

Association Between Binge Eating and Attention-Deficit/Hyperactivity Disorder in Two Pediatric Community Mental Health Clinics

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ABSTRACT

Objective: Attention-Deficit/Hyperactivity Disorder (ADHD) has been linked with obesity; however its relationship with binge eating (BE) is less clear. We aimed to explore the associations among ADHD, weight, and BE in pediatric mental health clinics.

Method: We retrospectively reviewed consecutive intakes in two pediatric mental health clinics ($N = 252$). BE was assessed using the C-BEDS scale. Associations between ADHD, BE, and BMI-z score were assessed via regression.

Results: Mean age was 10.8 (3.7 SD) years. Twelve percent ($n = 31$) had BE. The association between ADHD and BE was statistically significant (OR 16.1, $p < .001$), and persisted after adjusting for comorbid diagnoses, medications, demographic variables, and clinic. There was a statistically significant association between ADHD and BMI z-scores ($\beta = 0.54$, $p < .001$). After adjusting for BE, the relationship between ADHD and BMI z-scores was attenuated ($\beta = 0.35$, $p = .025$), and the coefficient for BE was decreased ($\beta = 0.75$, $p = .001$). Although stimulant use was associated with a

three-fold increase in odds of BE (OR 3.16, $p = .006$), stimulants were not associated with greater BMI-z scores ($\beta = 0.18$, $p = .32$).

Discussion: There was a significant association between ADHD and BE in two pediatric mental health clinics. Although these data are cross-sectional, and cannot be used to make causal inferences, these findings are compatible with the hypothesis that BE partially mediates the association between ADHD and BMI z-scores. In mental health clinics, children with ADHD may present as overweight or obese. Further, children with ADHD may exhibit BE. Future prospective studies should elucidate the complex relationships among ADHD, weight, stimulants, and BE. © 2014 Wiley Periodicals, Inc.

Keywords: binge eating; loss of control eating; children; adolescents; ADHD; overweight; obesity; disinhibited eating; Attention Deficit Hyperactivity Disorder; stimulant

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Introduction

Obesity is a particularly important and common public health issue among individuals with psychi-

atric disorders.¹ Attention-Deficit/Hyperactivity Disorder (ADHD) is also a prevalent disorder, with estimated prevalence rates of 5% in children and adolescents according to the Diagnostic and Statistical Manual-V (DSM-V) and other studies.^{2,3} ADHD has been associated with obesity, in both children and adults,^{4,5} although this may appear to be counterintuitive when hyperactivity is present, given the increased energy requirements of motoric hyperactivity.^{6,7} In addition to obesity, binge eating disorder (BE) has also been associated with ADHD in adults.⁸ In pediatric community mental health settings, there is a paucity of data regarding ADHD and binge eating (BE), although these disorders are frequently encountered in such settings.⁹ The term children will be used to describe children and adolescents.

Epidemiological studies have shown that children with ADHD typically tend to present a higher prevalence of obesity than expected; conversely,

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among obese children, rates of ADHD tend to be increased.^{4,10} Cross-sectional surveys have similarly reported that overweight and obese children were twice as likely to present with ADHD; similarly, children with ADHD had an odds ratio of 1.9 for obesity and overweight status.¹⁰ Despite epidemiological data supporting an association between ADHD and obesity, much less is known about the relationship between ADHD, obesity, and BE found in pediatric community mental health settings where many of these children are seen.

In addition to ADHD, other comorbid psychiatric disorders presenting to community mental health clinics also have increased rates of obesity. Psychiatrically-ill children often gain weight when they are administered psychotropic medications such as mood stabilizers and antipsychotics.¹¹ In one community psychiatric partial hospital program, the prevalence of obesity was double the national and county norms in adolescents with severe mental illness.¹² In that study, stimulant use was associated with a decreased risk of being overweight or obese; conversely, antidepressants increased the odds of being overweight or obese, as did antipsychotic use. However, BE was not examined.

In addition to data linking obesity and BED,^{11,12} and obesity with ADHD,⁴⁻⁶ emerging data in adults suggest a possible relationship between BED and ADHD.¹³ A large epidemiological study in adults suggested that the relationship between adult ADHD and obesity was partially mediated by the presence of BED.⁵ Similarly, a community-based adult study¹⁴ also reported that the relationship between obesity and ADHD was partially explained by BE. The association between BE and ADHD has also been described in adults with eating disorders, such as bulimia.⁸ Impulsivity may represent one hypothesized mechanism by which BE could affect ADHD and obesity. BE has been hypothesized to influence the association between ADHD and obesity in adults¹⁵; however, less is known regarding the relationship of BE and ADHD in pediatric community mental health clinics. Since pediatric eating disorders and ADHD often present to community mental health clinics, we aimed to explore the associations between BE and ADHD in this community setting.

