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The role of autism symptoms on eating behavior in children diagnosed with attention deficit hyperactivity disorder: A preliminary study

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MAIN POINTS

- Subclinical autistic traits are significantly associated with disordered eating behaviors in children with ADHD.
- Food fussiness is significantly higher in children with ADHD who also exhibit autistic traits than in those without autistic traits.
- The findings underscore the importance of a dimensional approach to clinical assessment, moving beyond categorical diagnoses.

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ABSTRACT

Aim: The literature highlights the overlap between Attention-Deficit/Hyperactivity Disorder (ADHD) and Autism Spectrum Disorder (ASD), yet the impact of subclinical autistic traits on feeding behavior in ADHD populations remains underexplored. This study aims to investigate eating behaviors in children diagnosed with ADHD compared with those of typically developing peers, and to assess how elevated autistic traits influence eating behaviors within the ADHD group.

Materials and Methods: A cross-sectional comparative design was employed, involving 67 children with ADHD and 75 healthy controls. Data were collected using the Children's Eating Behavior Questionnaire (CEBQ) and the Social Responsiveness Scale (SRS). Within the ADHD group, participants were stratified by the presence of autistic traits. Group comparisons and correlational analyses were conducted to examine associations between autistic traits and feeding behaviors.

Results: Compared with controls, children with ADHD exhibited significantly higher Desire to Drink scores and lower Emotional Under-Eating scores. Within the ADHD group, those with autistic traits had significantly higher Food Fussiness scores than those without autistic traits. Correlation analyses revealed significant associations between total SRS scores and multiple CEBQ subscales, including Emotional Overeating, Desire to Drink, Satiety Responsiveness, and Food Fussiness. Subscales, such as Pathognomonic Autistic Behaviors and Reciprocal Social Behavior, were especially predictive of disordered eating patterns.

Conclusion: Findings suggest that autistic traits---even at subclinical levels---are significantly associated with maladaptive eating behaviors in children with ADHD. These results emphasize the importance of transdiagnostic approaches in clinical assessments, moving beyond categorical diagnoses. Understanding the dimensional interplay between ADHD symptoms and autistic traits may improve early nutritional interventions and individualized treatment strategies.

Keywords: Autism spectrum disorder, Attention-deficit/hyperactivity disorder, Children, Eating behavior, Subclinical traits, Social responsiveness

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INTRODUCTION

Attention Deficit/Hyperactivity Disorder (ADHD) is characterized by symptoms of inattention, hyperactivity, and impulsivity that are inconsistent with the individual's age and developmental level. In contrast, Autism Spectrum Disorder (ASD) is defined by impairments in social communication, restricted interests, and repetitive behavioral patterns [1]. Although ADHD and ASD have historically been conceptualized as distinct conditions, converging neurobiological and genetic findings highlight shared etiological mechanisms between ADHD and ASD. Both disorders involve disruptions in large-scale neural circuits responsible for executive functioning, reward processing, and sensory regulation, supporting a dimensional rather than strictly categorical conceptualization of neurodevelopmental symptoms [2,3].

Clinical observations and epidemiological data further support this overlap. This overlap is not limited to full syndromal comorbidity but also encompasses subclinical traits. Studies report that 59–83% of children with ASD exhibit clinically significant ADHD symptoms, while 30–60% of children with

ADHD display autistic-like traits [4,5]. Moreover, some individuals may exhibit pronounced autistic features without fully meeting the diagnostic criteria for ASD. These features, often referred to as "subclinical autistic traits," include milder yet clinically meaningful difficulties in social reciprocity, flexibility, sensory modulation, and communication [6]. Increasing evidence indicates that these traits exert measurable effects on children's emotional, behavioral, and regulatory functioning, even in the absence of a formal ASD diagnosis. A transdiagnostic, dimensional framework is therefore essential for understanding how these overlapping features shape clinical presentations [3]. These autistic traits may play a particularly significant role in complex domains such as feeding behavior, where sensory sensitivities, emotional regulation, and behavioral inflexibility intersect.

