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ORIGINAL ARTICLE

Association between symptoms of attention-deficit/hyperactivity disorder and bulimic behaviors in a clinical sample of severely obese adolescents

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Objective: Preliminary evidence suggests a comorbidity between attention-deficit/hyperactivity disorder (ADHD) and obesity. This study was carried out to identify the clinical characteristics of obese adolescents with a higher probability of ADHD and advance the understanding of the potential factors underlying the comorbidity between obesity and ADHD. We evaluated the association between ADHD symptoms and bulimic behaviors, depressive and anxiety symptoms, degree of obesity, pubertal stage, age and gender in a clinical sample of obese adolescents.
Design: Cross-sectional study.
Subjects: Ninety-nine severely obese adolescents aged 12–17 years.
Measurements: Subjects filled out the Bulimic Investigatory Test, Edinburgh, the Beck Depression Inventory and the State-Trait Anxiety Inventory for Children. Their parents completed the Conners Parent Rating Scale, which assesses ADHD symptoms. The degree of overweight was expressed as body mass index-*z* score. Puberty development was clinically assessed on the basis of Tanner stages.
Results: Bulimic behaviors were significantly associated with ADHD symptoms after controlling for depressive and anxiety symptoms. The degree of overweight, pubertal stage, age and gender were not significantly associated with ADHD symptoms.
Conclusion: Obese adolescents with bulimic behaviors may have a higher probability to present with ADHD symptoms independently from associated depressive or anxiety symptoms. The degree of overweight, pubertal stage, age and gender might not be useful for detecting obese adolescents with ADHD symptoms. Therefore, we suggest systematic screening for ADHD in obese adolescents with bulimic behaviors. Further studies are needed to understand which specific dimension of ADHD primarily accounts for the association with bulimic behaviors. Future research should also investigate the causal link between bulimic behaviors and ADHD and explore potential common neurobiological alterations. This may lead to a better understanding of the effectiveness of stimulants for the treatment of bulimic behaviors in obese subjects.
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Keywords: ADHD; bulimic behaviors; eating disorders; children-adolescents

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a highly prevalent psychiatric disorder, estimated to affect 5–10% of school-aged children and 4% of adults.[1,2] According to the fourth edition of the diagnostic and statistical manual of mental disorders (DSM-IV),[3] ADHD is characterized by a persistent pattern of inattention, hyperactivity/impulsivity or both. Affected patients have difficulties focusing and sustaining attention, modulating activity level and controlling impulses and emotions.[4] DSM-IV defines three subtypes of ADHD: 'predominantly inattentive', 'predominantly hyperactive–impulsive' and 'combined'.[3] Onset before the age of seven and impaired functioning in two or more

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settings are essential for the diagnosis.[5] Commonly reported comorbid disorders include oppositional defiant disorder, conduct disorder, depressive disorders, anxiety disorders, speech and learning disorders.[2]

To date, the comorbidity between ADHD and obesity has been overlooked.[6] As it is often assumed that a low level of physical activity is involved in the etiology of obesity, one might think that ADHD (or at least the hyperkinetic component of this disorder) may be underrepresented in obese patients.[7] However, preliminary evidence surprisingly suggests a potential relationship between ADHD and obesity in clinical samples of obese adults and children seeking treatment. In a group of 215 adult patients treated for obesity, Altfas [8] found that 27.4% had ADHD (diagnosed according to DSM-IV criteria) and 33.5% presented with 'subthreshold' ADHD symptoms (i.e. symptoms of inattention and hyperactivity/impulsivity not meeting DSM-IV diagnostic criteria of ADHD). The prevalence of ADHD was particularly significant in patients with the highest degree of obesity: 42.6% of extremely obese subjects were diagnosed with ADHD. Holtkamp *et al*.[7] reported that the mean body mass index (BMI)-standard deviation score of a clinical sample of ADHD boys was significantly higher than the age-adapted reference values. In their sample, significantly more subjects than expected had a BMI $\geqslant$90th percentile (19.6%) and $\geqslant$97th percentile (7.2%). In a sample of 26 children hospitalized for the treatment of their obesity, Agranat-Meged *et al*.[6] found that over one-half (57.7%) had ADHD according to DSM-IV criteria. From a slightly different perspective, in a sample of 108 women with seasonal affective disorder, Levitan *et al*.[9] found a significant correlation between attentional problems in childhood and maximum lifetime BMI ($r=0.35$, $P=0.001$, $N=96$), after excluding subjects with extreme obesity (BMI >40kg/m^2), which may represent a genetically distinct subgroup.