Given the associations of both ADHD and BE with increased BMI as well as data suggesting an association between ADHD and BE in adults, perhaps the relationship between ADHD and increased BMI in children might be partially mediated by BE. This study aimed to explore the complex relationships among ADHD, Body Mass Index *z*-score (BMI *z*-score) and BE in a clinical sample of

children receiving outpatient community mental health treatment.

Method

Participants

Data were obtained from a retrospective chart review of 252 consecutively referred patients from two community child and adolescent psychiatry clinics. Chart review was performed on consecutive first clinic intake evaluations performed between January 2008 and December 2009. A total of 115 charts (46%) were reviewed from the Community Psychiatry Child and Adolescent Clinic at the Johns Hopkins Bayview Medical Center (Bayview) in Baltimore, Maryland; 137 charts (54%) were collected from the Children's Mental Health Center (CMHC) in Baltimore, Maryland. Both community child psychiatry clinics are affiliated with the Division of Child and Adolescent Psychiatry at the Johns Hopkins University.

Clinic Characteristics

Both the Bayview and CMHC programs serve diverse, low income neighborhoods. In Maryland, a family's adjusted income must be under two times the official United States poverty level to qualify for Medicaid. Seventy-eight percent of patients in the Bayview clinic were insured through medical assistance (Medicaid) versus 95% of patients at CMHC. Thus, the majority of children and youth were from a disadvantaged background. The clinics also both serve diverse neighborhoods. The children at Bayview are 45% Caucasian, 23% African American, and 12% Hispanic, 20% multiracial and other races; the majority of the children at CMHC are African American (90%).

Information regarding demographics (age, sex, and race), weight, height, clinic, current psychiatric diagnoses, current psychotropic medications, and Binge Eating Disorder symptoms (C-BEDS Scale)¹⁶ was collected. Weight and height had been measured at the time of clinical intake evaluation using a triple beam scale, which was calibrated monthly. The study protocol was approved by the Johns Hopkins School of Medicine Institutional Review Board.

Diagnoses of Psychiatric Disorders

Each child was clinically evaluated at the first intake visit to either of the previously described outpatient clinics (Bayview or CMHC). A standard clinical diagnosis of ADHD and other psychiatric disorders was established by a standard clinical diagnostic evaluation, using the *Diagnostic and Statistical Manual-IV Text Revision (DSM-IV TR)* criteria¹⁷ and recorded on a standard

clinical intake form. The diagnostic procedure included an interview with the child and parents/caregivers and reviewing any available medical and psychiatric ratings, including ADHD scales when available. Diagnosticians were all Johns Hopkins University full-time faculty who are board certified Child and Adolescent Psychiatrists, (with at least 3 years of experience diagnosing and treating children with ADHD and psychiatric illnesses), or Child and Adolescent Psychiatry fellows supervised by these board certified Child and Adolescent Psychiatrists. Seven of the 8 board-certified child and adolescent psychiatrists had been trained in structured assessments such as the Schedule for Affective Disorders and Schizophrenia for school-age children- Present and Lifetime Version (K-SADS); although the design of this study performed in community clinics precluded the use of the K-SADS. A structured checklist was used to record formal diagnostic criteria for ADHD according to DSM-IV criteria. Diagnosticians were blind to study aims.

Diagnosis of Binge Eating

The *Children's Binge Eating Disorder Scale (C-BEDS)*¹⁶ was completed upon each child's clinical intake evaluation to the outpatient clinics. The C-BEDS uses a simple, structured, brief interview to screen and diagnose children based on provisional criteria for pediatric Binge Eating Disorder (BED).¹⁸ There is a significant association between the C-BEDS and the Structured Clinical Interview for DSM Disorders (SCID) measure and both measures identified children with binge eating. This scale is used in community mental health clinics to screen for BE. This seven-item scale consists of five "yes or no" questions addressing binge eating behaviors, with one question addressing duration, and one "yes or no" question addressing the presence of purging behaviors. Children receive a diagnosis of full BED who: (1) affirmed questions 1 and 2 (referring to eating in the absence of hunger and inability to stop eating respectively), (2) affirmed at least one of questions 3, 4, or 5, (referring to eating due to low mood, eating due to reward or hiding food respectively), (3) endorsed a frequency of at least 3 weeks, and (4) denied purging behaviors. Unfortunately the C-BEDS lacks frequency criteria which would be necessary for a clinical diagnosis of BED. Therefore, for the purposes of this study, a diagnosis of BED on the C-BEDS was renamed as binge eating (or BE) to create a more conservative diagnostic category.