Feeding behavior represents a complex domain frequently impaired in both ADHD and ASD, attracting growing research interest regarding its underlying neurocognitive mechanisms [7,8]. In individuals with ADHD, impulsivity, heightened reward sensitivity, and inattention contribute to maladaptive eating patterns such as emotional overeating, a preference for rapidly consumed, high-calorie foods, irregular meal routines, and difficulty maintaining attention during meals [9–14]. Executive dysfunction may further impair the recognition of internal hunger and satiety cues, leading to behaviors such as prolonged mealtimes or forgetting to eat [9,15]. In ASD, feeding difficulties are typically driven by sensory hypersensitivity (e.g., aversions to specific textures, smells, or temperatures), cognitive rigidity, insistence on sameness, and strict mealtime routines, leading to food selectivity, limited dietary variety, and increased caregiver stress. Gastrointestinal symptoms, which are more prevalent in ASD, may further complicate feeding patterns [16–19].

Critically, when ADHD and ASD symptomatology co-occur, even at subclinical levels, the impact on eating behavior appears to be more severe than in either condition alone [20–22]. For example, sensory hyperreactivity linked to autistic traits can intensify food fussiness, while ADHD-related impulsivity and reward-driven eating may contribute to emotional overeating or an excessive preference for palatable foods. Cognitive rigidity associated with autistic traits may restrict dietary variety, whereas ADHD-related disinhibition may promote inconsistent mealtime behaviors. Despite strong theoretical justification for these interaction effects, existing research has largely excluded diagnostically complex cases or focused solely on categorical ASD–ADHD comorbidity, leaving the contribution of subclinical autistic traits insufficiently examined [23,24].

This study was designed to address the identified gap by pursuing two primary objectives: first, to compare the eating behaviors of drug-naïve children with ADHD with those of typically developing peers; second, to investigate whether varying levels of autistic traits (low vs. high) within the ADHD group are associated with distinct feeding patterns. By incorporating a dimensional assessment of autistic symptomatology, the study moves beyond categorical diagnostic boundaries and responds directly to the need for transdiagnostic approaches. This methodology provides a clear framework for elucidating how cross-diagnostic features shape eating behaviors in children with ADHD. Ultimately, the findings aim to enrich the limited literature on this topic and to offer a novel, clinically relevant perspective by focusing on symptom severity, which may, in turn, inform more individualized interventions and nutritional counseling strategies.

MATERIALS AND METHODS

Participants and procedure

This study was designed as a preliminary, cross-sectional, comparative investigation. This study was reviewed and approved by the Non-Interventional Clinical Research Ethics Committee of Recep Tayyip Erdoğan University on June 13, 2024 (Decision No: 2024/135). Written informed consent was obtained from the parents of all participating children. The study was conducted in accordance with the principles of the Declaration of Helsinki. Participants were recruited from children presenting to the Child and Adolescent Psychiatry Outpatient Clinic of Recep Tayyip Erdoğan University Training and Research Hospital between July 2024 and February 2025. The ADHD group consisted of 67 drug-naive children aged 6–12 years who met the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) diagnostic criteria for ADHD. Drug-naïve status was confirmed through multiple sources, including detailed parental interviews, review of electronic medical records, and verification that no stimulant or non-stimulant ADHD medications (e.g., methylphenidate, atomoxetine) had ever been prescribed. Diagnoses were established by a child and adolescent psychiatrist using clinical interviews, academic performance records, teacher reports, and standardized psychometric evaluations. A total of 85 children were initially screened. Following comprehensive evaluation, 18 children were excluded due to atypical autism (n = 2), intellectual disability (n = 2), or incomplete parent/teacher questionnaire forms (n = 14).The final ADHD sample comprised 67 eligible participants. The control group included 75 typically developing children aged 6–12 years with no history of neuropsychiatric disorders or chronic medical conditions, evaluated as psychiatrically healthy through structured clinical interviews. The ADHD and control groups were matched on age and major sociodemographic variables to ensure comparability.