The mechanisms explaining the link between obesity and ADHD have not been fully explored. Bazar *et al*.[10] reported several hypotheses, suggesting that obesity and ADHD may share etiologic pathways. Among the potential mechanisms linking ADHD and obesity, the 'reward deficiency syndrome' may play a key role. This syndrome is characterized by an insufficient dopamine-related natural reward that leads to the use of 'unnatural' immediate rewards, such as substance use, gambling, risk taking and inappropriate eating.[11] Several lines of evidence suggest that the patients with ADHD may present with behaviors consistent with the 'reward deficiency syndrome'.[12–15] In particular, the compromised regulation of impulses as well as the poor planning and organization described in patients with ADHD may lead to abnormal eating behaviors, such as bulimic behaviors, which, in turn, may be associated with obesity.[16,17] Therefore, bulimic behaviors may mediate, at least in part, the association between ADHD and obesity. To date, however, the relationship between ADHD symptoms and bulimic behaviors in obese patients has not been specifically evaluated. Exploring this association may be of relevance both to help identify obese patients with a higher probability of ADHD and to understand the potential mechanisms underlying the link between ADHD and obesity. As bulimic behaviors may be associated with anxiety and depressive symptoms[18–20] and depressive or anxiety symptoms may mimic ADHD symptoms,[4] it is important to control for depressive and anxiety symptoms when assessing the potential association between ADHD symptoms and bulimic behaviors.

To better characterize obese patients who may present with ADHD, further investigation is needed on other characteristics that might be associated with the severity of ADHD symptoms. To our knowledge, no study has specifically explored the potential association between the degree of obesity, age or gender and severity of ADHD symptoms in obese adolescents. Moreover, preliminary evidence from neurobiological studies suggests that hormonal factors may modulate ADHD symptoms.[21–24] However, the relationship between pubertal stage and severity of ADHD symptoms has not been investigated in obese subjects.

Therefore, in order to identify the clinical characteristics of obese adolescents with a higher probability of ADHD and advance the understanding of the potential mechanisms underlying the comorbidity between obesity and ADHD, the aims of this study were as follows:

(1) to assess symptoms of ADHD in a clinical sample of obese adolescents and
(2) to evaluate the association between ADHD symptoms and bulimic behaviors, depressive symptoms, anxiety symptoms, degree of obesity, pubertal stage, age and gender.

Materials and methods

Subjects

The study sample consisted of severely obese adolescents participating in a 6- to 11-month weight loss program in the residential Pediatric Therapeutic Center of Margency, in the Paris area. Criteria for the inclusion in the weight loss program were as follows: (1) seeking treatment for obesity, (2) a BMI above the 97th percentile and (3) age between 12 and 17 years. Exclusion criteria for our study were as follows: (1) symptomatic obesity from a polymalformation or a neuroendocrine syndrome and (2) patients or their parents unable to provide the requested information.

From January 1996 to December 1999, 106 patients were treated in the residential Pediatric Therapeutic Center of Margency. All of them were asked to participate in our research protocol. Ninety-eight percent of the patients agreed to participate. Five subjects met the exclusion criteria. Therefore, the final study sample consisted of 99 subjects.

All subjects were medication-free. After exclusion criteria, none of the subjects included in the study presented with other previous or current diseases.

All subjects and their parents gave written consent to participate in the study. The assessment protocol was approved by the appropriate institutional review board from Bichat-Claude Bernard Hospital, Paris.

Procedure

A senior pediatrician from the residential Pediatric Therapeutic Center of Margency conducted a pediatric consultation before the inclusion in the weight loss program. The pediatric assessment included measures of weight and height, stage of pubertal development, physical examination and laboratory screening to exclude associated organic etiology.[25] Height was measured to the nearest 0.5 cm on a standardized height board. Weight was rounded to the nearest 0.1 kg on a standard physician's beam scale, with the subject dressed only in light underwear and no shoes. The BMI was calculated as weight (kilograms) divided by height (meters) squared. Subjects with a BMI above the 97th percentile were included in the protocol. National BMI charts were used as the reference.[26] BMI values were standardized (BMI-*z* scores) using age- (to the nearest 6 months) and sex-specific median, s.d. and power of the Box–Cox transformation (LMS method) based on national norms.[26] BMI-*z* scores were used as an index of the degree of obesity. Puberty development was clinically assessed on the basis of Tanner stages.[27] Laboratory screening included measures of TSH, T3, T4, GH, FSH, LH, AST/ALT, cortisol, testosterone, estradiol, blood glucose, creatinine and insulin levels.