Assessment of Body Mass Index (BMI) and Overweight/Obese

Height and weight were used to calculate the BMI for each participant. Those with a BMI Percentile at or above 85 were categorized as overweight, while those with a BMI Percentile at or above 95 were categorized as obese.

BMI *z*-scores, which are transformations of the percentile BMI to a standard deviation scale, were also performed to compare youths of different weights.¹⁹

Data Analysis

Exploratory analyses of demographic data were performed using chi square and *t*-tests. Associations among variables were assessed via either linear or logistic regression models. All analyses were adjusted for clinic site, age, sex, race, and BMI *z*-score.²⁰ BE, ADHD, and stimulant use were treated as dichotomous variables. ADHD was also analyzed as a dichotomous variable. Statistical analyses were performed using STATA 11.²¹ We regressed BMI *z*-score, ADHD, and BE in separate regressions, and subsequently together. Further, we repeated these analyses, adjusting for stimulant use.

Results

Demographic characteristics of the sample are described in **Table 1**. BMIs were available for 96% (241/252) of the sample. The mean BMI *z*-score of the sample was 0.9 (1.15 SD). Nineteen percent (47/252) were overweight and 30% (75/252) were obese. Twenty-four percent (61/252) of children were diagnosed with an anxiety disorder (including any of: generalized anxiety disorder, panic disorder, social anxiety disorder, separation anxiety disorder or post-traumatic stress disorder). Sixty-nine percent of the children with an anxiety disorder did not have ADHD (42/252) ($\chi^2 = 4.8, p = .03$).

Forty-three percent (109/252) of the children presented with ADHD. Fifteen percent (16/109) of the children presented the inattentive subtype by DSM-IV criteria; the remainder presented the combined subtype of ADHD. Forty-four percent (48/109) of children with ADHD were taking stimulants at this first intake evaluation. Children with ADHD were more likely to be male (72/109), ($\chi^2_1 = 13.59, p < .001$). Twenty-six percent of children with ADHD (28/109) exhibited BE, compared to 2% (3/143) of children without ADHD ($\chi^2_1 = 31.91, p < .001$). Among children with ADHD, 26% (28/109) exhibited BE in contrast to 74% (81/109) who did not exhibit BE. Among ADHD children with calculable BMIs, 28% (30/109) were overweight and 35% (38/109) were obese, as compared to non-ADHD children, of whom 12% (17/143) were overweight, and 25% (36/135) were obese. Children with ADHD were more likely to be overweight or obese ($\chi^2_3 = 18.11, p < .001$).

Twelve percent ($n = 31$) had BE (met full C-BEDS criteria). Children with BE were more likely to have depressive disorder ($\chi^2 = 4.25, p = .04$), but were

TABLE 1. Sample characteristics, ADHD, and BE status (N = 252)