All participating children provided assent and written informed consent was obtained from their parents. Each child was administered a Sociodemographic Data Form, the Children's Eating Behavior Questionnaire (CEBQ), and the Social Responsiveness Scale (SRS). All scales were completed by the children's parents.

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Assessment tools

Sociodemographic data form: This form includes basic information such as the participant's age, gender, grade level, and parental education levels.

All participants were evaluated for psychiatric comorbidity by a specialist in child and adolescent psychiatry using the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version, Turkish Adaptation (K-SADS-PL-DSM-5-T) [25]. The K-SADS-PL-DSM-5-T is a comprehensive interviewer-administered assessment tool that allows evaluation of 23 diagnostic categories.

The Children's Eating Behavior Questionnaire (CEBQ), developed by Wardle and colleagues, is a 35-item parent-reported instrument that uses a five-point Likert scale to assess children's appetite characteristics and various eating behaviors [26]. Higher scores indicate more frequent expression of the related behaviors (e.g., food responsiveness, emotional overeating, food fussiness). The original internal consistency coefficients of the scale range between 0.74 and [upper bound missing]. 0.91. In the Turkish adaptation, internal consistency coefficients were reported to range from 0.61 to 0.84 for the subscales and 0.69 for the total scale [27].

The Social Responsiveness Scale (SRS) is a 65-item parent-report measure that assesses their child's social interactions and responsiveness over the past six months. It consists of five subscales: social awareness, social cognition, social communication, social motivation, and autistic mannerisms. Higher scores reflect greater impairment in social functioning. The scale has been shown to be significantly associated with autism diagnoses and is widely used to assess autistic traits [28,29]. Although a Turkish validation study has not yet been published, a large-

A scale study conducted by Ünal and colleagues on school-aged children reported a Cronbach's alpha of 0.86 and a test-retest reliability coefficient, Pearson r = 0.53 (p = 0.001) [30]. The total SRS scores range from 0 to 195. Scores between 60 and 80 indicate mild-to-moderate impairment in social reciprocity, while scores above 80 reflect severe impairment. In this study, children with SRS scores of 80 or higher were categorized into the "ADHD with autistic traits" group, whereas those with lower scores were categorized into the "ADHD without autistic traits" group.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version 25.0. The Shapiro–Wilk test was used to assess the normality of continuous variables. For normally distributed variables, means and standard deviations were reported; for non-normally distributed variables, medians with minimum and maximum values were reported. In comparisons between the ADHD and control groups, independent t-tests were used for normally distributed continuous variables (e.g., height), Fisher's exact test for categorical variables (e.g., gender), and Mann–Whitney U tests for non-normally distributed variables. The ADHD group was further subdivided into two subgroups based on the presence or absence of autistic traits, and the same statistical methods were applied for between-subgroup comparisons. To assess the relationship between the CEBQ subscales and the SRS total and subscale scores in the ADHD group, Spearman's correlation analysis was conducted. Only CEBQ subscales showing statistically significant correlations with SRS scores were subsequently included in the generalized linear model (GLM) analyses. To determine whether autistic traits independently predicted eating behaviors after accounting for potential confounders, age and sex were included as covariates in all multivariable GLMs. Because the CEBQ subscale scores displayed a non-normal, positively skewed distribution, a Gamma A distribution with a log link function was used in these models. A p-value of less than 0.05 was considered statistically significant for all analyses.

RESULTS

A total of 67 children diagnosed with ADHD and 75 age-matched healthy controls were included in the analyses. The groups did not differ significantly in age, height, weight, or BMI (all p>0.05). However, gender distribution differed significantly between the groups, with a higher proportion of boys in the ADHD group (Table 1).

