort with intimacy [70, 72]. Both forms of attachment insecurity have been implicated in disordered eating behaviors, EDs, and SRADs, particularly through their impact on emotion regulation strategies [73, 74]. Moreover, although findings show some variability in the relative contributions of attachment anxiety and avoidance, attachment anxiety is generally considered a stronger risk factor for the development of both substance use behaviors and dysfunctional eating patterns related to overeating [74] and, thus, likely addiction-like eating behaviors. Importantly, the literature suggests that attachment insecurities may mediate the relationship between CTEs and eating behaviors [49, 63, 64]. In the presence of attachment insecurity, higher rates of early abuse and neglect have been observed among individuals with disordered eating behaviors [64] and in nonclinical samples [49], supporting a mediating role for attachment.

Specifically, a study by Musetti and colleagues (2023) revealed that patterns of attachment insecurity mediate the association between CTEs and disordered eating behaviors via mentalization (i.e., impaired reflective functioning); however, only attachment anxiety significantly contributes [49].

Consistent with these findings, the literature shows that attachment and mentalization are deeply interconnected [75]. In fact, relational disruptions can impair the development of fundamental interpersonal functions, such as reflective functioning [42, 76, 77].

According to Fonagy, the construct of mentalization, often referred to as reflective functioning, is a capacity rooted in early child–caregiver interactions [78, 79]. Reflective functioning refers to the ability to understand and interpret one's own and others' behaviors by linking them to underlying mental states [80] such as thoughts, emotions, and intentions [75, 81]. This function allows individuals to attribute meaning to actions and experiences without relying solely on external cues or validation [80, 81]. For instance, adequate reflective functioning enables individuals to recognize and understand the emotional origins of their eating behaviors, such as identifying stress as the driver of an eating urge rather than physiological hunger. In contrast, impaired reflective functioning (hypomentalization) results in automatic, impulse-driven eating without awareness of the underlying emotional triggers.

Indeed, when reflective functioning is impaired, such as in the presence of CTEs or attachment insecurities, this crucial interpersonal ability becomes compromised [81]. Individuals with impaired reflective functioning (i.e., hypomentalization) operate automatically and unconsciously, hindering their capacity to interpret and regulate behavior [82], especially in emotionally intense situations [83]. Such impairment leads to a rigid and distorted perception of mental states, causing individuals to experience reality in a fragmented, prementalized way [42, 84], where internal emotions feel overwhelming and disorganizing [49, 81, 85, 86]. Consequently, to defend against these distressing thoughts and emotions [82, 87, 88], individuals may rely on maladaptive coping mechanisms [89, 90] or external regulators [84, 86, 91], such as substance use or food (i.e., UPFs), that provide an immediate response and relief [68, 76]. Research highlights hypomentalization as a key characteristic among those with disordered eating behaviors [27, 29, 49, 84]. Consequently, when faced with aversive internal states such as anxiety or intrusive thoughts, individuals with low reflective functioning often engage in impulsive, emotionally driven eating behaviors [81, 89, 90, 92], commonly known as emotional eating [93, 94].

As psychological stress or arousal intensifies, particularly following traumatic or highly stressful events, milder

coping strategies such as emotional eating often become insufficient [8, 27, 91]. Individuals may then resort to more severe, maladaptive behaviors, including compulsive, repetitive, or binge eating, as attempts to suppress or numb overwhelming negative emotions [8, 27, 42, 88, 95, 96]. This escalation typically leads to diminished control over eating and diet and fosters addiction-like eating patterns [27].

In summary, individuals exposed to CTEs may develop insecure attachment styles linked to impairments in reflective functioning (hypomentalization) [49, 81, 85]. These deficits in self-regulation [29, 30] require an external object to restore internal emotional regulation, such as food [49, 84, 86], which thus triggers addiction-like eating behaviors [27], including emotional eating, overeating, and loss of control over one's diet.

However, to the best of our knowledge, this model has never been tested before. Consequently, robust empirical confirmation is lacking. Indeed, despite the extensive literature, the relationships among CTEs, attachment insecurity, impaired reflective functioning (hypomentalization), and addiction-like eating behaviors remain unclear.

Consequently, on the basis of the abovementioned scientific literature, the aim of this study is to test a multiple mediation model in which CTEs (X) are associated with attachment anxiety (M1a), attachment avoidance (M1b), and hypomentalization (M2). In turn, hypomentalization (M2) should be associated with emotional eating (M3). Finally, emotional eating should be simultaneously associated with addiction-like eating behaviors such as overeating (Y1) and reduced dietary control (Y2). Explicit hypotheses about each path (relationship) between variables were formulated:

H1: CTEs, attachment anxiety, attachment avoidance, hypomentalization, emotional eating, overeating, and reduced dietary control are positively correlated with each other.

H2: CTEs statistically predict emotional eating via attachment anxiety, attachment avoidance and hypomentalization.

H3: CTEs statistically predict overeating behaviors and reduced dietary control via attachment anxiety, attachment avoidance, hypomentalization and emotional eating.

In other words, it was hypothesized that the positive associations between CTEs and addiction-like eating behaviors would be mediated by the presence of insecure attachment, hypomentalizing, and emotional eating.

Methods and materials
Procedure
Participants were recruited from the general population—a convenience sample—through snowball sampling [97] via social media platforms such as Facebook and Twitter/X. The inclusion criteria were as follows: (A) aged 18 years or older; (B) native Italian speaker; (C) full completion of the assessment battery (only compete submission were recorded; namely, no missing data); and (D) a total completion time between 10 and 20 min. In particular, this final criterion was established through a pilot study conducted with 20 participants (not included in the final sample) to assess the clarity of the questionnaires and determine an appropriate completion time. This approach aimed to prevent excessively fast responses (which could indicate random or automatic answers, or bots and/or imposters), or overly slow responses (for example, if the participant had been interrupted or was distracted) [98].

Moreover, in line with current recommendations, data quality screening was conducted prior to statistical analyses to ensure the validity of the online responses [98]. In the preliminary instructions for accessing the study, the participants were clearly informed that they would not receive any form of compensation.

All the participants took part voluntarily and all participants provided informed consent before accessing the survey. The study was approved by the Ethics Committee of the University of Padua (protocol no. 547-a) and was conducted in accordance with the institution's ethical standards.

Sample size determination
The required sample size was estimated through Monte Carlo simulation, an approach particularly suited for structural equation models involving multiple latent variables and indirect effects [99, 100]. The simulations were conducted in R via the lavaan package [101], following established recommendations for SEM power analysis [102, 103]. For each candidate sample size, ranging from 400 to 1200 in increments of 50, 1000 datasets were generated on the basis of population parameters consistent with theoretical expectations and prior research. These included moderate factor loadings, structural paths and indirect effects of realistic magnitude (small-to-medium, approximately 0.15–0.30) across mediators and outcomes. Power was defined as the proportion of replications in which at least 80% of the key structural coefficients reached significance at $p < 0.05$, a criterion that balances statistical sensitivity with model complexity. To ensure estimation stability, a minimum convergence rate of 90% was also needed. The estimation relies on maximum likelihood (ML), which is quite robust to violations of multivariate normality [104]. On the basis of these criteria, the simulation indicated that a minimum sample size greater than 800 participants would be sufficient for correctly estimating model parameters.

Participants

The sample comprised 1014 participants. The sample included 138 males (13.6%) and 876 females (86.4%), aged between 18 and 74 years (*mean* = 44.11, *SD* = 13.70), with a BMI ranging from 16.14 to 47.75 kg/m^2 (*mean* = 24.71 kg/m^2, *SD* = 4.98). More details are provided in Table 1.

Measures

Childhood trauma questionnaire–short form (CTQ-SF)

The CTQ-SF [40] is a self-reported measure designed to assess traumatic childhood experiences. It includes 28 items rated on a 5-point Likert scale ranging from 1 (= "*Never true*") to 5 (= "*Very often true*"). Of these, 25 items are grouped into five subscales that assess different domains of maltreatment, namely, emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect, with each domain represented by five items. The remaining three items function as a minimization/denial scale, capturing the tendency to under-report traumatic events. A total score reflecting overall childhood trauma can be obtained by summing the subscale scores. Higher scores indicate a greater presence of childhood traumatic experiences. In the present study, the Italian version of the CTQ-SF was used [105], which demonstrated excellent internal consistency, with McDonald's omega = 0.925.

Relationship questionnaire (RQ)

The RQ [72] is one of the most commonly used self-report measures for evaluating adult attachment styles [70, 71]. Individuals rate how well four brief descriptions reflect their typical relationship style, using a 7-point Likert scale ranging from 1 (= "strongly disagree") to 7 (= "strongly agree"). Each description corresponds to one of the four prototypical adult attachment styles: secure (positive view of self and others), dismissing (positive view of self, negative view of others), preoccupied (negative view of self, positive view of others), and fearful (negative view of both self and others). In line with the standard scoring procedure [72], the two core dimensions of insecure attachment—anxiety and avoidance—were derived by combining responses to the four prototypes of anxiety [(fearful + preoccupied) - (secure + dismissing)] and avoidance [(fearful + dismissing) - (secure + preoccupied)]. As these dimensions represent computed scores rather than multi-item scales, traditional internal consistency estimates (e.g., McDonald's omega) are not applicable. Higher scores indicate greater levels of either anxious or avoidant attachment, depending on the dimension assessed. Higher scores indicate greater levels of either anxious or avoidant attachment, depending on the dimension assessed. For the present study, the Italian version of the RQ was used [106].

Reflective functioning questionnaire (RFQ)

The RFQ [107] is a brief self-report questionnaire designed to assess a specific mentalization referred to as reflective functioning, namely, the capacity to interpret and understand one's own and others' mental states [81]. It comprises eight items rated on a 7-point Likert scale ranging from 1 (= "*completely disagree*") to 7 (= "*completely agree*"), where respondents express their agreement with statements related to mentalizing processes. The questionnaire captures two contrasting dimensions: certainty (i.e., hypermentalizing—excess reflective functioning) and uncertainty (i.e., hypomentalizing—lack of reflective functioning) about mental states. In this study, the uncertainty subscale was used, which reflects impairments in reflective functioning. Higher scores

Table 1 Sample descriptive statistics

	Descriptives (*N* = 1014)	
Age (*mean*, *SD*)	44.11	13.70
BMI (Kg/m^2) (*mean*, *SD*)	24.71	4.98
Sex (*n*, %)		
Male	138	13.6%
Female	876	86.4%
Education (*n*, %)		
Middle school	98	9.7%
High school	495	48.8%
Bachelor	345	34.0%
Master degree/PhD	76	7.5%
Work status (*n*, %)		
Student	114	11.2%
Full-time worker	534	52.7%
Entrepreneur	208	20.5%
Unemployed	87	8.6%
Retired	71	7.0%
Civil status (*n*, %)		
Single	164	16.2%
In a relationships	280	27.6%
Married	473	46.6%
Separated/divorced	80	7.9%
Widowed	17	1.7%
BMI class (*n*, %)		
Underweight (16–18.49)	46	4.5%
Normal weight (18.5–24.99)	563	55.5%
Overweight (25–29.99)	266	26.2%
Class I obesity (30–34.99)	89	8.8%
Class II obesity (35–39.99)	40	3.9%
Class III obesity (> 40)	10	1.0%
Eating disorder (*n*, %)		
No ED	912	89.9%
Anorexia nervosa (AN)	23	2.3%
Bulimia nervosa (BN)	12	1.2%
Binge eating disorder (BED)	36	3.6%
Otherwise specified feeding and eating disorder (OSFED)	31	3.1%

indicate greater difficulty in understanding mental states. The Italian version of the RFQ [108] was used, and it showed good internal consistency, with McDonald's omega = 0.772.

Three-factor eating questionnaire-revised 18 (TFEQ-R-18)

The TFEQ-R-18 [109] is a widely used self-report measure designed to evaluate three primary dimensions of eating behavior: cognitive restraint (CR), uncontrolled eating (UE), and emotional eating (EE). The questionnaire comprises 18 items rated on a 4-point Likert scale, with higher scores indicating stronger tendencies in the corresponding domain. The CR subscale assesses individuals' efforts to restrict food intake to manage body weight or shape. The UE subscale reflects a loss of control over eating, often resulting in episodes of overeating. The EE subscale captures the propensity to eat in response to negative emotional experiences. The present study employed the Italian version of the TFEQ-R-18 [110] and only the EE dimension, which is in line with the study objectives. The EE scale demonstrated good internal consistency: McDonald's omega = 0.888.