Variable	Overall	ADHD+	ADHD–	Significance test ^a	BE+	BE–	Significance test ^a
N (%)	252	109 (43)	143 (57)		31 (12)	221 (88)	
Age (mean/SD)	10.8 (3.7)	10.9 (3.5)	10.8 (3.9)	–0.12 (0.904)	12.5 (3.3)	10.61 (3.7)	–2.67 (.008) ^b
Gender				13.59 (<.001) ^b			3.18 (.075)
Male	133 (52.8)	72 (66.1)	61 (42.7)		21 (67.7)	112 (50.7)	
Female	119 (47.2)	37 (33.9)	82 (57.3)		10 (32.3)	109 (49.3)	
Race				5.31 (.379)			8.41 (.135)
Caucasian	83 (32.9)	32 (29.4)	51 (35.7)		11 (35.5)	72 (32.6)	
African–American	145 (57.5)	66 (60.6)	79 (55.2)		14 (45.2)	131 (59.3)	
Other	24 (3.9)	11 (10.1)	13 (9.1)		6 (19.4)	18 (8.1)	
Clinic				2.96 (.085)			3.49 (0.062)
Bayview	115 (45.6)	43 (39.5)	72 (50.4)		19 (61.3)	96 (43.4)	
CMHC	137 (54.4)	66 (60.6)	71 (50.0)		12 (38.7)	125 (56.6)	
BMI							
BMI z-score	0.87 (1.2)	1.17 (1.1)	0.64 (1.2)	–3.61 (<.001) ^b	1.70 (1.1)	0.75 (1.1)	–4.44 (<.001) ^b
BMI Percentile	71.8 (27.3)	78.9 (24.5)	66.2 (28.1)	–3.70 (<.001) ^b	87.5 (19.2)	69.4 (27.6)	–3.52 (<.001) ^b
Obese	75 (29.8)	38 (34.9)	37 (25.9)	2.39 (.122)	16 (51.6)	59 (26.7)	8.07 (.004) ^b
Depressive Disorder	74 (29.4)	27 (24.8)	47 (32.9)	1.95 (.162)	14 (45.2)	60 (27.2)	4.25 (.039) ^b
Medications							
Stimulant	52 (20.6)	48 (44.0)	4 (2.8)	64.23 (<.001) ^b	12 (38.7)	40 (18.1)	7.05 (.008) ^b
Antidepressant	41 (16.3)	20 (18.4)	21 (14.7)	0.61 (.435)	8 (25.8)	33 (14.9)	2.36 (.124)
Antipsychotic	16 (6.4)	4 (3.7)	12 (8.4)	2.32 (.128)	2 (6.5)	14 (6.3)	0.0006 (.980)
Mood Stabilizer	4 (1.6)	1 (0.9)	3 (2.1)	0.55 (.458)	0 (0.0)	4 (1.8)	0.57 (.450)

^aAll values are pearson chi² (*p* value) except for Age, BMI z score and BMI Percentile, which are *t*-test (*p* value).

^bSignificant values. ADHD, attention-deficit/hyperactivity disorder; BE, binge eating; BMI z-score, body mass index z-score; BMI percentile, body mass index percentile.

not more likely to have an anxiety disorder ($\chi^2 = 0.12$, $p = .7$) than those without BE. Children with BE were more likely to be taking stimulants ($\chi^2_1 = 7.05$, $p = .008$). Both children with BE ($t_{239} = -4.44$, $p < .001$) and ADHD ($t_{239} = -3.61$, $p < .001$) were significantly more likely to have a higher BMI z-score. In terms of weight status, those children who were prescribed antidepressants had a significantly higher BMI z-score ($\beta = 0.53$, $p = .008$, 95% confidence interval (95% CI) 0.14–0.92).

ADHD, BE, and BMI Model

The relationship between ADHD and BMI was 0.54 after adjusting for clinic ($\beta = 0.54$, CI 0.25–0.83). The relationship between BE and BMI was 0.95 after adjusting for clinic ($\beta = 0.95$, CI 0.52–1.37). The association between ADHD and BE symptoms was statistically significant (OR 16.13, $p < .001$, CI 4.75–54.73), and persisted after adjusting for comorbid diagnoses, medications, demographic variables, BMI z-score, and clinic (Table 2). There was a statistically significant association between BE and BMI z-score ($\beta = 0.95$, $p < .001$, CI 0.52–1.37), and between ADHD and BMI z-score ($\beta = 0.54$, $p < .001$, CI 0.25–0.83). After adjusting for BE, the relationship between ADHD and BMI z-score persisted, but was attenuated ($\beta = 0.35$, $p = .03$, CI 0.04–0.66), and the coefficient for BE was decreased ($\beta = -0.75$, $p = .001$, CI 0.30–1.21).

TABLE 2. Association between ADHD and binge eating

Association ^a	OR	95% CI	<i>p</i> -value
Crude Association	16.1	4.8–54.7	<.001
Adjusted for:			
Demographics ^b	18.3	5.1–65.2	<.001
Comorbid disorders ^c	25.7	7.1–93.2	<.001
BMI z-score ^d	15.5	4.4–54.7	<.001
Medications ^e	22.8	5.8–89.0	<.001
Demographics, disorders, medications	21.9	4.8–99.6	<.001

ADHD, attention-deficit/hyperactivity disorder; OR, odds ratio; CI, confidence interval.

^aLogistic regression analyses were conducted. All models with the exception of the crude association were adjusted for clinic.

^bDemographic variables adjusted for included: age, sex, race.

^cComorbid Disorders adjusted for included: Depressive Disorder, Anxiety Disorder and Disruptive Behavior Disorder.

^dBMI z-score, body mass index z-score.

^eMedications adjusted for included: stimulants, antidepressants, antipsychotics, and mood stabilizers. The only medication that was significant was stimulant medication.