No significant differences between groups were observed on the eating-behavior subscale scores: Enjoyment of Food, Emotional Overeating, Satiety Responsiveness, Slowness in Eating, Food Fussiness, and Food Responsiveness (p>0.05). However, the Desire to Drink score was significantly higher in the ADHD group. Conversely, the Emotional Under-Eating subscale was significantly higher in the control group. Regarding social functioning, the ADHD group scored significantly higher on the SRS total score and its subscales Reciprocal Social Behavior, Social Use of Language, and Pathognomonic Autistic Behaviors, compared with the control group. These differences were statistically significant and are presented in Table 1.

Within the ADHD group, comparisons were made between those with autistic traits (n=12) and those without (n=55). No significant differences were found between these subgroups in terms of age, height, weight, or BMI. Although the gender distribution (6 males/6 females vs. 37 males/18 females) differed between groups, this difference was not statistically significant (p=0.211).

Regarding the subscales of eating behavior, no significant differences were observed between ADHD children with and without autistic traits in the domains of Enjoyment of Food, Emotional Overeating, Desire to Drink, Satiety Responsiveness, Slowness in Eating, Emotional Under-Eating, and Food Responsiveness (p>0.05). However, the Food Fussiness subscale scores were significantly higher in children with autistic traits. These results are presented in Table 2.

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Table 1. Comparison of demographic data and scale scores between the ADHD group and the control group.

Variable	ADHD (n = 67)	Control (n = 75)	p
Age	9 (7-14)	9 (7-13)	0.834*
Sex (Male/Female)	43 /24	33 / 42	**0.012****
Height (cm) (mean ± SD	132±10.8	133±11.2	0.337***
Weight (kg)	30 (20-70)	30 (18-60)	0.888*
BMI	17.7 (12.2-31.2)	17.2 (12.5-32.6)	0.378*
Enjoyment of Food	16 (6-25)	16 (6-25)	0.817*
Emotional Overeating	6 (4-19)	5 (4-14)	0.246*
Desire to Drink	10 (3-39)	6 (3-14)	<.001*
Satiety Responsiveness	20 (7-50)	19 (8-33)	0.874*
Slowness in Eating	8 (4-20)	8 (4-20)	0.329*
Emotional Under-Eating	9 (4-19)	11 (4-20)	**0.046***
Food Fussiness	7 (3-14)	8 (3-17)	0.122*
Food Responsiveness	10 (5-25)	8 (5-21)	0.150*
SRS-Reciprocal Social Behavior	40 (15-84)	35(21-60)	**0.009***
SRS-Social Use of Language	7 (0-17)	7 (2-12)	**0.046***
SRS-Pathognomonic Autistic Behaviors	16 (1-39)	9 (2-22)	**<.001***
SRS- Total Score	64 (16-127)	52 (27-80)	**<.001***

BMI: Body Mass Index, SD, standard deviation.SRS: Social Responsiveness Scale. Data are presented as median (minimum–maximum) or mean ± SD, as appropriate. Mann–Whitney U test unless otherwise stated; Fisher's exact test for sex; independent t test for height.

Table 2. Comparison of ADHD children with and without autistic traits

Variable	ADHD with Autistic Trait (n = 12)	ADHD without Autistic Trait (n = 55)	p
Age	10 (7-13)	9 (7-14)	0.278*
Sex (Male/Female)	6 M / 6 F	37 M / 18 F	0.211**
Height (cm) (mean ± SD	135.8±8.9	131.6±11.2	0.448***
Weight (kg)	32.5 (22-48)	30 (20-70)	0.572*
BMI	17.5 (14-21.6)	17.7 (12.2-31.2)	0.671*
Enjoyment of Food	15.5 (7-25)	16 (6-25)	0.600*
Emotional Overeating	8 (4-16)	6 (4-19)	0.274*
Desire to Drink	9 (3-39)	10 (5-15)	0.549*
Satiety Responsiveness	22.5 (15-32)	20 (7-50)	0.527*
Slowness in Eating	8.5 (4-15)	8 (4-20)	0.164*
Emotional Under-Eating	10.5 (7-15)	9 (4-19)	0.282*
Food Fussiness	9 (5-14)	6 (3-13)	**0.042***
Food Responsiveness	10 (5-25)	10 (5-25)	0.838*

BMI: Body Mass Index, SD, standard deviation.SRS: Social Responsiveness Scale. Data are presented as median (minimum–maximum) or mean ± SD, as appropriate. Mann–Whitney U test unless otherwise stated; Fisher's exact test for sex; independent t test for height.