Addiction-like eating behavior scale (AEBS)

The AEBS [111] is a 15-item self-report instrument developed to assess addictive patterns of eating behavior, namely, food addiction–like behaviors. The items are rated on a 5-point Likert scale ranging from 1 (= "*strongly disagree/never*") to 5 (= "*strongly agree/always*"). The scale includes two dimensions: appetite drive (AD), which reflects food addiction–related behaviors such as heightened responsiveness to food-related rewards and tendencies toward overeating, and low dietary control (LDC), which captures reduced self-regulation in managing eating impulses. Higher scores on each subscale indicate more pronounced difficulties in the respective area. Additionally, a general score can be computed. The AEBS has shown good psychometric properties in evaluating maladaptive eating patterns. In the present study, the validated Italian version of the AEBS [112] was used, with both dimensions demonstrating excellent internal consistency: McDonald's omega = 0.879 and McDonald's omega = 0.857 for AD and LDC, respectively.

Statistical analysis

Statistical analyses were performed via R software. According to the predefined inclusion criteria, none of the observations provided missing values. Consistent with prior research, a series of preparatory steps were completed before the structural equation model (SEM) was estimated [113, 114]. Moreover, as SEM relies on the general linear model framework, its underlying assumptions were examined and found to be met.

First, as preliminary analyses, Pearson correlation coefficients (r) were calculated to examine the strength of the relationships among variables and identify potential issues. Correlation coefficients were interpreted via Cohen's benchmarks [115]: $r < 0.10$, trivial; r from 0.10 to 0.30, small; r from 0.30 to 0.50, moderate; and $r > 0.50$, large. Correlations exceeding $|0.80|$ were considered indicative of potentially problematic overlaps [116].

Second, the factorial structure (i.e., measurement model) of each questionnaire was assessed via confirmatory factor analysis (CFA), with the aim of verifying whether the observed data conformed to the theoretical structure established in previous validation studies. The scales tested included the CTQ-SF, the RFQ uncertainty, and the AEBS. Importantly, the factorial structure was not tested for the RQ scale. Indeed, consistent with the original validation study, a factorial structure for the RQ cannot be meaningfully evaluated. CFAs were performed via the diagonally weighted least squares (DWLS) estimator [117, 118]. Model adequacy was evaluated via fit indices and their thresholds [118–120]: (A) a nonsignificant χ^2 test ($p > 0.05$) was desirable; (B) RMSEA values below 0.08 indicated acceptable fit; (C) CFI values above 0.90 suggested a satisfactory fit; and (D) SRMR values below 0.08 were interpreted as indicative of good model fit.

Third, Harman's single-factor test was conducted [121] to assess the potential presence of common method bias, namely, whether a single latent factor accounts for the majority of the covariance among observed variables. First, a model with correlated latent factors was specified, reflecting the theoretical structure of the included scales. Next, a constrained model was tested in which all the items were loaded onto a single latent factor. The two models were then compared using established thresholds for changes in fit indices: a non-significant change in χ^2 ($\Delta\chi^2$, $p > 0.05$), along with ΔCFI ≤ 0.01 and ΔRMSEA ≤ 0.015, was used to assess model deterioration [122]. These comparisons followed standard interpretive criteria [122]. A significantly better fit of the correlated-factor model over the single-factor model would indicate that common method bias is unlikely to be a concern.

Fourth, latent variables were represented via item parcels as indicators. This approach offers several psychometric benefits, including improved reliability and a reduced likelihood of violating assumptions such as normality [123]. A partially disaggregated parceling strategy was adopted, following established guidelines [124]. Specifically, for the majority of the scales ('RFQ uncertainty', 'AD', and 'LDC'), parcels were constructed via the 'item-to-construct balance' method [123, 125]. For the CTQ-SF, parcels were created via the 'domain-representative strategy' [123, 125]. For each latent construct, a minimum of three parcels was created to ensure that the model was at least just identified [118, 123, 126]. After parcel creation,

descriptive statistics were checked to ensure that the parcel scores did not display excessive skewness (Sk >|3|) or kurtosis (K >|10|), in line with recommended thresholds [118]. No item parcels were created for the EE scale, as it consists of only three items. Finally, considering that RQ is composed of two single items/dimensions (i.e., attachment anxiety and attachment avoidance, see dedicated section), two single-item latent variables were created via the reliability correction approach [124, 127, 128], preserving its theoretical meaning. In accordance with the procedure of Jorgensen [129] and following established recommendations [124, 127, 128], the residual variance of each single-item indicator was fixed to a value equal to the items' total variance multiplied by 1-α (α = item internal consistency). Alpha (α) was established a priori (0.75)[1] according to established and conventional recommendations for single-item reliability [130–132].

Fifth, an SEM with latent variables was estimated, incorporating a predictor, four mediators, and two outcome variables [126, 133]. Specifically, childhood trauma (X) was modeled as a predictor of addiction-like eating behaviors (Y1) and low diet control (Y2), with attachment anxiety (M1a), attachment avoidance (M1b), reflective functioning (M2) and emotional eating (M3) serving as mediating variables (see Fig. 1). Sex and BMI were used as covariates. The model was estimated via the maximum likelihood (ML) estimator. Given that not all parcel indicators followed a perfectly normal distribution, a nonparametric bootstrapping procedure with 10,000 resamples and Bollen–Stine correction was applied [118,

134, 135]. Model fit was assessed via the standard set of fit indices (χ^2, RMSEA, CFI, and SRMR) and their conventional thresholds [118, 120]. All regression coefficients (β) presented in the results are unstandardized.

Results

Preliminary analysis

Correlation analyses revealed statistically significant linear associations of varying strength—from small to large—among the psychological variables included in the mediation model. Childhood trauma showed moderate positive associations with both attachment anxiety ($r = 0.315$) and hypomentalizing ($r = 0.241$) and a weaker correlation with attachment avoidance ($r = 0.156$). It was also positively associated with all the eating-related variables (i.e., emotional eating, appetite drive, and low diet control), with correlations ranging from $r = 0.205$ to $r = 0.347$. Attachment anxiety was positively correlated with hypomentalizing ($r = 0.260$) and with all eating-related variables (r range: 0.188 to 0.245). In contrast, attachment avoidance did not show significant associations with any variables included in the mediation model. Hypomentalizing was significantly associated with all eating-related variables, with correlations ranging from $r = 0.217$ to $r = 0.302$. Moreover, emotional eating, appetite drive, and low diet control showed moderate-to-large intercorrelations, with coefficients ranging from $r = 0.352$ to $r = 0.610$. With respect to BMI, no significant correlations were found with childhood trauma, attachment dimensions, or hypomentalizing, except for a small
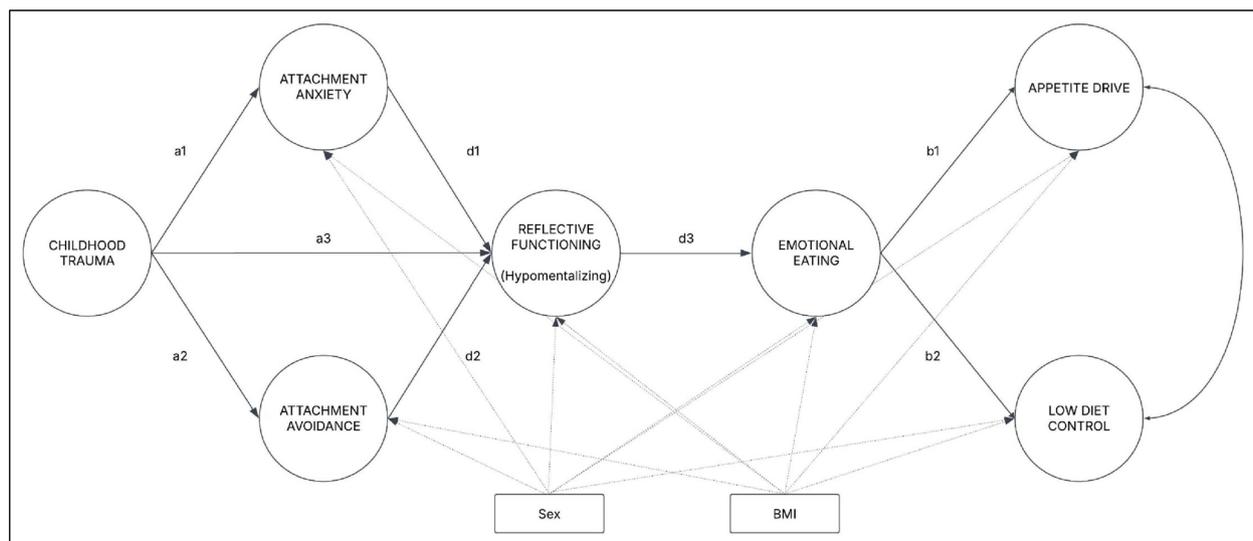
Fig. 1 SEM conceptual representation. Sex and BMI were included as covariates. For the sake of clarity in the presentation of this graph, item parcels and indirect effects (e.g., childhood trauma → appetite drive) were not included; only direct effects were depicted. The circles represent latent variables, whereas the rectangles represent observed variables

---

[1] Different SEMs were tested, systematically varying the assumed alpha coefficient from 1 (i.e., assuming no measurement error) to 0.60. The results did not show substantial changes across the models.

positive association between BMI and hypomentalizing. Moderate positive associations emerged between BMI

and emotional eating, appetite drive, and low perceived diet control. The results are reported in Table 2.

Measurement models—factorial structure analysis

The CTQ-SF (childhood trauma) showed adequate goodness-of-fit indices: χ^2 (270) = 935.216; $p < 0.001$; RMSEA = 0.049; 90% CI [0.046; 0.053]; $p(\text{RMSEA} < 0.05) = 0.622$, CFI = 0.995, SRMR = 0.073. Additionally, the RFQ uncertainty (hypomentalizing) scale showed adequate goodness-of-fit indices: χ^2 (9) = 55.532; $p < 0.001$; RMSEA = 0.071; 90% CI [0.054; 0.090]; $p(\text{RMSEA} < 0.05) = 0.022$, CFI = 0.985, SRMR = 0.068. Finally, the appetite drive and low-diet control scales (AEBS) showed adequate fit indices: χ^2 (74) = 316.806; $p < 0.001$; RMSEA = 0.057; 90% CI [0.051; 0.063]; $p(\text{RMSEA} < 0.05) = 0.037$; CFI = 0.996; SRMR = 0.047. As noted in the statistical analysis section, evaluating the factorial structure of the RQ is inappropriate. Additionally, the emotional eating (EE) subscale of the TFEQ-R-18 consists of only three items, resulting in a saturated model with perfect fit.

Harman's single-factor test

A single-factor test revealed the absence of 'common method bias'. The model with correlated factors provided adequate fit indices: χ^2 (1091) = 4394.998; $p < 0.001$; RMSEA = 0.055; 90% CI [0.053; 0.056]; $p(\text{RMSEA} < 0.05) < 0.001$, CFI = 0.986, SRMR = 0.066. In contrast, the single-factor model provided poor fit indices: χ^2 (1127) = 43431.177; $p < 0.001$; RMSEA = 0.192; 90% CI [0.191; 0.194]; $p(\text{RMSEA} < 0.05) < 0.001$, CFI = 0.820, SRMR = 0.216. The model comparison further suggested the absence of 'common method bias': $\Delta\chi^2$ (36) = 39,036, $p < 0.001$; $|\Delta\text{RMSEA}| = 0.138$, and $|\Delta\text{CFI}| = 0.166$.

Structural equation model with latent variables and item parcels

All item pairs were statistically significantly associated with their latent variable. As reported in Table 3, their standardized factor loadings range from a minimum of 0.316 to a maximum of 0.901. After confirming that the indicators appropriately captured the underlying latent construct, the SEM was examined. Sex and BMI were included as covariates in the SEM.

The hypothesized model (Figs. 1 and 2) provided adequate goodness-of-fit indices: χ^2 (177) = 801.072; $p < 0.001$; RMSEA = 0.059; 90% CI [0.055; 0.063]; $p(\text{RMSEA} < 0.05) < 0.001$, CFI = 0.937, SRMR = 0.040. To keep this section concise, only the sequential paths (e.g., X → M1a, M1a → M2, etc.) are reported here. For a comprehensive overview of the results, please refer to Tables 4 and 5.