Stimulants

The relationship between ADHD and stimulants was 27.01 after adjusting for clinic (OR 27.01, CI 9.31–78.37). Stimulant use was associated with a three-fold increase in odds of having BE (OR 3.16, $p = .006$, CI: 1.39–7.17), but was not associated with greater BMI z-score ($\beta = 0.18$, $p = .33$, CI –0.18–0.54), after adjusting for clinic.

The relationship between BE and BMI z-score ($\beta = 0.94$, $p < .001$, CI 0.51–1.37) persisted after adjusting for stimulants. Further, the association

between ADHD and BMI *z*-score persisted after adjusting for BE and stimulant use ($\beta = 0.43$, $p = .02$, CI 0.08–0.78), and BE also continued to be a significant predictor of BMI ($\beta = 0.75$, $p = .001$, CI 0.30–1.21). The relationship between BE and ADHD persisted after adjusting for stimulant use (OR = 12.26, $p < .001$, CI 3.23–46.51).

Gender and Age

With regards to gender, males with BE had a mean BMI *z*-score of 1.85 (SD = 0.24) versus males without BE who had a mean BMI *z*-score of 0.68 (SD = 0.11) ($t_{125} = 4.35$, $p < .001$). For females with BE, mean BMI *z*-score did not differ significantly between BE [BMI *z*-score of 1.38 (SD 0.92)] and non-BE children [BMI *z*-score of 0.82 (SD 1.1)], ($t_{112} = -1.55$, $p = .12$). BE was significantly associated with increased BMI *z*-score in males but not females; however when the model was fit with an interaction term between bingeing and sex the interaction was not significant [$\beta = -0.59$, $p = .19$, CI (–0.5–0.31)].

With regards to age, there was no significant difference between children 12 and under and older children ($t_{250} = -0.056$, $p = .23$), when the experience of loss of control (or the inability to stop eating) was examined (question 2 of the C-BEDS).

Discussion

In this retrospective study of two pediatric community mental health centers, ADHD was significantly associated with BE. Children with ADHD were also more likely to be overweight or obese than those without ADHD. Additionally, the association between ADHD and obesity remained significant (although attenuated) after accounting for BE in this pediatric sample. Although there is literature to support an association between children with ADHD and obesity,⁸ less is known regarding the relationship between children with ADHD and BE. This preliminary study suggests that BE, particularly in children with ADHD, is an issue in need of further research.

Our model examined the complex cross-sectional relationship between ADHD, BE, and BMI in children receiving treatment at two mental health clinics. We found the relationship between ADHD and BE was significant; a child with ADHD had 16 times the odds of presenting BE than a child without ADHD. Although rates of BE were much higher (26%) in children with ADHD than in children without ADHD (2%), it is noteworthy that many children with ADHD did not have BE (74%).

This suggests that although children with ADHD had greater odds of BE, many children with ADHD didn't have BE. Perhaps children with both ADHD and BE represent a subgroup with different characteristics (such as inhibitory control deficits) than the larger group of ADHD children who do not have BE. Future studies should explore these hypotheses, which are beyond the scope of this study.

The relationship between ADHD and BMI *z*-score was significant and may partially be affected by BE. Although this cross-sectional study cannot explore causality, our data tend to support the hypothesis that binge eating may partially mediate the relationship between ADHD and obesity. However, mediation cannot be formally assessed in this study. A large epidemiologic study of adults⁵ and a community-based study of adults¹⁴ have previously reported that the relationship between obesity and ADHD was partly explained by binge eating. Perhaps there is an independent pathway from ADHD to obesity or some BE behavior that may somehow contribute to obesity. Impulsivity represents one possible factor that may influence ADHD and obesity,²² although other variables are possible.

Associations between ADHD and obesity may also be influenced by stimulant treatment of ADHD; however the effect of such pharmacological treatment on BE is less clear. Although stimulants were associated with three times the odds of BE in this study, they were not significantly associated with an increase in BMI *z*-score. This study did not find that stimulant medication use was associated with obesity (after adjusting for ADHD and BE). Our findings contrast with an epidemiological report that found that youth with unmedicated ADHD were as likely (or more likely) than children without ADHD to be overweight or obese.²³ Similarly, a recent epidemiologic study also reported that children with ADHD who are medicated long-term with stimulants are more likely to be obese.²⁴ It is conceivable that while stimulant medications may decrease BE by suppressing appetite, they may also increase BE after the appetite-suppressant effect wears off. Stimulants were significantly associated with BE behavior in this study; however, stimulant use did not appear to moderate the association between BE and ADHD.