Spearman correlation analyses examining the relationships between SRS subscales and eating behaviors revealed several significant associations. The Reciprocal Social Behavior subscale was positively and significantly correlated with Emotional Overeating (p=0.002), Desire to Drink (p=0.023), Emotional Undereating (p=0.004), and Food Fussiness (p=0.017). A positive correlation with Food Responsiveness (p=0.083) was also observed, though this did not reach statistical significance. The Social Use of Language subscale showed a significant positive correlation only with Emotional Under-Eating (p=0.011), whereas other correlations with eating behaviors did not reach statistical significance. The Pathognomonic Autistic Behaviors subscale was significantly and positively associated with Desire to Drink (p=0.016), Satiety Responsiveness (p=0.015), Emotional Under-Eating (p=0.015), and Food Fussiness (p=0.045). Although a positive correlation with Food Responsiveness was also noted, it did not reach statistical significance (p=0.385).

Finally, the total SRS score was positively and significantly correlated with Emotional Overeating (p=0.008), Desire to Drink (p=0.008), Satiety Responsiveness (p=0.039), Emotional Undereating (p<0.001), and Food Fussiness (p=0.018). The correlation with Food Responsiveness approached statistical significance (p=0.062). These findings are summarized in Table 3.

GLMs were conducted in the ADHD group (n=67) to examine the predictive power of subclinical autistic traits (SRS Total Score) on various CEBQ subscales, controlling for potential confounding variables, namely sex and age (gamma distribution with a log link

function). The results of the four separate GLM analyses revealed that the SRS Total Score significantly predicted three out of the four examined CEBQ subscales.

Satiety Responsiveness: The SRS total score was the strongest predictor ($\chi^2(1) = 7.069$, p=0.008). This finding indicates that an increase in the severity of autistic traits is associated

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Table 3. Correlation between CEBQ and SRS in the ADHD group.

Variable		Enjoyment of Food	Emotional Overeating	Desire to Drink	Satiety Responsiveness	Slowness in Eating	Emotional Under-Eating	Food Fussiness	Food Responsiveness	SRS-Reciprocal Social Behavior	SRS-Social Use of Language	SRS-Social Use of Language
Emotional Overeating	r	.294*										
	p	*(.016)*										
Desire to Drink	r	.290*	.338**									
	p	*(.017)*	*(.005)*									
Satiety Responsiveness	r	-.393**	.016	.254*								
	p	*(.001)*	(.898)	(.038)								
Slowness in Eating	r	-.029	.022	-.020	.114							
	p	(.815)	(.861)	(.873)	(.357)							
Emotional Under-Eating	r	.141	.377**	.283*	.261*	.236						
	p	(.255)	*(.002)*	*(.020)*	*(.033)*	(.055)						
Food Fussiness	r	.208	.132	-.005	-.163	-.158	-.104					
	p	(.092)	(.288)	(.968)	(.187)	(.201)	(.402)					
Food Responsiveness	r	.643***	.646***	581***	-.229	.021	.298*	.194				
	p	*(<.001)*	*(<.001)*	*(<.001)*	(.062)	(.868)	*(.014)*	(.116)				
Reciprocal Social Behavior	r	-.001	.375**	.278*	.206	.090	.350**	.290*	213*			
	p	(.991)	*(.002)*	*(.023)*	(.094)	(.469)	*(.004)*	*(.017)*	(.083)			
SRS-Social Use of Language	r	.061	.153	.204	.108	.067	310*	.012	214	.426***		
	p	(.621)	(.215)	(.097)	(.386)	(.590)	*(.011)*	(.923)	(.082)	*(<.001)*		
SRS-Pathognomonic Autistic Behaviors	r	.048	.150	294*	.295*	.004	295*	245*	.108	.634***	515***	
	p	(.700)	(.225)	*(.016)*	(.015)	(.976)	*(.015)*	*(.045)*	(.385)	*(<.001)*	*(<.001)*	
SRS- Total	r	.038	322**	321**	252*	.105	.395 ***	289*	.229	911***	624***	.860***
	p	(.762)	*(.008)*	*(.008)*	*(.039)*	(.396)	*(<.001)*	*(.018)*	(.062)	*(<.001)*	*(<.001)*	*(<.001)*