Childhood trauma (X) was positively associated with attachment anxiety (M1a), *path a1*: β = 5.253

Table 2 Correlations among variables

|	Descriptives				Correlations								
	M	SD	Sk	K	1	2	3	4	5	6	7	8	9
1 Childhood trauma	37.59	11.53	1.37	2.16	–								
2 Attachment anxiety	−1.18	4.03	0.27	0.18	0.315***	–							
3 Attachment avoidance	0.83	3.65	0.24	0.09	0.156***	0.132***	–						
4 Hypomentalizing	2.95	3.52	1.44	1.80	0.241***	0.260***	0.034	–					
5 Emotional eating	6.69	2.71	0.25	−0.98	0.275***	0.245***	0.020	0.271***	–				
6 AEBS total score	32.38	9.80	0.92	1.02	0.327***	0.226***	−0.001	0.302***	0.571***	–			
7 Appetite drive	16.75	6.19	1.45	2.24	0.347***	0.188***	0.001	0.297***	0.610***	0.888***	–		
8 Low diet control	15.63	5.16	0.26	−0.41	0.205***	0.204***	−0.003	0.217***	0.352***	0.834***	0.487***	–	
9 BMI	24.71	4.98	1.11	1.34	0.052	0.007	−0.046	0.072*	0.346***	0.380***	0.412***	0.227***	–

M mean, *SD* standard deviation, *Sk* skewness, *K* kurtosis. Childhood Trauma = CTQ-SF Total score; Attachment anxiety = attachment anxiety = attachment anxiety dimension of the RQ; Attachment avoidance = attachment avoidance dimension of the RQ; Hypomentalizing = uncertainty scale of the RFQ; Emotional Eating = emotional eating scale of the TFEQ-R-18; Appetite Drive = AD scale of the AEBS; AEBS Total score = total score of the AEBS; Low diet control = LDC of the AEBS; *BMI* body mass index

*** $p < 0.001$; * $p < 0.050$

Table 3 Descriptive statistics and factor loadings (λ) of the item parcels

	Descriptives				Item parcel/latent variable relationship				
	M	SD	Sk	K	λ(se)	z value	p value	λ*	R²
Childhood trauma (X)									
pCTQ#1	1.33	0.43	1.89	4.92	1.000			0.642	0.413
pCTQ#2	1.67	0.81	1.43	1.70	2.476 (0.152)	16.262	<0.001	0.846	0.716
pCTQ#3	2.21	0.92	0.64	−0.27	2.748 (0.184)	14.901	<0.001	0.822	0.676
pCTQ#4	1.17	0.42	3.64	16.85	0.828 (0.084)	9.853	<0.001	0.548	0.301
pCTQ#5	1.15	0.49	4.46	22.65	0.560 (0.095)	5.911	<0.001	0.316	0.100
Attachment anxiety (M1a)									
Att. Anx.	−1.18	4.03	0.27	0.18	1.000			0.866	0.750
Attachment avoidance (M1b)									
Att. Av.	0.83	3.65	0.24	0.09	1.000			0.866	0.750
Hypomentalizing (M2)									
pRFQ#1	0.40	0.65	1.83	3.04	1.000			0.686	0.498
pRFQ#2	0.58	0.79	1.35	1.01	1.243 (0.098)	12.717	<0.001	0.706	0.597
pRFQ#3	0.49	0.69	1.43	1.55	1.185 (0.087)	13.622	<0.001	0.772	0.471
Emotional eating (M3)									
EE#1	2.42	1.03	0.02	−1.17	1.000			0.841	0.707
EE#2	2.25	1.02	0.19	−1.15	1.058 (0.027)	39.272	<0.001	0.901	0.812
EE#3	2.02	0.96	0.47	−0.88	0.888 (0.026)	33.701	<0.001	0.808	0.652
Appetite drive (Y1)									
pAD#1	2.40	0.90	0.43	−0.21	1.000			0.699	0.488
pAD#2	2.12	1.01	0.83	−0.15	1.246 (0.052)	24.049	<0.001	0.770	0.592
pAD#3	1.54	0.71	1.83	3.89	0.971 (0.047)	20.649	<0.001	0.853	0.728
pAD#4	1.54	0.68	1.79	3.52	0.959 (0.042)	22.821	<0.001	0.887	0.786
Low diet control (Y2)									
pLDC#1	2.60	0.88	0.28	−0.28	1.000			0.823	0.677
pLDC#2	2.71	1.02	0.15	−0.73	1.263 (0.042)	30.125	<0.001	0.899	0.809
pLDC#3	2.50	0.96	0.52	−0.21	1.080 (0.041)	26.517	<0.001	0.819	0.671

M mean, *SD* standard deviation, *Sk* skewness, *K* kurtosis, *λ* unstandardized factor loading, *se* standard error, *λ** standardized factor loading, *R2* explained variance, *p(…)* item parcel; childhood trauma = CTQ-SF total score; attachment anxiety = attachment anxiety dimension of the RQ; attachment avoidance = attachment avoidance dimension of the RQ; hypomentalization = RFQ uncertainty scale; emotional eating = TFEQ-R-18 EE scale; appetite drive = AEBS AD scale; low diet control = AEBS LDC
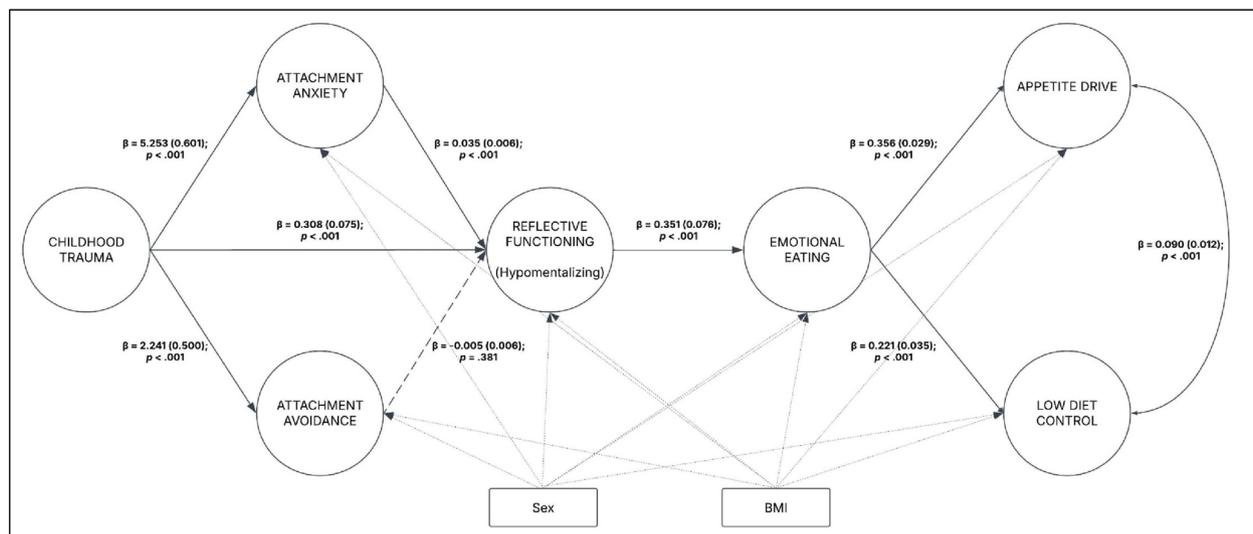
Fig. 2 SEM statistical results. Sex and BMI were included as covariates. For the sake of clarity in the presentation of this graph, item parcels and indirect effects (e.g., childhood trauma → appetite drive) were not included; only direct effects were depicted. The circles represent latent variables, whereas the rectangles represent observed variables. *β* = unstandardized beta; SE = standard error; *p* = *p* value

Table 4 Summary of simple regression parameter estimates (β) with 95% confidence intervals (Fig. 2)

	Path	β*	β (SE)	95%CI [L - U]	z value	p value	R^2
Childhood trauma (X) → Attachment Anxiety (M1a)	a1	0.415	5.253 (0.601)	[4.151; 6.523]	8.741	< 0.001	0.175
Childhood trauma (X) → Attachment Avoidance (M1b)	a2	0.195	2.241 (0.500)	[1.302; 3.267]	4.480	< 0.001	0.045
Attachment Anxiety (M1a) → Hypomentalizing (M2)	d1	0.270	0.035 (0.006)	[0.022; 0.048]	5.342	< 0.001	0.158
Attachment Avoidance (M1b) → Hypomentalizing (M2)	d2	− 0.036	− 0.005 (0.006)	[− 0.016; 0.006]	− 0.876	0.381	
Hypomentalizing (M2) → Emotional eating (M3)	d5	0.181	0.351 (0.076)	[0.207; 0.503]	4.599	< 0.001	0.301
Emotional eating (M3) → Appetite drive (Y1)	b1	0.495	0.356 (0.029)	[0.302; 0.414]	12.443	< 0.001	0.535
Emotional eating (M3) → Low diet control (Y2)	b2	0.264	0.221 (0.035)	[0.152; 0.290]	6.333	< 0.001	0.206
Childhood trauma (X) → Appetite drive (Y1)	c1	0.239	0.543 (0.100)	[0.354; 0.745]	5.442	< 0.001	
Childhood trauma (X) → Low diet control (Y2)	c2	0.082	0.217 (0.115)	[− 0.011; 0.439]	1.895	0.058	
Attachment Anxiety (M1a) ↔ Attachment Avoidance (M1b)		0.104	1.021 (0.374)	[0.282; 1.758]	2.730	0.006	
Appetite drive (Y1) ↔ Low diet control (Y2)		0.327	0.090 (0.012)	[0.066; 0.114]	7.399	< 0.001	
Childhood trauma (X) → Hypomentalizing (M2)	a3	0.196	0.318 (0.075)	[0.174; 0.464]	4.259	< 0.001	
Childhood trauma (X) → Emotional eating (M3)	a4	0.183	0.578 (0.131)	[0.332; 0.847]	4.421	< 0.001	
Attachment Anxiety (M1a) → Emotional eating (M3)	d3	0.150	0.037 (0.011)	[0.017; 0.059]	3.475	0.001	
Attachment Anxiety (M1a) → Appetite drive (Y1)	b3	− 0.060	− 0.011 (0.007)	[− 0.024; 0.003]	− 1.564	0.118	
Attachment Anxiety (M1a) → Low diet control (Y2)	b4	0.111	0.023 (0.009)	[0.005; 0.041]	2.456	0.014	
Attachment Avoidance (M1b) → Emotional eating (M3)	d4	− 0.044	− 0.012 (0.010)	[− 0.031; 0.006]	− 1.266	0.205	
Attachment Avoidance (M1b) → Appetite drive (Y1)	b5	− 0.027	− 0.005 (0.006)	[− 0.018; 0.007]	− 0.830	0.407	
Attachment Avoidance (M1b) → Low diet control (Y2)	b6	− 0.042	− 0.010 (0.009)	[− 0.027; 0.008]	− 1.085	0.278	
Hypomentalizing (M2) → Appetite drive (Y1)	b7	0.140	0.196 (0.053)	[0.093; 0.302]	3.684	< 0.001	
Hypomentalizing (M2) → Low diet control (Y2)	b8	0.102	0.165 (0.072)	[0.027; 0.308]	2.310	0.021	
Sex → Attachment Anxiety (M1a)		0.037	0.373 (0.352)	[− 0.323; 1.057]	1.061	0.289	
Sex → Attachment Avoidance (M1b)		0.048	0.445 (0.333)	[− 0.225; 1.092]	1.338	0.181	
Sex → Hypomentalizing (M2)		0.015	0.019 (0.047)	[− 0.077; 0.111]	0.403	0.687	
Sex → Emotional eating (M3)		0.121	0.307 (0.069)	[0.170; 0.442]	4.434	< 0.001	
Sex → Appetite drive (Y1)		− 0.066	− 0.121 (0.041)	[− 0.200; − 0.039]	− 2.937	0.003	
Sex → Low diet control (Y2)		− 0.030	− 0.064 (0.064)	[− 0.192; 0.059]	− 1.007	0.314	
BMI → Attachment Anxiety (M1a)		− 0.009	− 0.006 (0.024)	[− 0.054; 0.040]	− 0.264	0.792	
BMI → Attachment Avoidance (M1b)		− 0.060	− 0.038 (0.023)	[− 0.083; 0.008]	− 1.615	0.106	
BMI → Hypomentalizing (M2)		0.072	0.006 (0.003)	[− 0.000; 0.013]	1.911	0.056	
BMI → Emotional eating (M3)		0.347	0.060 (0.005)	[0.050; 0.071]	11.365	< 0.001	
BMI → Appetite drive (Y1)		0.186	0.023 (0.004)	[0.015; 0.031]	5.721	< 0.001	
BMI → Low diet control (Y2)		0.121	0.018 (0.005)	[0.008; 0.027]	3.628	< 0.001	

β* standardized beta, β unstandardized beta, SE standard error, 95% CI 95% confidence intervals for the unstandardized beta, R2 explained variance

(SE = 0.601), [95% CI: 4.151; 6.523], z = 8.741, p < 0.001. Moreover, childhood trauma (X) was positively associated with attachment avoidance (M1b), *path a2*: β = 2.241 (SE = 0.500), [95% CI: 1.302; 3.267], z = 4.480, p < 0.001. Additionally, childhood trauma (X) was positively associated with hypomentalizing (M2), *path a3*: β = 0.318 (SE = 0.075), [95% CI: 0.174; 0.464], z = 4.259, p < 0.001.