The psychiatric assessment portion of the research protocol was performed at the outpatient clinic of the Service of Child and Adolescent Psychopathology of the University Hospital Robert Debré, in Paris, at the beginning of the weight loss program. Demographic data (age, sex, ethnic group, socio-economic status (SES)) as well as familial and clinical history of the subjects were directly obtained from the parents and the subjects by an experienced child psychiatrist during the first consultation of the research protocol. Each subject was asked to complete the following questionnaires:

(1) *the Bulimic Investigatory Test, Edinburgh* (*BITE*):[28] it is a 33-item, self-reported questionnaire that was designed as an objective screening test to identify subjects with bulimic symptoms. The BITE consists of two subscales: the symptoms scale, which measures the level of the symptoms, and the severity scale, which provides a measure of the frequency of the symptoms. Results from the symptoms scale were analyzed in this study. The BITE has been translated into French;[29]
(2) *the Beck Depression Inventory* (*BDI*)[30] *13-item short form*: it is a highly reliable and valid measure of depressive symptoms. This scale has been used with adolescent populations. It has been translated into French and is used widely in France;[31] and *the State-Trait Anxiety Inventory for Children* (*STAIC*):[32] it measures the severity of anxiety symptoms in children. This questionnaire consists of two scales, each containing 20 items. The trait and state scales measure trait and situational anxiety, respectively. Data from the trait subscale were used for this study. The French version has been used in several studies.[33]

The parents of the subjects were asked to complete the Conners Parent Rating Scale (CPRS).[34] It is a widely used questionnaire that assesses ADHD symptoms. We used the CPRS-48 items, which contains 48 items scored on a four-point scale. Parents rate the frequency of their child's behavior on each item as occurring 0 = not at all, 1 = just a little, 2 = pretty much or 3 = very much. The CPRS provides an ADHD -index score, which covers symptoms of hyperactivity, impulsivity and inattention, as well as the following subscales: conduct problems, learning problems, psychosomatic problems, impulsive–hyperactive behaviors and anxiety. For the purposes of this study, we analyzed the scores on the impulsive–hyperactive subscale (CPRS-H–I subscale) and the ADHD index (ADHD index of the Conners Parent Rating Scale (*t*-scores)(CPRS-ADHD index)) score. The CPRS is the only ADHD rating scale translated into French and validated on a French childhood population.[35]

Statistical analysis

Demographic and clinical data (including age, weight, height, BMI, BMI-*z* scores, race, SES, stage of pubertal development and scores on questionnaires) were shown as means and s.d. or percentages.

The association between ADHD symptoms (inattention, impulsivity and hyperactivity) and bulimic behaviors, degree of obesity, anxiety and depressive symptoms, age, sex and pubertal stage was assessed using bivariate correlations (Pearson's or Spearman's when indicated) considering the following variables: CPRS-ADHD index scores, BITE scores, BMI-*z* scores, STAIC scores, BDI scores, age, sex and pubertal stage. To assess specifically the association between hyperactivity–impulsivity symptoms and bulimic behavior, degree of obesity, anxiety and depressive symptoms, age, sex and pubertal stage, bivariate correlations (Pearson's or Spearman's as appropriate) were performed considering the following variables: CPRS-H–I subscale scores, BITE scores, BMI-*z* scores, STAIC scores, BDI scores, age, sex and pubertal stage.

To evaluate the impact of bulimic behaviors on ADHD symptoms controlling for depressive and anxiety symptoms, a multiple linear regression was performed considering CPRS-ADHD index scores as the dependent variable and BITE scores, STAIC scores and BDI scores as independent variables. The impact of bulimic behaviors on hyperactivity–impulsivity symptoms controlling for depressive and anxiety symptoms was assessed using a multiple regression considering CPRS-H–I subscale scores as the dependent

variable and BITE scores, STAIC scores and BDI scores as independent variables.