SRS: Social Responsiveness Scale, Correlation coefficients (r) and p-values (in parentheses) are shown *: p < .05, **: p < .01, ***: p < .001.

Table 4. Predictive power of subclinical autistic traits on CEBQ subscales: generalized linear model results in the ADHD Group.

Dependent Variable (CEBQ Subscale)	Omnibus Test χ^2(df)	Model p	Predictors	Wald χ2 p
Satiety Responsiveness	$\chi^2(3) = 7.802$	**0.05**	SRS Total Score	**0.008**
			Sex	0.372
			Age	0.291
Food Fussiness	$\chi^2(3) = 8.992$	**0.029**	SRS Total Score	**0.017**
			Sex	0.418
			Age	0.104
Desire to Drink	$\chi^2(3) = 10.766$	**0.013**	SRS Total Score	**0.016**
			Sex	**0.016**
			Age	0.324
Emotional Overeating	$\chi^2(3) = 5.535$	0.137	SRS Total Score	**0.057**
			Sex	0.217
			Age	0.418

CEBQ: Children's Eating Behavior.

with a significantly reduced response to satiety signals (i.e., lower Satiety Responsiveness scores). The overall model fit was at the boundary of marginal significance ($\chi^2(3) = 7.802$, p=0.050).

Food Fussiness: The SRS Total Score also significantly predicted Food Fussiness ($\chi^2(1) = 5.719$, p=0.017). This evidence established that increasing severity of autistic traits resulted in a significant increase in Food Fussiness behavior,

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which is related to sensory sensitivity and rigidity (Omnibus Test: $\chi^2(3) = 8.992$, $p = 0.029$).

The SRS Total Score was also found to have a significant independent effect on Desire to Drink ($\chi^2(1) = 5.792$, $p=0.016$). This behavior is associated with oral sensory seeking or excessive thirst, demonstrating that autistic traits influence behaviors in this domain as well.

Conversely, Emotional Overeating behavior was marginally predicted by the SRS Total Score ($\chi^2(1) = 3.608$, $p=0.057$), which falls just below the conventional threshold for statistical significance, suggesting a trend-level association between Emotional Overeating and autistic traits (Omnibus Test: $\chi^2(3) = 5.535$, $p=0.137$). Among the control variables included in the model, the effects of Age and Sex were generally non-significant. Age was not identified as a significant predictor of any of the four eating behaviors examined ($p>0.291$). Sex had a significant effect only on Desire to Drink ($\chi^2(1) = 5.753$, $p=0.016$), but not on the other three eating behaviors (Table 4).

DISCUSSION

This preliminary study aimed to compare the eating behaviors of children diagnosed with ADHD with those of healthy controls and to examine the impact of subclinical autistic traits on eating behaviors within the ADHD group. The findings indicate that ADHD is associated with specific alterations in eating behavior and that autistic traits—even below the diagnostic threshold—contribute meaningfully to feeding difficulties.

In the present study, the Desire to Drink score was significantly higher in the ADHD group, whereas Emotional Under-Eating was more prevalent in the control group. Previous literature has consistently reported a higher prevalence of abnormal eating behaviors in children with ADHD than in their typically developing peers [9,11]. Studies in preschool and school-aged samples show positive associations between ADHD symptoms and emotional overeating, desire to drink, food responsiveness, and irregular eating patterns [12,13].