Attachment anxiety (M1a) was positively associated with hypomentalizing (M2), *path d1*: β = 0.035 (SE = 0.006), [95% CI: 0.022; 0.048], z = 5.342, p < 0.001. In contrast, attachment avoidance (M1b) was not positively associated with hypomentalizing (M2), *path d2*: β = − 0.005 (SE = 0.006), [95% CI: − 0.016; 0.006], z = − 0.876, p = 0.381. Attachment anxiety (M1a) and attachment avoidance (M1b) were slightly positively associated with each other: β = 1.021 (SE = 0.374), [95% CI: 0.282; 1.758], z = 2.730, p = 0.006.

Moreover, hypomentalizing (M2) predicted emotional eating (M3), *path d5*: β = 0.351 (SE = 0.076), [95% CI: 0.207; 0.503], z = 4.599, p < 0.001.

Finally, emotional eating (M3) was positively associated with appetite drive (Y1), *path b1*: β = 0.356 (SE = 0.029), [95% CI: 0.302; 0.414], z = 12.443, p < 0.001. Moreover, emotional eating (M3) was positively associated with low-level diet control (Y2), *path b2*: β = 0.221 (SE = 0.035), [95% CI: 0.152; 0.290], z = 6.333, p < 0.001. The appetite drive (Y1) and low-diet control (Y2) were moderately positively associated with each other: β = 0.090 (SE = 0.012), [95% CI: 0.066; 0.114], z = 7.399, p < 0.001.

Furthermore, an examination of the four indirect paths was performed (the detailed results are reported in Table 5). The effects on appetite drive (Y1) were primarily examined. The *first* indirect effect (controlling for M1b and Y2: childhood trauma → attachment anxiety → hypomentalizing → emotional eating → appetite

Table 5 Summary of parameter estimates for the model's indirect and total effects (Fig. 2)

	Path	β*	β (SE)	95%CI [L - U]	z value	p value
Effect of X on Y1 via M1a	a1*b3	− 0.025	− 0.056 (0.037)	[− 0.130; 0.014]	− 1.533	0.125
Effect of X on Y1 via M1b	a2*b5	− 0.005	− 0.012 (0.015)	[− 0.042; 0.017]	− 0.799	0.424
Effect of X on Y1 via M2	a3*b7	0.027	0.062 (0.022)	[0.024; 0.109]	2.885	0.004
Effect of X on Y1 via M3	a4*b1	0.091	0.206 (0.048)	[0.117; 0.304]	4.282	< 0.001
Effect of X on Y1 via M1a and M2	a1*d1*b7	0.016	0.036 (0.013)	[0.014; 0.064]	2.788	0.005
Effect of X on Y1 via M1b and M2	a2*d2*b7	− 0.001	− 0.002 (0.003)	[− 0.009; 0.003]	− 0.757	0.449
Effect of X on Y1 via M1a and M3	a1*d3*b1	0.031	0.070 (0.023)	[0.030; 0.121]	2.996	0.003
Effect of X on Y1 via M1b and M3	a2*d4*b1	− 0.004	− 0.010 (0.008)	[− 0.028; 0.005]	− 1.160	0.246
Effect of X on Y1 via M2 and M3	a3*d5*b1	0.018	0.040 (0.013)	[0.017; 0.068]	3.059	0.002
Effect of X on Y1 via M1a, M2, and M3	a1*d1*d5*b1	0.010	0.023 (0.007)	[0.012; 0.038]	3.374	0.001
Effect of X on Y1 via M1b, M2, and M3	a2*d2*d5*b1	− 0.001	− 0.001 (0.002)	[− 0.005; 0.002]	− 0.810	0.418
Total indirect effect of X on Y1		0.156	0.355 (0.063)	[0.238; 0.486]	5.629	< 0.001
Total effect on Y1		0.396	0.897 (0.100)	[0.711; 1.100]	8.987	< 0.001
Effect of X on Y2 via M1a	a1*b4	0.046	0.121 (0.052)	[0.025; 0.230]	2.308	0.021
Effect of X on Y2 via M1b	a2*b6	− 0.008	− 0.022 (0.021)	[− 0.067; 0.017]	− 1.022	0.307
Effect of X on Y2 via M2	a3*b8	0.020	0.053 (0.026)	[0.008; 0.111]	1.992	0.046
Effect of X on Y2 via M3	a4*b2	0.048	0.128 (0.036)	[0.064; 0.207]	3.527	< 0.001
Effect of X on Y2 via M1a and M2	a1*d1*b8	0.011	0.030 (0.015)	[0.005; 0.062]	2.045	0.041
Effect of X on Y2 via M1b and M2	a2*d2*b8	− 0.001	− 0.002 (0.003)	[− 0.008; 0.002]	− 0.724	0.469
Effect of X on Y2 via M1a and M3	a1*d3*b2	0.016	0.043 (0.015)	[0.018; 0.077]	2.878	0.004
Effect of X on Y2 via M1b and M3	a2*d4*b2	− 0.002	− 0.006 (0.005)	[− 0.018; 0.003]	− 1.116	0.264
Effect of X on Y2 via M2 and M3	a3*d5*2	0.009	0.025 (0.009)	[0.010; 0.044]	2.823	0.005
Effect of X on Y2 via M1a, M2, and M3	a1*d1*d5*b2	0.005	0.014 (0.005)	[0.007; 0.025]	3.076	0.002
Effect of X on Y2 via M1b, M2, and M3	a2*d2*d5*b2	− 0.000	− 0.001 (0.001)	[− 0.003; 0.001]	− 0.800	0.424
Total indirect effect of X on Y2		0.145	0.383 (0.067)	[0.260; 0.525]	5.681	< 0.001
Total effect on Y2		0.228	0.601 (0.103)	[0.401; 0.804]	5.821	< 0.001

β standardized beta, *β* unstandardized beta, *SE* standard error, *95% CI* 95% confidence intervals for the unstandardized beta. X = childhood trauma; M1a = attachment anxiety; M1b = attachment avoidance; M2 = hypomentalizing; M3 = emotional eating; Y1 = appetite drive; Y2 = low diet control

drive) was statistically significant: β = 0.023 (SE = 0.007), [95% CI: 0.012; 0.038], z = 3.374, *p* = 0.001. In contrast, the *second* indirect effect (controlling for M1a and Y2: childhood trauma → attachment avoidance → hypomentalizing → emotional eating → appetite drive) was not statistically significant: β = − 0.001 (SE = 0.002), [95% CI: − 0.005; 0.002], z = − 0.810, *p* = 0.418. The total indirect effect (controlling for Y2: childhood trauma → attachment anxiety → hypomentalizing → emotional eating → appetite drive *plus* attachment avoidance) was statistically significant: β = 0.355 (SE = 0.063), [95% CI: 0.238; 0.486], z = 5.629, *p* < 0.001.

Second, the effects on the low dietary control (Y2) were examined. The first indirect effect (controlling for M1a and Y1: childhood trauma → attachment anxiety → hypomentalizing → emotional eating → low diet control) was statistically significant: β = 0.014 (SE = 0.005), [95% CI: 0.007; 0.025], z = 3.076, *p* = 0.002. In contrast, the second indirect effect (controlling for M1a and Y1: childhood trauma → attachment avoidance → hypomentalizing → emotional eating → low diet control) was not statistically significant: β = − 0.001 (SE = 0.001), [95% CI: − 0.003; 0.001], z = − 0.800, *p* = 0.424. The total indirect effect (controlling for Y1: childhood trauma →

attachment anxiety → hypomentalizing → emotional eating → appetite drive plus attachment avoidance) was statistically significant: β = 0.383 (SE = 0.067), [95% CI: 0.260; 0.525], z = 5.681, *p* < 0.001.

The degree of explained variance (R^2) was 53.5% ($R^2 = 0.535$) for appetite drive (Y1) and 20.6% ($R^2 = 0.206$) for low-diet control (Y2).

Discussion

The present study sought to address a critical gap in the literature by proposing and testing a theory-driven multiple mediation model that links CTEs to addiction-like eating behaviors (i.e., overeating/appetite drive and low diet control) through impairments in reflective functioning (i.e., hypomentalization) and the use of maladaptive self-regulation strategies, such as emotional eating.

Previous research has identified the dual transdiagnostic nature of food addiction and addiction-like eating behaviors [136–138]. Early psychological vulnerabilities such as CTEs, attachment insecurity, and impaired reflective functioning have also been recognized as risk factors for disordered eating [27, 29, 49].

However, no comprehensive model has integrated these variables to explain the developmental pathway

from early adversity to addiction-like eating behaviors [27, 91]. Understanding these mechanisms within a trauma-informed framework is essential for clarifying how early relational trauma and deficits in reflective functioning shape disordered eating patterns [49, 84].

The findings of the present study fully support the hypothesized multiple mediation model and are consistent with previous literature emphasizing the key role of early traumatic experiences in the development of disordered and addiction-like eating behaviors [27, 49] as well as reduced diet control. Empirical support was provided for the proposed developmental model [29, 30, 41], in which the effects of CTEs on addiction-like eating behaviors and low diet control were mediated by attachment anxiety, hypomentalization, and emotional eating. Statistically significant associations were observed in the expected directions among all study variables, suggesting that this pathway is shaped by deficits in both interpersonal functioning and self-regulation [27, 49, 81].

The main findings of this study revealed several key points that substantially advance the understanding of trauma-based pathways to addiction-like eating behaviors and low diet control. First, in line with previous studies [49], CTEs demonstrated significant positive associations with both attachment anxiety and hypomentalizing, which is consistent with attachment theory's predictions regarding the impact of early relational trauma on internal working models and mentalizing capacity [42, 66, 69]. Notably, while CTEs predict both dimensions of attachment insecurity, only attachment anxiety—not attachment avoidance—has emerged as a significant predictor of hypomentalizing [49]. This differential pattern aligns with recent theoretical developments suggesting that attachment anxiety, characterized by negative self-views and a heightened need for proximity, may be more directly linked to mentalization difficulties than attachment avoidance, which involves defensive deactivation of the attachment system [49, 70, 71, 73, 74].

The finding that attachment anxiety, but not attachment avoidance, mediates the relationship between CTEs and hypomentalizing has important theoretical implications and is in line with the findings of previous studies [49]. Indeed, individuals with high attachment avoidance may maintain some mentalizing capacity through emotional suppression and cognitive distancing, although this may come at the cost of emotional authenticity and interpersonal connection [74, 139, 140]. In contrast, individuals with high attachment anxiety tend to exhibit hypervigilance to attachment-related threats and intense emotional responses that can overwhelm their mentalizing capacity, particularly during stress [78–81]. This emotional flooding interferes with the individual's ability to accurately understand both their own and others' mental states, resulting in rigid and distorted interpretations of internal experiences [78–81, 88], which could lead to impulsive/instinctual and unregulated behaviors.

Moreover, the central role of hypomentalizing (namely, impaired reflective functioning) in the model represents perhaps one of the most significant theoretical contributions of this study. These results align with recent findings [27, 91] that impaired reflective functioning serves as a crucial bridge—via attachment anxiety—between early relational trauma and disordered eating behaviors [49, 59, 84, 86, 141] related to food addictive behaviors (e.g., emotional eating and appetite drive) and low dietary control [27]. When individuals cannot adequately mentalize—that is, when they cannot understand their own and others' behaviors in terms of underlying mental states—they become vulnerable to using external regulators such as food to manage overwhelming internal states [76, 81, 84]. This finding is consistent with mentalization-based theories of psychopathology, which propose that deficits in reflective functioning (i.e., hypomentalization) create vulnerability to various forms of behavioral dysregulation, including substance use and disordered eating [29, 49, 84].

The pathway from hypomentalizing to emotional eating represents a particularly important mechanism identified in this study, which aligns with recent literature [91]. Emotional eating—the tendency to consume food in response to negative emotional states rather than physiological hunger—emerged as a direct consequence of impaired reflective functioning [92, 93]. When individuals cannot adequately understand and regulate their emotional experiences through mentalizing, they may resort to food as an external regulator to achieve emotional homeostasis [49, 84, 86]. This process is consistent with contemporary literature on FA, which emphasizes the role of UPFs in providing rapid but temporary relief from negative affect, thereby reinforcing cyclical patterns of consumption [8, 13, 16, 17]. The mediating role of emotional eating identified in this study provides important insights into the overlap between addiction-like eating behaviors and other EDs [23, 137] via disordered eating behaviors [8, 27, 31, 32], reinforcing the transdiagnostic nature of emotional eating behaviors [23]. Emotional eating represents a common feature across multiple eating disorder diagnoses and has been identified as a key maintaining factor in both clinical and subclinical eating pathology [93, 94, 96]. The finding that emotional eating—predicted by hypomentalization—serves as the proximal predictor of addiction-like eating behaviors suggests that interventions targeting emotion regulation, mentalizing abilities, and the relationship between affect and eating may be particularly beneficial for individuals with a history of trauma [31, 58–60, 94, 95].