A probability level of $P<0.05$ was used to indicate statistical significance. All statistical analyses were performed using the SPSS v. 13.0 software for Windows package for personal computers (SPSS, Inc., Chicago, IL, USA).

Results

Table 1 shows the demographic and anthropometric characteristics of the subjects. There was a significant difference between male and female subject with regard to age, height and BMI-*z* scores. Table 2 reports the mean scores on the questionnaires. Bivariate correlations showed that CPRS-ADHD index scores were significantly correlated with BITE scores ($r=0.234$, $P=0.020$). No other variable (BMI-*z* scores, STAIC scores, BDI scores, age, sex and pubertal stage) was significantly correlated with CPRS-ADHD index scores ($P>0.05$). As expected, BITE scores were significantly correlated to BDI scores ($r=0.499$, $P<0.001$) and to STAIC scores ($r=0.540$, $P<0.001$) and BDI scores were significantly correlated to STAIC scores ($r=0.615$, $P<0.001$). The CPRS-H–I subscale scores were not significantly correlated with any variable (BITE scores, BMI-*z* scores, STAIC scores, BDI scores, age, sex and pubertal stage) ($P>0.05$).

Multiple regression showed that, after controlling for STAIC and BDI scores, BITE scores were significantly correlated with the CPRS-ADHD index scores ($P=0.043$). Table 3 reports the standardized beta-coefficients, the standard error and the R^2 coefficient of the regression analysis.

Multiple regression confirmed that BITE was not significantly correlated with CPRS-H–I subscale scores after controlling for BDI and STAIC scores ($P>0.05$).

The degree of collinearity seems not to have led to erroneous conclusions as the coefficients of correlations were about 0.5–0.6 ($r=0.499$ for the correlation between BDI and BITE, $r=0.540$ for the correlation between STAIC and BITE and $r=0.615$ for the correlation between BDI and STAIC); moreover, s.d.'s of beta weights in the regression models were relatively low (<0.3).

Discussion

To our knowledge, this is the first study to assess the relationship between ADHD symptoms and bulimic behaviors in a clinical sample of obese adolescents. Our findings indicated that the CPRS-ADHD index scores were significantly associated with bulimic behaviors after controlling for depressive and anxiety symptoms. Obese adolescents with bulimic behaviors may thus have a higher probability to present with ADHD symptoms independently from associated depressive or anxiety symptoms.

Table 1 Demographic and clinical characteristics of the subjects

	Females	*Males*	*All adolescents*
N	65	34	99
Age (years)	14.5 (1.2)**	13.7 (1.1)	14.2 (1.2)
Weight (kg)	99.21 (16.10)	106.30 (22.78)	101.36 (18.50)
Height (cm)	164.04 (7.42)*	167.87 (9.35)	165.20 (8.85)
BMI (kg/m^2)	36.78 (5.57)	37.51 (5.94)	37.00 (5.67)
BMI-*z* scores	4.15 (0.72)**	4.74 (0.75)	4.33 (0.78)
Race			
Caucasian	55 (55.55)	28 (28.28)	83 (83.83)
African	8 (8.08)	5 (5.05)	13 (13.13)
Asian	2 (2.02)	1 (1.01)	3 (3.03)
SES			
High	15 (15.15)	7 (7.07)	22 (22.22)
Middle	22 (22.22)	7 (7.07)	29 (29.29)
Low	28 (28.28)	20 (20.20)	48 (48.48)
Puberty stage			
0	3 (3.03)	1 (1.01)	4 (4.04)
1	6 (6.06)	3 (3.03)	9 (9.09)
2	5 (5.05)	5 (5.05)	10 (10.10)
3	4 (4.04)	2 (2.02)	6 (6.06)
4	2 (2.02)	6 (6.06)	8 (8.08)
5	45 (45.45)	17 (17.17)	62 (62.62)

Abbreviations: BMI, body mass index; SES, socio-economic status. Significant difference: *$P<0.05$; **$P<0.01$. Data are shown as mean and s.d. or percentage.