Our finding of an increased Desire to Drink may reflect heightened reward sensitivity and impulsive reward-seeking, well-documented features of ADHD that often lead children to prefer rapidly consumed sweetened beverages [13,31]. Conversely, higher Emotional Under-Eating in the control group may suggest that children with ADHD are less responsive to internal emotional states during meals, potentially due to impaired interoceptive awareness and impulse control [11,32].

In the ADHD group, the SRS subscale scores were significantly higher, indicating that these individuals experience considerable difficulties in social functioning[28]. When individuals with ADHD were further stratified based on the presence of autistic traits, a significant difference emerged only in the *Food Fussiness* subscale. Food selectivity was more common among children exhibiting autistic traits [7,21]. This finding aligns with previous research suggesting that sensory sensitivities and a tendency toward routine—features commonly associated with ASD—may contribute to increased food selectivity [16,17,33].

The number of studies examining eating behaviors in ADHD–ASD comorbidity remains limited. Prior research has shown that children with both ADHD and ASD differ from typically developing peers in domains such as food responsiveness, enjoyment of food, satiety responsiveness, slowness in eating, and picky eating [21,22]. Our study differs in that it highlights similar disruptions even when autistic traits are subclinical, suggesting that the influence of autistic features on feeding behavior is dimensional and not restricted to formal ASD diagnoses.

Correlation analyses revealed that higher levels of autistic traits were associated with more pronounced eating problems, including emotional overeating, desire to drink, satiety responsiveness, emotional undereating, and food fussiness. These associations support the hypothesis that autistic traits contribute to dysregulated eating through mechanisms such as sensory processing atypicalities, social communication deficits affecting emotion regulation, and behavioral rigidity influencing dietary variety [17,34].

Previous research [7,35,36] has highlighted the role of sensory sensitivity — a diagnostic feature of ASD that is also frequently observed in children with ADHD — in contributing to food selectivity. The significant association between Emotional Under-Eating and both the total SRS score and all subscale scores mirrors findings from samples with ASD-only or ASD–ADHD comorbidity [12,21,22,34]; this study demonstrates that the same association holds in children with ADHD who exhibit subclinical autistic traits. Difficulties with emotion regulation, a commonly reported factor in both ADHD and ASD, are thought to underlie emotional eating behaviors [23,24].

The observed positive correlations between the Pathognomonic Autistic Behaviors subscale and the Satiety Responsiveness and Desire to Drink subscales may indicate a prominent role for hedonic hunger mechanisms in this population. This is a critical finding in terms of both increased obesity risk and reduced nutritional quality [9,37].

To directly test our primary hypothesis and control for confounding variables, GLM analyses controlling for the effects of sex and age were performed and provided evidence that subclinical autistic traits (SRS Total Score) are independent and significant predictors of several problematic eating behaviors in children diagnosed with ADHD (see Table 4). This finding supports the presence of a transdiagnostic mechanism, given the high comorbidity and dimensional overlap between ADHD and ASD [38]. Our analyses showed that the SRS Total Score significantly predicted three of the four primary eating behaviors: Satiety Responsiveness, Food Fussi-

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ness, and Desire to Drink.

The influence of autistic traits on eating behavior appears to be concentrated in two key areas. First, a significant negative effect on satiety responsiveness suggests that children with high autistic traits have an impaired ability to perceive and respond to satiety signals. The literature suggests that interoceptive awareness deficits, commonly reported in ASD [39], hinder appropriate regulation of eating in response to physiological cues in these children. Second, the positive effects on picky eating and drink cravings are consistent with sensory-seeking and sensory-avoidant behaviors frequently associated with ASD [36]. These children may be selective eaters because of hypersensitivity to the texture, temperature, or taste of foods, or they may engage in oral-gratification-seeking behavior (Drink to Desire). These findings indicate that autistic traits trigger eating problems in the ADHD population that are particularly related to sensory and physiological signal processing, whereas their contribution to emotion-regulation-related eating problems (emotional overeating) is marginal. Furthermore, the limited independent effects of the control variables (age and gender) on overall eating behavior — except for a significant effect of gender on 'Drink to Desire' — confirm that the observed effect is specific to the severity of autistic traits.