The final links in the proposed model—from emotional eating to both appetite drive and low dietary

control—demonstrate how trauma-related vulnerabilities could ultimately manifest as addiction-like eating behaviors related to increased appetite and overeating as well as deficits in dietary self-regulation [23, 24, 34]. In this context, the addiction-like eating behaviors measured in this study—encompassing both heightened overeating/appetite drive and impaired dietary control—reflect the complex profile characteristic of FA. This pattern aligns with the DSM-5 criteria for substance use disorders, which include both approach behaviors (craving, seeking) and control deficits (inability to limit use, continued use despite consequences) [25].

The findings of this study contribute significantly to ongoing debates regarding the conceptualization of FA and its eating-related manifestations [23, 31, 32, 142]. The results suggest that FA shares etiological pathways with both SRADs, EDs, and disordered eating behaviors [33, 35, 37], reinforcing its transdiagnostic nature [23, 25]. The identification of CTEs as a common risk factor, mediated through attachment and difficulties in reflective functioning (i.e., hypomentalization), provides support for trauma-informed approaches to understanding FA, EDs and disordered eating behaviors [41, 43, 61]. Furthermore, as expected, consistent with the correlation analyses, positive associations were observed between BMI and eating-related variables (emotional eating, appetite drive, and low dietary control)—supporting the bidirectional relationship between weight status and eating behavior dysregulation [111, 143]. These findings align with existing literature demonstrating that BMI (model covariate) is linked to difficulties in appetite regulation and dietary self-control [91].

Considering the model as a whole, it is important to emphasize that there is a difference in the predictive capacity of the two outcomes. The difference in explained variance between model outcomes (53.5% for appetite drive versus 20.6% for low dietary control) indicates that the hypothesized model accounts well for eating-related addiction-like behaviors, but other variables (e.g., impulsivity, cognitive restraint, habitual eating routines, sleep quality) may be needed to more comprehensively explain dietary control.

The stronger prediction of appetite drive than of low dietary control suggests that the pathway from trauma to attachment anxiety, hypomentalization and emotional eating may be particularly relevant for understanding the motivational and behavioral aspects of addiction-like eating behavior [41, 144]. Thus, individuals with a history of CTEs may be more likely to develop an attachment anxiety pattern characterized by heightened hypervigilance and preoccupation with external threats [78–81]. This, in turn, may result in the experience and expression of intense and overwhelming emotions—often negative—that impair the self-regulatory functioning of mentalization [30]. As a consequence, the inability to self-regulate in the presence of such emotional states may lead to reliance on an external regulator, such as food [27, 30, 49, 86]. Over time, this regulatory sequence may become rigid and automatized, progressively applied across various stress-inducing contexts regardless of their specific characteristics. As such, food intake may become the individual's dominant coping strategy in response to the inability to self-regulate [145]. Additionally, the repetitive and compulsive nature of this mechanism—while initially effective in producing short-term relief—may lead to the development of tolerance, requiring progressively larger quantities of food to achieve the same regulatory effect [1, 8, 27, 32]. This process may ultimately contribute to the emergence of addiction-like eating patterns, particularly those related to food intake, while having only a marginal impact on low dietary control, suggesting that additional, and potentially more salient, factors may underlie difficulties in maintaining dietary control.

Clinical implications

The results of this study have substantial implications for clinical assessment, case formulation, and treatment planning for individuals presenting with addiction-like eating behaviors [27, 49]. The identification of a sequential developmental pathway from CTEs through attachment insecurity and impaired mentalization to emotional eating and addiction-like behaviors suggests several important clinical considerations.

First, a comprehensive assessment of individuals with suspected FA and addiction-like behaviors should include careful evaluation of childhood trauma history, attachment patterns, and mentalizing capacity [49, 63, 86]. These findings suggest that individuals with significant CTEs and attachment anxiety may be at particular risk for developing hypomentalizing and subsequent addiction-like eating behaviors. Clinicians should be aware that problematic eating behaviors may represent attempts at emotional regulation in individuals with trauma histories and mentalization difficulties [27, 49, 56, 84].

Second, the mediation pathway identified in this study points toward specific therapeutic targets [89, 146]. Rather than focusing solely on eating behaviors, treatment approaches should address the underlying psychological vulnerabilities that contribute to addiction-like eating patterns [30, 146–149]. Mentalization-based treatment (MBT) has emerged as a particularly promising intervention, given its focus on enhancing reflective functioning and improving emotional regulation capacity [147]. MBT is a structured intervention that aims to enhance the individual's capacity to understand behavior in terms of underlying mental states It employs a curious,

exploratory therapeutic stance to help individuals recognize and label thoughts, feelings, and desires in themselves and others. Key components include monitoring and regulating emotional arousal to maintain optimal conditions for reflective functioning and foster more secure attachment dynamics [146, 147]. MBT has demonstrated effectiveness for various trauma-related conditions and could be adapted for individuals with FA by incorporating specific attention to eating-related triggers and behaviors [145, 150].

The role of attachment anxiety in the developmental pathway also suggests that therapeutic interventions should address interpersonal difficulties and relationship patterns that may maintain emotional dysregulation and reliance on food for self-soothing [66, 145, 150]. Approaches that combine mentalization-based techniques with attachment-focused interventions may be particularly beneficial for individuals with trauma histories and addiction-like eating behaviors [27].

Additionally, the central role of emotional eating in linking mentalization deficits to addiction-like behaviors suggests that interventions targeting the relationship between emotions and eating may be crucial [151–153]. Dialectical behavior therapy (DBT) skills training, mindfulness-based interventions, and emotion regulation strategies may complement mentalization-focused work by providing concrete tools for managing emotional distress without resorting to food consumption [1, 32, 153].

Strengths and limitations

However, several limitations should be acknowledged. First, the cross-sectional design precludes causal inferences, and while the theoretical model suggests temporal ordering of variables, longitudinal research is necessary to establish causality definitively. However, in this regard, certain variables, such as CTEs, attachment, and hypomentalization, are inherently unchangeable [56, 154], and as such, their role in statistical models is typically limited to that of predictors. Second, the reliance on self-report measures introduces potential biases, including social desirability response and retrospective recall bias, particularly for CTEs. Moreover, while the CTQ-SF provides a valid assessment of CTEs within the caregiving context, it does not capture the full range of adversities measured by broader ACE questionnaires (e.g., parental divorce, household substance abuse, witnessing domestic violence). Future research should employ more comprehensive assessments of childhood adversity to examine how different types of early life stress contribute to addiction-like eating behaviors. Third, the sample composition, while large, was predominantly female (86.4%)—a common pattern in online survey research using convenience sampling methods—which may limit its generalizability to male populations [155, 156]. Sex differences in trauma

responses, attachment patterns, and eating behaviors are well documented, and future research should examine whether the identified pathways operate similarly across sexes. However, the present study sought to address this limitation by including sex as a covariate in the model. Fourth, the convenience sample recruited through social media may not be representative of the broader population, potentially limiting generalizability; however, to mitigate this issue, important checks were carried out to ensure data quality [98]. Fifth, the study focused on a specific set of mediating variables (attachment dimensions, mentalization, emotional eating), whereas other potentially important factors (such as impulsivity, emotion dysregulation, social support, or genetic factors) were not included. Future research should examine how additional variables might modify or enhance the proposed pathway.

Despite these limitations, this study presents several notable strengths that enhance confidence in the findings and their clinical applicability. First, the use of SEM with latent variables and bootstrapped confidence intervals provides robust statistical testing of the hypothesized mediational pathways while accounting for measurement error [118]. Second, the use of item parceling to create latent variables helps address some of the limitations associated with self-report measures by reducing the impact of measurement error and improving model fit [125]. Third, the large sample size ($N = 1014$) provides adequate statistical power for detecting the hypothesized effects and testing complex mediational models [99]. Finally, this study is grounded in well-established theoretical frameworks from attachment theory, mentalization theory, and addiction research, providing strong conceptual foundations for the tested model.

Future research directions

The findings of this study suggest several important directions for future research [27, 49]. Longitudinal investigations following individuals from childhood through adulthood provide more definitive evidence regarding the temporal sequencing of the proposed pathway and could identify critical periods for intervention. Such studies could also examine how the pathway might differ across developmental stages and identify factors that promote resilience in individuals with trauma histories [49, 92, 147].

Research examining other potential mediators and/or moderators of the identified pathway would enhance the understanding of individual differences in vulnerability and resilience [30, 41]. Factors such as social support, therapeutic intervention, positive relationship experiences, or genetic variations in stress response systems might influence the strength of the associations between trauma, attachment, mentalization, and eating behaviors.

Despite controlling for sex as a covariate, the gender imbalance in the current sample (86.4% female) limits generalizability to males. In the present study, sex showed significant effects on emotional eating and appetite drive but did not significantly affect the mediational pathway from childhood trauma through attachment and mentalization to eating outcomes. Nevertheless, future research should specifically examine sex differences in the trauma-to-addiction pathway [49], as males and females may exhibit different patterns of attachment insecurity, mentalization difficulties, and eating-related coping strategies following childhood trauma [49, 73], with potential implications for targeted interventions.

Additionally, future research should examine the role of emotional eating in response to positive emotions. While the present study focused on emotional eating driven by negative affect—consistent with our trauma-based theoretical framework and the TFEQ-R-18 measurement approach—emotional eating can also occur in response to positive emotions (e.g., celebratory eating, reward-based consumption). Employing measures that distinguish between negative and positive affect-driven eating would allow researchers to examine whether these pathways operate similarly or through different mechanisms, providing a more comprehensive understanding of emotional eating processes in the context of addiction-like eating behaviors.

Clinical research examining the effectiveness of mentalization-based and attachment-focused interventions for individuals with FA would provide important evidence regarding the clinical utility of the theoretical model [27, 145, 146, 153]. Such research could examine whether improvements in mentalization capacity lead to reductions in addiction-like eating behaviors and whether attachment security can be enhanced through therapeutic intervention.

Conclusions

This study provides the first comprehensive empirical test of a trauma-based developmental model of addiction-like eating behaviors, offering important insights into the complex pathways linking childhood adversity to addiction-like eating behaviors [1, 27]. The findings suggest that the relationship between CTEs and addiction-like eating behaviors operates through a sophisticated network of variables, highlighting the importance of attachment security, mentalization capacity, and emotional eating in understanding eating-related psychopathology.

The identification of hypomentalizing as a central mediator in the pathway from trauma to addiction-like eating behaviors has significant theoretical and clinical implications [16, 76, 146, 157]. These findings support the growing recognition that food addiction and eating disorders share common etiological factors and suggest

that trauma-informed, mentalization-based interventions may be particularly beneficial for individuals with addiction-like eating patterns.

The results contribute to the evolving understanding of FA as a legitimate clinical phenomenon that bridges traditional boundaries between substance use disorders and eating disorders [1, 22, 33, 36]. By demonstrating shared developmental pathways with other trauma-related conditions, this research supports integrative approaches to conceptualizing and treating addiction-like eating behaviors [27, 30, 41].

Ultimately, this investigation advances both theoretical understanding and clinical practice by providing empirical support for a comprehensive developmental model of food addiction. The findings underscore the importance of addressing underlying trauma and attachment vulnerabilities in treating addiction-like eating behaviors and suggest that effective interventions should target mentalization capacity and emotional regulation skills rather than focusing solely on eating behaviors or weight management. As the field continues to evolve in its understanding of food addiction and its relationship with eating disorders, this research provides a foundation for more sophisticated, trauma-informed approaches to prevention, assessment, and treatment.