Table 2 Scores on psychological measures

	Females	*Males*	*All adolescents*
BDI	6.3 (6.6)	8.2 (7.2)	6.9 (6.8)
STAIC	32.8 (8.3)	32.4 (7.6)	32.7 (8.1)
BITE	10.1 (5.1)	10.7 (5.6)	10.3 (5.2)
CPRS-ADHD index	54.3 (13.2)	52.9 (9.4)	53.9 (12.1)
CPRS-H–I subscale	51.2 (11.5)	48.1 (8.5)	50.2 (10.8)

Abbreviations: BDI, Beck Depression Inventory; BITE, Bulimic Investigatory Test, Edinburgh (Symptoms scale); CPRS-ADHD index, ADHD index of the Conners Parent Rating Scale (*t*-scores); CPRS-H–I subscale, Hyperactivity–impulsivity subscale of the Conners Parent Rating Scale (*t*-scores); STAIC, State-Trait Anxiety Inventory for Children (Trait subscale). Data are shown as mean and s.d.

Table 3 Multiple regression analysis: final model

	R^2	*Standardized beta-coefficients*	*s.e.*	P-*value*
Dependent variable: CPRS-ADHD index	0.07			
Independent variables				
BITE		0.246	0.288	0.043
BDI		0. 062	0.228	0.630
STAIC		−0.025	0.202	0.850

Abbreviations: BDI, Beck Depression Inventory; BITE, Bulimic Investigatory Test, Edinburgh (Symptoms scale); CPRS-ADHD index, ADHD Index of the Conners Parent Rating Scale (*t*-scores); s.e.; standard error; STAIC, State-Trait Anxiety Inventory for Children (Trait subscale).

It would be interesting to understand what specific dimension of ADHD (i.e. inattention, impulsivity or hyperactivity) primarily accounts for the association with bulimic behaviors. Unfortunately, the CPRS does not include a subscale specific for inattention and we are not aware of other scales validated in French that provide separate subscales of inattention, impulsivity and hyperactivity. However, the differential results obtained using the CPRS-ADHD index scores (which provides a measures of symptoms of inattention, impulsivity and hyperactivity taken together) and the CPRS-H–I allow us to make an hypothesis on the dimensions of ADHD mostly involved in the association. As reported previously, the CPRS-H–I subscale scores were not significantly associated with BITE scores. It is noteworthy that only one of the four items of the CPRS-H–I subscale refers to impulsivity. This, in addition to the significant correlation between the BITE and CPRS-ADHD index, leads us to infer that the association between ADHD symptoms and bulimic behaviors may be accounted for mostly by symptoms of inattention and impulsivity, whereas hyperactivity does not play a significant role in explaining the link. We suggest that further studies, using validated tools that allow separate measurement of the symptoms of inattention, hyperactivity and impulsivity, should test this preliminary hypothesis.

The cross-sectional nature of our study does not allow us to infer causality between symptoms of inattention/impulsivity and bulimic behaviors. One possibility is that impulsivity found in ADHD may lead or contribute to impulsive bulimic behaviors. Inattention may also contribute to bulimic behaviors. Schweickert *et al*.[36] hypothesized that compulsive eating may be a compensatory mechanism to help the person control the frustration associated with attentional and organizational difficulties. It is also possible that attentional and organizational deficit contribute to the difficulty in adhering to a regular eating pattern. Further studies are necessary to better assess the mechanisms explaining the link between abnormal eating behaviors and attentional difficulties.

On the other hand, it is also possible that bulimic behaviors may facilitate symptoms of inattention and impulsivity. Patients with bulimic behaviors, in fact, may present with repeated and impulsive interruptions of their activities in order to get food, resulting in disorganized activities and inattention.

Alternatively, a third factor may underlie both ADHD symptoms and bulimic behaviors. Preliminary evidence suggests that common neurobiological factors might be involved. Alterations in the dopamine receptor D2 (DRD2)[10] and, to a less extent, DRD4[37–39] have been associated with the above-mentioned 'reward deficiency syndrome', which include abnormal eating behaviors. Several studies suggest a role of altered DRD4 and DRD2 in ADHD as well (although the alterations in DRD2 has not been replicated in other studies).[40] Given the inconsistency in the above-mentioned genetic studies, the hypothesis of common genetic alterations in bulimic behaviors and ADHD must be considered with caution, at least at the present time. Another potential common neurobiological mechanism involves alterations in the brain-derived neurotrophic factor (BDNF) gene. Lyons *et al*.[41] found that in heterozygous $BDNF^{+/-}$ mice a partial impairment of BDNF expression caused impaired impulse control (which is a clinical feature found in children with ADHD) associated with aggressiveness and excessive appetite/food intake. Clearly, these data on animals need further replication in humans.