This study makes a valuable contribution to the limited literature exploring the relationship between ADHD and autistic traits in the context of eating behavior. It is among the few studies that specifically examine the influence of subclinical autistic traits on the feeding behaviors in children diagnosed with ADHD. While there is substantial literature on feeding behaviors in children with either ADHD or ASD, research addressing their comorbidity and overlapping symptomatology remains scarce. By evaluating autistic traits through a transdiagnostic lens and examining their behavioral implications, this study adopts an approach that more accurately reflects clinical reality and highlights the importance of features beyond categorical diagnoses. Furthermore, the inclusion of a healthy control group matched on age and sociodemographic characteristics enhances the comparative strength of the analysis and allows for contextual interpretation of feeding behavior deviations within the framework of neurodevelopmental disorders. Stratifying the ADHD group by SRS scores and conducting secondary analyses according to the presence of autistic traits add dimensional depth to the study. This enabled not only diagnosis-based evaluation but also interpretation based on symptom severity, clarifying the specific associations between autistic features and distinct eating behaviors.

This study, which presents a preliminary evaluation of the impact of eating behaviors and autistic traits in children diagnosed with ADHD, is subject to several methodological and sampling limitations. Notably, the number of participants in the ADHD group who exhibited autistic traits (n = 12) was relatively small compared to the rest of the sample. This may have limited the statistical power of subgroup comparisons and hindered adequate representation of variance. Given that this study was conceived as a preliminary investigation to explore initial patterns and guide future hypothesis-driven research, no a priori power analysis was conducted. As a result, the study may be underpowered to detect small-to-moderate effects, particularly in subgroup analyses. Furthermore, the sample was drawn from a single clinical center, which restricts the generalizability of the findings to the broader ADHD population. Longitudinal studies are necessary to assess how these variables influence one another over time. All assessment tools used in the study were parent-report questionnaires and therefore may be subject to systematic biases, including social desirability bias, recall bias and perceptual distortion. The absence of direct access to the child's internal experiences—particularly for insight-dependent behaviors like emotional eating—represents an important limitation. Additionally, autistic traits were classified based on SRS scores, and cases meeting the diagnostic criteria for ASD were excluded. Although participants were screened for ASD using diagnostic criteria, the SRS, while highly sensitive and valid, measures the severity of autistic traits rather than providing a diagnosis. Therefore, the clinical significance of intergroup differences must be interpreted with caution. Eating behaviors were assessed exclusively through questionnaire data. Apart from BMI, no other objective anthropometric measures, biochemical indicators (e.g., leptin and ghrelin levels), or dietary logs were collected, limiting the ability to relate findings to physiological outcomes. Lastly, there was a significant difference in gender distribution between groups.

CONCLUSION

In conclusion, this study suggests that autistic traits may significantly affect eating behaviors in individuals with ADHD. In recent years, there has been a growing body of research examining the relationship between ADHD, obesity, and disordered eating, with particular emphasis on the roles of neurobiological factors and emotional symptoms. On the other hand, the association between ASD symptoms and feeding behavior is well established. Given our findings, it becomes evident that clinical evaluations should not rely solely on categorical DSM diagnoses but should also account for spectrum-based traits, underscoring the importance of a transdiagnostic approach. Considering the high prevalence of co-occurring ADHD and ASD, understanding how these conditions interact in the context of feeding behavior is essential for designing more effective and developmentally timely intervention programs. Future longitudinal studies with larger and more diverse samples will be instrumental in establishing causal relationships and deepening our understanding of these complex interactions.

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