Declarations

Ethics approval and consent to participate
Ethics approval was provided by the Ethics Committee of the University of Padua (n° 547-a). Informed consent was obtained from all participants. All procedures adhered to the relevant guidelines and regulations. The study described has been carried out in accordance with the Declaration of Helsinki.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of Philosophy, Sociology, Education, and Applied Psychology, Section of Applied Psychology, University of Padova, Padova, Italy

[2]Center for Intervention and Research on Family Studies-CIRF-Department of Philosophy, Sociology, Education, and Applied Psychology, Section of Applied Psychology, University of Padova, Padova, Italy

References

1. Gearhardt AN, Brownell KD, Gold MS, Potenza MN. Food & addiction: a comprehensive handbook. New Edition, Second Edition, New Edition, Second Edition. Oxford, New York: Oxford University Press; 2024.
2. Heeren FAN, Darcey VL, Deemer SE, Menon S, Tobias D, Cardel MI. Breaking down silos: the multifaceted nature of obesity and the future of weight management. Philos Trans R Soc B Biol Sci Royal Soc. 2023;378:20220215. https://doi.org/10.1098/rstb.2022.0215.
3. Haslam DW, James WPT. Obesity. The lancet. Elsevier. 2005;366:1197–209. https://doi.org/10.1016/S0140-6736(05)67483-1.
4. Rossi AA, Mannarini S, Semonella M, Castelnuovo G, Pietrabissa G. The association between grazing and food addiction: the Italian version of the repetitive eating questionnaire (Rep(Eat)-Q) and its relationships with food addiction Criteria. Nutrients. Volume 16. Multidisciplinary Digital Publishing Institute; 2024. p. 949. https://doi.org/10.3390/nu16070949.
5. Conceição EM, Mitchell JE, Engel SG, Machado PPP, Lancaster K, Wonderlich SA. What is grazing? Reviewing its definition, frequency, clinical characteristics, and impact on bariatric surgery outcomes, and proposing a standardized definition. Surg Obes Relat Dis Elsevier. 2014;10:973–82. https://doi.org/10.1016/j.soard.2014.05.002.
6. Fairburn CG, Brownell KD. Eating disorders and obesity: a comprehensive handbook. Guilford Press; 2005.
7. Rossi AA, Pietrabissa G, Tagliagambe A, Scuderi A, Montecchiani L, Castelnuovo G, et al. Many facets of eating disorders: profiling key psychological features of anorexia nervosa and binge eating disorder. Behav Sci. 2023;13. https://doi.org/10.3390/bs13030276.
8. Rossi AA. Tying food addiction to uncontrolled eating: the roles of eating-Related thoughts and emotional eating. Nutrients Multidisciplinary Digit Publishing Inst. 2025;17:369. https://doi.org/10.3390/nu17030369.
9. Gearhardt AN, Corbin WR, Brownell KD. Food addiction: an examination of the diagnostic criteria for dependence. J Addict Med. 2009;3:1–7. https://doi.org/10.1097/ADM.0b013e318193c993.
10. Volkow ND, Wang G-J, Fowler JS, Telang F. Overlapping neuronal circuits in addiction and obesity: evidence of systems pathology. Philos Trans R Soc B Biol Sci Royal Soc. 2008;363:3191–200. https://doi.org/10.1098/rstb.2008.0107.
11. Wang G-J, Volkow ND, Thanos PK, Fowler JS. Similarity between obesity and drug addiction as assessed by neurofunctional imaging. J Addict Dis Taylor Francis. 2004;23:39–53. https://doi.org/10.1300/J069v23n03_04.
12. Davis C, Curtis C, Levitan RD, Carter JC, Kaplan AS, Kennedy JL. Evidence that 'food addiction' is a valid phenotype of obesity. Appetite. 2011;57:711–7. https://doi.org/10.1016/j.appet.2011.08.017.
13. Gearhardt AN, Schulte EM. Is food addictive? A review of the science. Annu Rev Nutr. 2021;41:387–410. https://doi.org/10.1146/annurev-nutr-110420-111710.
14. Schulte EM, Avena NM, Gearhardt AN. Which foods May be addictive? The roles of processing, fat content, and glycemic load. PLoS ONE. 2015;10. https://doi.org/10.1371/journal.pone.0117959.
15. Monteiro CA, Cannon G, Moubarac J-C, Levy RB, Louzada MLC, Jaime PC. The UN decade of Nutrition, the NOVA food classification and the trouble with ultra-processing. Public Health Nutr Camb Univ Press. 2018;21:5–17. https://doi.org/10.1017/S1368980017000234.
16. LaFata EM, Moran AJ, Volkow ND, Gearhardt AN. Now is the time to recognize and respond to addiction to ultra-processed foods. Nat Med. 2025;1–2. https://doi.org/10.1038/s41591-025-03858-6.
17. Neff KMH, Fay A, Saules KK. Foods and nutritional characteristics associated with Addictive-Like eating. Psychol Rep SAGE Publications Inc. 2022;125:1937–56. https://doi.org/10.1177/00332941211014156.
18. Gearhardt AN, DiFeliceantonio AG. Highly processed foods can be considered addictive substances based on established scientific criteria. Addiction. 2023;118:589–98. https://doi.org/10.1111/add.16065.
19. Volkow ND, Wise RA, Baler R. The dopamine motive system: implications for drug and food addiction. Nat Rev Neurosci Nat Publishing Group. 2017;18:741–52. https://doi.org/10.1038/nrn.2017.130.
20. Davis C, Loxton NJ, Levitan RD, Kaplan AS, Carter JC, Kennedy JL. Food addiction' and its association with a dopaminergic multilocus genetic profile. Physiol Behav. 2013;118:63–9. https://doi.org/10.1016/j.physbeh.2013.05.014.
21. Gearhardt AN, Yokum S, Orr PT, Stice E, Corbin WR, Brownell KD. Neural correlates of food addiction. Arch Gen Psychiatry. 2011;68:808–16. https://doi.org/10.1001/archgenpsychiatry.2011.32.
22. Leigh S-J, Morris MJ. The role of reward circuitry and food addiction in the obesity epidemic: an update. Biol Psychol. 2018;131:31–42. https://doi.org/10.1016/j.biopsycho.2016.12.013.
23. Meule A, Gearhardt AN. Ten years of the Yale food addiction scale: a review of version 2.0. Curr Addict Rep. 2019;6:218–28. https://doi.org/10.1007/s40429-019-00261-3.
24. Meule A, Gearhardt AN. Five years of the Yale food addiction scale: taking stock and moving forward. Curr Addict Rep. 2014;1:193–205. https://doi.org/10.1007/s40429-014-0021-z.
25. Meule A, Gearhardt AN. Food addiction in the light of DSM-5. Nutrients. Multidisciplinary Digit Publishing Inst. 2014;6:3653–71. https://doi.org/10.3390/nu6093653.
26. Parnarouskis L, Leventhal AM, Ferguson SG, Gearhardt AN, Withdrawal. A key consideration in evaluating whether highly processed foods are addictive. Obes Rev. 2022;23:e13507. https://doi.org/10.1111/obr.13507.
27. Rossi AA, Tagliagambe A, Scuderi A, Dalla Ragione L, Mannarini S. The path from childhood emotional maltreatment to disordered eating behaviors: the role of reflective functioning and food addiction. Nutrients. 2025. https://doi.org/10.3390/nu17111863.
28. Conceição EM, Utzinger LM, Pisetsky EM. Eating disorders and problematic eating behaviours before and after bariatric surgery: Characterization, assessment and association with treatment outcomes. Eur Eat Disord Rev. 2015;23:417–25. https://doi.org/10.1002/erv.2397.
29. Santoro G, Cannavò M, Schimmenti A, Barberis N. Childhood trauma and eating disorder risk among young adult females: the mediating role of mentalization. Dev Psychopathol. 2025;1–8. https://doi.org/10.1017/S0954579425100205.
30. Santoro G, Musetti A, Costanzo A, Schimmenti A. Self-discontinuity in behavioral addictions: a psychodynamic framework. Addict Behav Rep. 2025;100601. https://doi.org/10.1016/j.abrep.2025.100601.
31. Brewerton TD. Food addiction as a proxy for eating disorder and obesity severity, trauma history, PTSD symptoms, and comorbidity. Eat Weight Disord - Stud Anorex Bulim Obes. 2017;22:241–7. https://doi.org/10.1007/s40519-016-0355-8.
32. Krupa H, Gearhardt AN, Lewandowski A, Avena NM. Food addiction. Brain Sci Multidisciplinary Digit Publishing Inst. 2024;14:952. https://doi.org/10.3390/brainsci14100952.
33. Davis C. Compulsive overeating as an addictive behavior: overlap between food addiction and binge eating disorder. Curr Obes Rep. 2013;2:171–8. https://doi.org/10.1007/s13679-013-0049-8.
34. Schulte EM, Gearhardt AN. Attributes of the food addiction phenotype within overweight and obesity. Eat Weight Disord - Stud Anorex Bulim Obes. 2020; https://doi.org/10.1007/s40519-020-01055-7
35. Schulte EM, Joyner MA, Potenza MN, Grilo CM, Gearhardt AN. Current considerations regarding food addiction. Curr Psychiatry Rep [Internet]. 2015. https://doi.org/10.1007/s11920-015-0563-3. 17.
36. Schulte EM, Wadden TA, Allison KC. An evaluation of food addiction as a distinct psychiatric disorder. Int J Eat Disord. 2020;53:1610–22. https://doi.org/10.1002/eat.23350.
37. Stice E, South,Kelsey, Shaw H. Future directions in Etiologic, Prevention, and treatment research for eating disorders. J Clin Child Adolesc Psychol Routledge. 2012;41:845–55. https://doi.org/10.1080/15374416.2012.728156.
38. Hilbert A, Pike KM, Goldschmidt AB, Wilfley DE, Fairburn CG, Dohm F-A, et al. Risk factors across the eating disorders. Psychiatry Res. 2014;220:500–6. https://doi.org/10.1016/j.psychres.2014.05.054.
39. Fairburn CG, Cooper Z, Shafran R. Cognitive behaviour therapy for eating disorders: a transdiagnostic theory and treatment. Behav Res Ther. 2003;41:509–28. https://doi.org/10.1016/S0005-7967(02)00088-8.
40. Bernstein DP, Stein JA, Newcomb MD, Walker E, Pogge D, Ahluvalia T, et al. Development and validation of a brief screening version of the childhood trauma questionnaire. Child Abuse Negl. 2003;27:169–90. https://doi.org/10.1016/S0145-2134(02)00541-0.

41. Schimmenti A, Billieux J, Santoro G, Casale S, Starcevic V. A trauma model of substance use: elaboration and preliminary validation. Addict Behav. 2022;134:107431. https://doi.org/10.1016/j.addbeh.2022.107431.

42. Garon-Bissonnette J, Dubois-Comtois,Karine S-L. ,Diane, and Berthelot N. A deeper look at the association between childhood maltreatment and reflective functioning. Attach Hum Dev Routledge. 2023;25:368–89. https://doi.org/10.1080/14616734.2023.2207558.

43. Teicher MH, Gordon JB, Nemeroff CB. Recognizing the importance of childhood maltreatment as a critical factor in psychiatric diagnoses, treatment, research, prevention, and education. Mol Psychiatry Nat Publishing Group. 2022;27:1331–8. https://doi.org/10.1038/s41380-021-01367-9.

44. Midolo LR, Santoro G, Ferrante E, Pellegriti P, Russo S, Costanzo A, et al. Childhood trauma, attachment and psychopathology: A correlation network approach. Mediterr J Clin Psychol. 2020;8:1–25. https://doi.org/10.6092/2282-1619/mjcp-2418.

45. Somer E, Trauma. Dissociation, and opiate use disorder. Curr Addict Rep. 2019;6:15–20. https://doi.org/10.1007/s40429-019-0236-0.

46. Caslini M, Bartoli F, Crocamo C, Dakanalis A, Clerici M, Carrà G. Disentangling the association between child abuse and eating disorders: A systematic review and Meta-Analysis. Biopsychosoc Sci Med. 2016;78:79. https://doi.org/10.1097/PSY.0000000000000233.

47. Craparo G, Ardino V, Gori A, Caretti V. The relationships between early Trauma, Dissociation, and alexithymia in alcohol addiction. Psychiatry Investig Korean Neuropsychiatric Association. 2014;11:330–5. https://doi.org/10.4306/pi.2014.11.3.330.

48. Vanderlinden J, Palmisano GL. Trauma and eating disorders: the state of the art. Springer Publishing Company; 2024. https://connect.springerpub.com/content/book/978-0-8261-7265-5/part/part01/chapter/ch02. Accessed 27 Mar 2025.

49. Musetti A, Gagliardini G, Lenzo V, Cella S. From childhood emotional maltreatment to disordered eating: A path analysis. Psychoanal psychol. US: Educational Publishing Foundation; 2023. https://doi.org/10.1037/pap0000438. No Pagination Specified-No Pagination Specified.

50. Kimber M, McTavish JR, Couturier J, Boven A, Gill S, Dimitropoulos G, et al. Consequences of child emotional abuse, emotional neglect and exposure to intimate partner violence for eating disorders: a systematic critical review. BMC Psychol. 2017;5:33. https://doi.org/10.1186/s40359-017-0202-3.

51. Mills P, Newman EF, Cossar J, Murray G. Emotional maltreatment and disordered eating in adolescents: testing the mediating role of emotion regulation. Child Abuse Negl. 2015;39:156–66. https://doi.org/10.1016/j.chiabu.2014.05.011.

52. Pesavento F, Rossi AA. The association between different types of childhood trauma and orthorexia nervosa: an observational study. Front Psychol. Frontiers; 2025;16. https://doi.org/10.3389/fpsyg.2025.1665790.

53. Emery RL, Yoon C, Mason SM, Neumark-Sztainer D. Childhood maltreatment and disordered eating attitudes and behaviors in adult men and women:findings from project EAT. Appetite. 2021;163:105224. https://doi.org/10.1016/j.appet.2021.105224.