Impulsivity, a core feature of ADHD, has emerged as an important concept in the realm of eating behavior and obesity. Impulsivity, inattention and deficient inhibitory control, which can be found in ADHD, have been suggested to contribute to abnormal eating behaviors and obesity in

children.^{4,25} Evolutionarily it may have been advantageous to impulsively eat whatever food might be found; however this is not the case in today's modern society.²⁶ Overweight adults have also been shown to be less able to inhibit their responses than nonoverweight adults and seem more sensitive to reward, particularly with regards to palatable food.^{27,28} These deficits may be particularly salient for adults presenting with binge eating behaviors. Binge eating in adults has been reported to be associated with greater impulsivity as compared with controls.²⁹ A recent study in adolescents described a greater increase in impulsivity and loss of control eating after a social exclusion paradigm, in addition to stronger reactivity of negative mood.³⁰ Future prospective studies should examine the possible role of impulsivity as a hypothesized factor that may influence children with both BE and ADHD.

There may be tendencies for inhibitory deficiencies in overweight children that are similar to those found in adults. Obese children have been found to exhibit heightened reward sensitivity, decreased inhibitory control, and increased impulsivity when compared to children of normal weight. A recent study examining inhibitory control in children suggested that high impulsivity was linked with obesity.³¹ Impulsivity and reward sensitivity have specifically been found to influence BMI through overeating³² and high levels of impulsivity may be associated with a rebound effect after eating.⁷ Impulsivity has also been linked to weight loss failure, with more impulsive children losing less weight. Preadolescents with higher BMIs preferred immediate smaller rewards over larger delayed rewards indicating a deficit in delayed gratification³³ that may also contribute to weight gain. Additionally, obesity might lead to ADHD symptoms of inattention and impulsivity through sleep-disordered breathing such as sleep apnea.³⁴

Limitations

A limitation of this study is that the data were retrospective in nature. The clinical sample was collected from an urban population of primarily low socioeconomic status, which may also limit the generalizability of the results when comparing to groups of higher socioeconomic status. This study was also conducted in mental health clinics which could be different from community samples and potentially also limit the generalizability of findings. Despite these limitations, there are few pediatric obesity or binge eating studies in community mental health settings that focus on a large and diverse population.

Although there was no standardized measure of ADHD or other psychiatric diagnoses, an expert board certified Child and Adolescent Psychiatrist established the diagnoses. All our psychiatric interviewers used the same checklist for diagnoses, were experienced and had similar training. Additionally it is not known how well standardized diagnostic interviews (SDIs) generalize in community settings.³⁵ We recognize that this method might not be considered the gold standard for ADHD research, however given the community setting and expert diagnosticians; we believe these to be valid evaluations. Since the evaluations were not recorded, reliability could not be established. Although interviewers were blind to study hypotheses; these interviewers knew if the children had an eating or psychiatric diagnosis. The results need to be replicated using prospective semistructured interviews that establish diagnosis and severity of BED, BE, and ADHD.

The C-BEDS screening diagnostic tool does not contain measures for binge frequency or binge amount, and has few available psychometrics, but it served as a rough screening tool for binge eating behaviors rather than a means to assign the full diagnoses of Binge Eating Disorder. The study design did not allow for measures of symptom severity; however, the goal of the study was to study the presence or absence of binge eating behavior rather than behavioral severity.

Given the cross-sectional nature of this study, we cannot establish a causal link between ADHD, obesity and BE; longitudinal studies should be performed to ascertain causality. Although we cannot conclude any causal hypotheses, these preliminary data may inform future studies that can investigate temporality.

Conclusions

Children and adolescents in community mental health clinics who present with ADHD may also present increased BMI-*z* scores and disinhibited eating such as BE. ADHD was associated with increased odds of BE, which in turn was associated with higher BMI *z*-score in community mental health settings. Although mediation cannot be formally assessed in this cross-sectional study, our findings suggest that the relationship between ADHD and BMI *z*-score may be partially, but not completely mediated by BE. Stimulant use does not appear to be associated with BMI *z*-score after controlling for ADHD and BE. Future prospective, longitudinal studies should elucidate the complex

relationships between ADHD, stimulant use, eating behaviors, and BMI *z*-score. Results should be replicated using prospective semi-structured interviews that establish research diagnoses and severity of BE and ADHD. In terms of clinical implications, our findings suggest mental health clinicians should consider screening for elevated BMI *z*-scores and BE in children with ADHD symptomatology who present to pediatric mental health clinics.

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