54. Bou Khalil R, Sleilaty G, Richa S, Seneque M, Iceta S, Rodgers R, et al. The impact of retrospective childhood maltreatment on eating disorders as mediated by food addiction: A Cross-Sectional Study. Nutrients. Multidisciplinary Digit Publishing Inst. 2020;12:2969. https://doi.org/10.3390/nu12102969.

55. Stojek MM, Maples-Keller JL, Dixon HD, Umpierrez GE, Gillespie CF, Michopoulos V. Associations of childhood trauma with food addiction and insulin resistance in African-American women with diabetes mellitus. Appetite. 2019;141:104317. https://doi.org/10.1016/j.appet.2019.104317.

56. Guney N, Snaychuk LA, Kim HS. An investigation into the relationship between adverse childhood experiences and food addiction: the role of urgency and emotion dysregulation. Psychiatry Res Commun. 2025;5:100206. https://doi.org/10.1016/j.psycom.2025.100206.

57. Holgerson AA, Clark MM, Ames GE, Collazo-Clavell ML, Kellogg TA, Graszer KM, et al. Association of adverse childhood experiences and food addiction to bariatric surgery completion and weight loss outcome. Obes Surg. 2018;28:3386–92. https://doi.org/10.1007/s11695-018-3370-1.

58. Hoover LV, Yu HP, Duval ER, Gearhardt AN. Childhood trauma and food addiction: the role of emotion regulation difficulties and gender differences. Appetite. 2022;177:106137. https://doi.org/10.1016/j.appet.2022.106137.

59. Imperatori C, Innamorati M, Lamis DA, Farina B, Pompili M, Contardi A, et al. Childhood trauma in obese and overweight women with food addiction and clinical-level of binge eating. Child Abuse Negl. 2016;58:180–90. https://doi.org/10.1016/j.chiabu.2016.06.023.

60. Lin X, Wang X, Wang C, Wu J, Yang W, Wang J, et al. Bidirectional mediation of psychological distress and food addiction influenced by childhood trauma among adolescents. J Affect Disord. 2025;375:448–55. https://doi.org/10.1016/j.jad.2025.01.123.

61. Brewerton TD. Eating Disorders, Trauma, and comorbidity: focus on PTSD. Eat Disord Routledge. 2007;15:285–304. https://doi.org/10.1080/10640260701454311.

62. Mason SM, Flint AJ, Field AE, Austin SB, Rich-Edwards JW. Abuse victimization in childhood or adolescence and risk of food addiction in adult women. Obesity. 2013;21:E775–81. https://doi.org/10.1002/oby.20500.

63. Tasca GA. Attachment and eating disorders: a research update. Attach Adulthood. 2019;25:59–64. https://doi.org/10.1016/j.copsyc.2018.03.003.

64. Tasca GA, Ritchie K, Zachariades F, Proulx G, Trinneer A, Balfour L, et al. Attachment insecurity mediates the relationship between childhood trauma and eating disorder psychopathology in a clinical sample: A structural equation model. Child Abuse Negl. 2013;37:926–33. https://doi.org/10.1016/j.chiabu.2013.03.004.

65. Schimmenti A. The trauma factor: examining the relationships among different types of trauma, dissociation, and psychopathology. J Trauma Dissociation Routledge. 2018;19:552–71. https://doi.org/10.1080/15299732.2017.1402400.

66. Cyr C, Euser EM, Bakermans-Kranenburg MJ, Van Ijzendoorn MH. Attachment security and disorganization in maltreating and high-risk families: a series of meta-analyses. Dev Psychopathol. 2010;22:87–108. https://doi.org/10.1017/S0954579409990289.

67. Topino E, Gori A. Childhood neglect and its effect on self-conception and attachment in adulthood: a structural equation model. Psychol Trauma Theory Res Pract Policy. US: Educational Publishing Foundation; 2025; https://doi.org/10.1037/tra0001921.

68. Musetti A, Pingani L, Zagaria A, Uberti D, Meli S, Lenzo V, et al. Insecure adult attachment and reflective functioning as mechanisms of the relationship between traumatic life events and suicidal ideation: a path analysis. Front Psychol. 2022;13. https://doi.org/10.3389/FPSYG.2022.985148.

69. Bowlby J. Attachment and loss. Separation. Basic Books; 1973.

70. Mikulincer M, Shaver PR, Pereg D. Attachment theory and affect regulation: the Dynamics, Development, and cognitive consequences of Attachment-Related strategies. Motiv Emot. 2003;27:77–102. https://doi.org/10.1023/A:1024515519160.

71. Mikulincer M, Shaver PR. The attachment behavioral system in adulthood: activation, psychodynamics, and interpersonal processes. In: Zanna M, editor. Advances in experimental social psychology, vol. 35. Academic; 2003. pp. 53–152.

72. Bartholomew K, Horowitz LM. Attachment styles among young adults: A test of a four-category model. J Pers Soc Psychol US: Am Psychol Association. 1991;61:226–44. https://doi.org/10.1037/0022-3514.61.2.226.

73. Jewell T, Collyer H, Gardner T, Tchanturia K, Simic M, Fonagy P, et al. Attachment and mentalization and their association with child and adolescent eating pathology: A systematic review. Int J Eat Disord. 2016;49:354–73. https://doi.org/10.1002/eat.22473.

74. Jewell T, Apostolidou E, Sadikovic K, Tahta-Wraith K, Liston S, Simic M, et al. Attachment in individuals with eating disorders compared to community controls: A systematic review and meta-analysis. Int J Eat Disord. 2023;56:888–908. https://doi.org/10.1002/eat.23922.

75. Fonagy P, Bateman AW. Adversity, attachment, and mentalizing. Compr Psychiatry. 2016;64:59–66. https://doi.org/10.1016/j.comppsych.2015.11.006.

76. Luyten P, Campbell C, Allison E, Fonagy P. The mentalizing approach to psychopathology: state of the Art and future directions. Annu Rev Clin Psychol Annual Reviews. 2020;16:297–325. https://doi.org/10.1146/annurev-clinpsy-071919-015355.

77. Penner F, Gambin M, Sharp C. Childhood maltreatment and identity diffusion among inpatient adolescents: the role of reflective function. J Adolesc. 2019;76:65–74. https://doi.org/10.1016/j.adolescence.2019.08.002.

78. Fonagy P, Target M. Playing with reality: I. Theory of Mind and the normal development of psychic reality. Int J Psychoanal. 1996;77:217–33.

79. Target M, Fonagy P. Playing with reality: II. The development of psychic reality from a theoretical perspective. Int J Psychoanal. 1996;77:459–79.

80. Fonagy P, Target M, Steele H, Steele M. Reflective-functioning manual version 5 for application to adult attachment interviews. UCL Discov. 1998. https://discovery.ucl.ac.uk/id/eprint/1461016/. Accessed 1 2025.

81. Fonagy P, Gergely G, Jurist E, Target M. Affect. Regulation, mentalization, and the development of the self. Other; 2002.

82. Musetti A, Brazzi F, Folli MC, Plazzi G, Franceschini C. Childhood Trauma, reflective Functioning, and problematic mobile phone use among male and female adolescents. Open Psychol J. 2020;13:242–52. https://doi.org/10.2174/1874350102013010242.

83. Handeland TB, Kristiansen VR, Lau B, Håkansson U, Øie MG. High degree of uncertain reflective functioning in mothers with substance use disorder. Addict Behav Rep. 2019;10:100193. https://doi.org/10.1016/j.abrep.2019.100193.

84. Gagliardini G, Gullo S, Tinozzi V, Baiano M, Balestrieri M, Todisco P et al. Mentalizing subtypes in eating disorders: a latent profile Analysis. Front psychol. 2020;11. https://doi.org/10.3389/fpsyg.2020.564291.

85. Musetti A, Starcevic V, Boursier V, Corsano P, Billieux J, Schimmenti A. Childhood emotional abuse and problematic social networking sites use in a sample of Italian adolescents: the mediating role of deficiencies in self-other differentiation and uncertain reflective functioning. J Clin Psychol. 2021;77:1666–84. https://doi.org/10.1002/jclp.23138.

86. Gagliardini G, Pandolfi G, Colli A, Attachment. Mentalization, emotion Dysregulation, and empathy in eating disorders: comparing anorexic and bulimic patients. J Nerv Ment Dis. 2024;212:370. https://doi.org/10.1097/NMD.0000000000001781.

87. Cicchetti D, Toth SL. Child Maltreatment. Annu rev clin psychol. Annual Reviews. 2005;1:409–38. https://doi.org/10.1146/annurev.clinpsy.1.102803.144029.

88. Lorenzini N, Campbell C, Fonagy P. Mentalization and its role in processing trauma. Approaches psych trauma theory pract. Lanham, MD, US: Rowman & Littlefield; 2019. pp. 403–22.

89. Cosenza M, Ciccarelli M, Nigro G. The steamy mirror of adolescent gamblers: Mentalization, impulsivity, and time horizon. Addict Behav. 2019;89:156–62. https://doi.org/10.1016/j.addbeh.2018.10.002.

90. Morosan L, Fonseca-Pedrero E, Debbané M. Network analysis of reflective functioning and conduct problems during adolescence. Psychol Violence US: Educational Publishing Foundation. 2020;10:300–11. https://doi.org/10.1037/vio0000258.

91. Rossi AA, Tagliagambe A, Scuderi A, Dalla Ragione L, Mannarini S. Uncontrolled eating through the lens of mentalization and emotional eating: the moderating role of food Addiction. Nutrients. Multidisciplinary Digit Publishing Inst. 2025;17:3233. https://doi.org/10.3390/nu17203233.

92. Fonagy P, Target M. Mentalization and the changing aims of child psychoanalysis. Psychoanal Dialogues Routledge. 1998;8:87–114. https://doi.org/10.1080/10481889809539235.

93. Macht M, Simons G. Emotional eating. In: Nyklíček I, Vingerhoets A, Zeelenberg M, editors. Emot regul Well-Being [Internet]. New York, NY: Springer; 2011. pp. 281–95. [cited 2025 May 3]. https://doi.org/10.1007/978-1-4419-6953-8_17.

94. Stark ML, Katariina. Emotional eating and eating disorder psychopathology. Eat Disord Routledge. 2001;9:251–9. https://doi.org/10.1080/10640260127552.

95. Nasirzadeh Y, Kantarovich K, Wnuk S, Okrainec A, Cassin SE, Hawa R, et al. Binge eating, loss of control over eating, emotional eating, and night eating after bariatric surgery: results from the Toronto Bari-PSYCH cohort study. Obes Surg. 2018;28:2032–9. https://doi.org/10.1007/s11695-018-3137-8.

96. Meule A, Richard A, Schnepper R, Reichenberger J, Georgii C, Naab S, et al. Emotion regulation and emotional eating in anorexia nervosa and bulimia nervosa. Eat Disord Routledge. 2021;29:175–91. https://doi.org/10.1080/10640266.2019.1642036.

97. Fricker RD. Sampling methods for web and E-mail surveys. SAGE Handb Online Res Methods. 2012;195–216. https://doi.org/10.4135/9780857020055.n11.

98. Pozzar R, Hammer MJ, Underhill-Blazey M, Wright AA, Tulsky JA, Hong F, et al. Threats of bots and other bad actors to data quality following research participant recruitment through social media: Cross-Sectional questionnaire. J Med Internet Res. 2020;22:e23021. https://doi.org/10.2196/23021.

99. Muthén LK, Muthén BO. How to use a Monte Carlo study to decide on sample size and determine power. Struct Equ Model Multidiscip J. 2002;9:599–620. https://doi.org/10.1207/S15328007SEM0904_8.

100. Wang AY, Rhemtulla M. Power analysis for parameter Estimation in structural equation modeling: A discussion and tutorial. Adv Methods Pract Psychol Sci. 2021;4:1–17. https://doi.org/10.1177/2515245920918253.

101. Rosseel Y. Lavaan: an R package for structural equation modeling. J Stat Softw. 2012;48:1–36. https://doi.org/10.18637/jss.v048.i02.

102. Jak S, Jorgensen TD, Verdam MGE, Oort FJ, Elffers L. Analytical power calculations for structural equation modeling: A tutorial and Shiny app. Behav Res Methods. 2021;53:1385–406. https://doi.org/10.3758/s13428-020-01479-0.

103. Wolf EJ, Harrington KM, Clark SL, Miller MW. Sample size requirements for structural equation models: an evaluation of power, bias, and solution propriety. Educ Psychol Meas. Volume 73. SAGE Publications Inc; 2013. pp. 913–34. https://doi.org/10.1177/0013164413495237.

104. Yuan K-H, Bentler PM. 5. Three likelihood-based methods for mean and covariance structure analysis with nonnormal missing data. Sociol Methodol. SAGE Publications Inc; 2000;30:165–200. https://doi.org/10.1111/0081-1750.00078.

105. Sacchi C, Vieno A, Simonelli A. Italian validation of the childhood trauma Questionnaire—Short form on a college group. Psychol trauma theory res pract policy. Volume 10. US: Educational Publishing Foundation; 2018. pp. 563–71. https://doi.org/10.1037/tra0000333.

106. Carli L. Attaccamento e Rapporto Di Coppia [Attachment and couple relationships]. Milano, IT: Raffaello Cortina; 1995.

107. Fonagy P, Luyten P, Moulton-Perkins A, Lee YW, Warren F, Howard S, et al. Development and validation of a self-report measure of mentalizing: the reflective functioning questionnaire. PLoS ONE. 2016;11:0158678. https://doi.org/10.1371/journal.pone.0158678.

108. Morandotti N, Brondino N, Merelli A, Boldrini A, Vidovich GZ, Ricciardo S, et al. The Italian version of the reflective functioning questionnaire: validity data for adults and its association with severity of borderline personality disorder. PLoS ONE. 2018;13:0206433. https://doi.org/10.1371/journal.pone.0206433.

109. Karlsson J, Persson L-O, Sjöström L, Sullivan M. Psychometric properties and factor structure of the Three-Factor eating questionnaire (TFEQ) in obese men and women. Results from the Swedish obese subjects (SOS) study. Int J Obes Nat Publishing Group. 2000;24:1715–25. https://doi.org/10.1038/sj.ijo.0801442.

110. Rossi AA, Pietrabissa G, Castelnuovo G, Mannarini S. Cognitive restraint, uncontrolled eating, and emotional eating. The Italian version of the three factor eating Questionnaire-Revised 18 (TFEQ-R-18): a three-step validation study. Eat Weight Disord - Stud Anorex Bulim Obes. 2024. https://doi.org/10.1007/s40519-024-01642-y.

111. Ruddock HK, Christiansen P, Halford JCG, Hardman CA. The development and validation of the Addiction-like eating behaviour scale. Int J Obes. 2017;41:1710–7. https://doi.org/10.1038/ijo.2017.158.

112. Rossi AA, Mannarini S, Castelnuovo G, Pietrabissa G. Disordered eating behaviors related to food addiction/Eating addiction in inpatients with obesity and the general population: the Italian version of the addiction-like eating behaviors scale (AEBS-IT). Nutrients. 2023;15:1–19. https://doi.org/10.3390/nu15010104.

113. McDonald RP, Ho M-HR. Principles and practice in reporting structural equation analyses. Psychol methods. Volume 7. US: American Psychological Association; 2002. pp. 64–82. https://doi.org/10.1037/1082-989X.7.1.64.

114. Rucker DD, Preacher KJ, Tormala ZL, Petty RE. Mediation analysis in social psychology: current practices and new recommendations. Soc Personal Psychol Compass. 2011;5:359–71. https://doi.org/10.1111/j.1751-9004.2011.00355.x.

115. Cohen J. Statistical power analysis for the behavioral sciences. Hillsdale, NJ: Lawrence Erlbaum Associates.; 1988.

116. Tabachnick BG, Fidell LS. Using multivariate statistics. 7th ed. Harlow: Pearson; 2019.

117. Brown TA. Confirmatory factor analysis for applied research, 2nd ed. New York, NY, US: The Guilford Press; 2015. p. xvii, 462.

118. Kline RB. Principles and practice of structural equation modeling. New York, NY, US: The Guilford Press; 2023.

119. Hu L, Bentler PM. Cutoff criteria for fit indexes in covariance structure analysis: conventional criteria versus new alternatives. Struct Equ Model Multidiscip J Routledge. 1999;6:1–55. https://doi.org/10.1080/10705519909540118.

120. Hoyle RH. Handbook of structural equation modeling: Second Edition. New York, NY, US: The Guilford Press; 2023. https://www.guilford.com/books/Handbook-of-Structural-Equation-Modeling/Rick-Hoyle/9781462544646. Accessed 8 Dec 2023.

121. Harman HH. Modern factor analysis. University of Chicago Press; 1976.

122. van de Schoot R, Lugtig P, Hox J. A checklist for testing measurement invariance. Eur J Dev Psychol Routledge. 2012;9:486–92. https://doi.org/10.1080/17405629.2012.686740.

123. Little TD, Rhemtulla M, Gibson K, Schoemann AM. Why the items versus parcels controversy needn't be one. Psychol Methods US: Am Psychol Association. 2013;18:285–300. https://doi.org/10.1037/a0033266.

124. Coffman DL, MacCallum RC. Using parcels to convert path analysis models into latent variable models. Multivar Behav Res Routledge. 2005;40:235–59. https://doi.org/10.1207/s15327906mbr4002_4.
125. Little TD, Cunningham WA, Shahar G, Widaman KF. To parcel or not to parcel: exploring the Question, weighing the merits. Struct Equ Model Multidiscip J Routledge. 2002;9:151–73. https://doi.org/10.1207/S15328007SEM0902_1.
126. Mackinnon DP. Introduction to statistical mediation analysis. Introd Stat Mediat Anal. 2012;1–477. https://doi.org/10.4324/9780203809556.
127. Bollen KA. Structural equations with latent variables. Wiley, Ltd; 1989. https://doi.org/10.1002/9781118619179.ch1. Accessed 30 Jun 2024.
128. Williams LJ, O'Boyle EH. Measurement models for linking latent variables and indicators: A review of human resource management research using parcels. Hum Resour Manag Rev. 2008;18:233–42. https://doi.org/10.1016/j.hrmr.2008.07.002.
129. Jorgensen SJ, and TD. 20 Factors with single indicators | A lavaan Compendium for Structural Equation Modeling in Educational Research. 2025. https://tdjorgensen.github.io/SEM-in-Ed-compendium/ch20.html. Accessed 3 Aug 2025.
130. Jöreskog KG, Sörbom D. LISREL 8: user's reference guide. Scientific Software International; 1996.
131. Nunnally JC. An overview of psychological measurement. In: Wolman BB, editor. Clin diagn ment Disord handb [Internet]. Boston, MA: Springer US; 1978. pp. 97–146. https://doi.org/10.1007/978-1-4684-2490-4_4.
132. Hayduk LA, Littvay L. Should researchers use single indicators, best indicators, or multiple indicators in structural equation models? BMC Med Res Methodol. 2012;12:159. https://doi.org/10.1186/1471-2288-12-159.
133. Iacobucci D. Everything you always wanted to know about SEM (structural equations modeling) but were afraid to ask. J Consum Psychol. 2009;19:673–80. https://doi.org/10.1016/j.jcps.2009.09.002.
134. MacKinnon DP, Kisbu-Sakarya Y, Gottschall AC. Developments in mediation analysis. Oxf Handb Quant Methods. 2013;2:338–60.
135. Bollen KA. Stine. Bootstrapping goodness of-fit measures in structural equation models. 1993.
136. Schulte EM, Gearhardt AN. Associations of food addiction in a sample recruited to be nationally representative of the united States. Eur Eat Disord Rev. 2018;26:112–9. https://doi.org/10.1002/erv.2575.
137. Aloi M, Liuzza MT, Rania M, Carbone EA, de Filippis R, Gearhardt AN, et al. Using latent class analysis to identify different clinical profiles according to food addiction symptoms in obesity with and without binge eating disorder. J Behav Addict Akadémiai Kiadó. 2024;13:262–75. https://doi.org/10.1556/2006.2023.00074.
138. Bonder R, Davis C, Kuk JL, Loxton NJ. Compulsive grazing and addictive tendencies towards food. Eur Eat Disord Rev. 2018;26:569–73. https://doi.org/10.1002/erv.2642.
139. Messina I, Calvo V, Grecucci A. Attachment orientations and emotion regulation: new insights from the study of interpersonal emotion regulation strategies. Res Psychother Psychopathol Process Outcome. 2023;26. https://doi.org/10.4081/ripppo.2023.703.
140. Schwarzer N-H, Dietrich L, Gingelmaier S, Nolte T, Bolz T, Fonagy P. Mentalizing partially mediates the association between attachment insecurity and global stress in preservice teachers. Front Psychol [Internet] Front. 2023. https://doi.org/10.3389/fpsyg.2023.1204666. [cited 2025 Aug 7];14.
141. Innamorati M, Imperatori C, Harnic D, Erbuto D, Patitucci D, Eleonora P, Janiri L, et al. Emotion regulation and mentalization in people at risk for food addiction. Behav Med. 2017;43:21–30. https://doi.org/10.1080/08964289.2015.1036831.
142. Panzeri A, Castelnuovo G, Spoto A. Assessing discriminant validity through structural equation modeling: the case of eating compulsivity. Nutrients Multidisciplinary Digit Publishing Inst. 2024;16:550. https://doi.org/10.3390/nu16040550.
143. Teodoro MC, Conceição EM, de Lourdes M, Alves JR, Neufeld CB. Grazing's frequency and associations with obesity, psychopathology, and loss of control eating in clinical and community contexts: A systematic review. Appetite. 2021;167:105620. https://doi.org/10.1016/j.appet.2021.105620.
144. Schimmenti A. Beyond addiction: rethinking problematic internet use from a motivational framework | clinical neuropsychiatry. Clin Neuropsychiatry. 2023;6. https://doi.org/10.36131/cnfioritieditore20230601.
145. Robinson P, Skårderud F, Sommerfeldt B, Hunger: mentalization-based treatments for eating disorders. Springer; 2018.
146. Allen JG, Fonagy P, Bateman AW. Mentalizing in clinical practice. Arlington, VA, US: American Psychiatric Publishing, Inc.; 2008. p. xx, 403.
147. Allen JG, Fonagy P, Sussex W. England: Wiley, Ltd; 2006. https://doi.org/10.1002/9780470712986.ch1
148. Bateman A, Fonagy P, Campbell C, Luyten P, Debbané M. Cambridge guide to mentalization-based treatment (MBT). Cambridge, UK: Cambridge University Press; 2023. https://www.cambridge.org/it/academic/subjects/medicine/mental-health-psychiatry-and-clinical-psychology/cambridge-guide-mentalization-based-treatment-mbt, https://www.cambridge.org/it/academic/subjects/medicine/mental-health-psychiatry-and-clinical-psychology. Accessed 2 Apr 2025.
149. Sekowski MM, Gambin M. Attachment anxiety, avoidance, and disorganization: latent profiles of attachment and their associations with hypomentalization, depressive symptoms, and suicidality. J Clin Psychol. n/a. https://doi.org/10.1002/jclp.70019.
150. Robinson P, Hellier J, Barrett B, Barzdaitiene D, Bateman A, Bogaardt A, et al. The NOURISHED randomised controlled trial comparing mentalisation-based treatment for eating disorders (MBT-ED) with specialist supportive clinical management (SSCM-ED) for patients with eating disorders and symptoms of borderline personality disorder. Trials. 2016;17:549. https://doi.org/10.1186/s13063-016-1606-8.
151. Zeeck A, Lau I, Endorf K, Schaefer L, Euler S, Lahmann C, et al. Mentalizing in psychotherapeutic processes of patients with eating disorders. Front Psychiatry [Internet] Front. 2024. https://doi.org/10.3389/fpsyt.2024.1367863. [cited 2025 Apr 2];15.
152. Zeeck A, Endorf K, Euler S, Schaefer L, Lau I, Flösser K, et al. Implementation of mentalization-based treatment in a day hospital program for eating disorders—A pilot study. Eur Eat Disord Rev. 2021;29:783–801. https://doi.org/10.1002/erv.2853.
153. O'Hea EL, Edwards-Hampton SA, Beall Brown DL, Sonneville KR, Ziedonis DM, Gearhardt AN. The food addiction clinical treatment (FACT) manual: A harm reduction treatment approach. Behav Sci Multidisciplinary Digit Publishing Inst. 2024;14:557. https://doi.org/10.3390/bs14070557.
154. Nolen-Hoeksema S, Watkins ER. A heuristic for developing transdiagnostic models of psychopathology: explaining multifinality and divergent trajectories. Perspect Psychol Sci SAGE Publications Inc. 2011;6:589–609. https://doi.org/10.1177/1745691611419672.
155. Sax LJ, Gilmartin SK, Bryant AN. Assessing response rates and nonresponse bias in web and paper surveys. Res High Educ. 2003;44:409–32. https://doi.org/10.1023/A:1024232915870.
156. Smith WG. Does gender influence online survey participation? A record-linkage analysis of university faculty online survey response behavior. Online Submiss. 2008. https://eric.ed.gov/?id=ED501717. Accessed 28 Oct 2025.
157. Salucci KG, Austin A. Mentalisation-based therapy for eating disorder treatment: protocol for a systematic review. BMJ Open Br Med J Publishing Group. 2025;15:e097639. https://doi.org/10.1136/bmjopen-2024-097639.

Publisher's note