The understanding of the relationship between bulimic behaviors and ADHD symptoms in obese subjects may have relevant implications for the pharmacological treatment of bulimic behaviors in obese patients presenting with ADHD. Stimulants, including methylphenidate and methylamphetamine, represent the most widely prescribed medications for ADHD.[42] Several reports show the effectiveness of stimulants for the treatment of bulimia nervosa. Schweickert *et al*.[36] reported the effectiveness of methylphenidate on the binge eating component of bulimia nervosa in a 25-year-old woman with ADHD. In a double-blind study, Ong *et al*.[43] found that intravenous methylamphetamine was superior to placebo in treating eight patients with bulimia. Sokol *et al*.[44] described two patients with bulimia nervosa who responded to treatment with methylphenidate. Drimmer[45] reported the effectiveness of stimulants (methylphenidate, mixed salts of amphetamine and dextroamphetamine) in three patients with bulimia (two with comorbid diagnosis of ADHD according to DSM-IV criteria). Durkam[46] treated six patients with bulimia and ADHD with the stimulant dextroamphetamine; all the patients reported complete abstinence from binge eating and purging. Larger controlled studies are needed to confirm the effectiveness of stimulants in the pharmacological treatment of bulimia or other abnormal eating behaviors in adolescent samples. Moreover, further research is needed to understand the mechanisms explaining the improvement of bulimic symptoms with stimulants in obese subjects with ADHD symptoms.

In our study, degree of obesity was not significantly associated with CPRS-ADHD index scores. This suggests that, although ADHD may be more frequent among obese than non-obese patients, the degree of overweight may not be associated with severity of ADHD symptoms. However, most of our subjects had severe obesity and so it is possible that including subjects with a less severe obesity might have led to different results.

A lack of significant association was also found between gender and severity of ADHD symptoms. Biederman *et al*.[47] recently reported no significant differences between male and female subjects in the rate of any of the ADHD symptoms in a sample of non-referred children, suggesting that the gender differences identified previously in referred ADHD subjects[48] could represent an artifact of referral bias rather than true gender effects. Our data confirm this finding in a sample of obese adolescents who were not specifically referred for ADHD symptoms. As, in our study, female

subjects represented 65% of the total sample, studies of obese subjects with a more balanced sex ratio are necessary to confirm our finding.

We found that the CPRS-ADHD index (which measures hyperactivity, impulsivity and inattention) was not associated with age. This is in agreement with the notion that, although hyperactivity may be less evident when children with ADHD grow up,[49] they may still present with impairing ADHD symptoms. However, as we evaluated a sample of adolescent subjects, we cannot exclude that age would have been significantly associated with ADHD symptoms in a sample including younger children.

Pubertal stage was not associated with severity of ADHD symptoms. Most of the subjects (64.5%) had a Tanner stage = 5, so it is possible that results would have been different in a sample of subjects with a more heterogeneous distribution of Tanner stages. Although it has been reported that gonadal hormone metabolism may be involved in the clinical manifestation of ADHD symptoms, these data were obtained in animal models during the development of frontocortical neurons.[22] It is premature to extrapolate the results of these neurobiological studies to human adolescents. Therefore, further studies evaluating the effect of puberty on the expression of ADHD symptoms in obese adolescents are needed.

Several factors contribute to increase the strength of our results:

(a) the use of a standardized procedure (BMI-*z* scores) to assess the level of overweight;
(b) the use of well-validated psychological instruments; and the high rate of participation that minimizes sample selection bias.

However, some limitations of this study should be reported:

(a) we analyzed a clinical sample of severe obese patients, so our conclusions may not be extended to the general (non-clinic) population of obese patients. Therefore, epidemiological surveys considering the relationship between obesity and ADHD in the general population should be encouraged; and we assessed symptoms of ADHD and bulimic behaviors, not ADHD and bulimia according to standardized criteria of psychiatric international classifications. Therefore, further studies are welcome that evaluate the relationship between ADHD and eating disorders using international criteria.

In spite of these limitations, our study suggests that obese children with bulimic behaviors should be systematically screened for ADHD symptoms. Obese children with ADHD and bulimic behaviors might benefit from stimulant treatment. Larger controlled studies are necessary to confirm the effectiveness of stimulants to improve bulimic symptoms in obese patients with ADHD. The treatment of ADHD and bulimic symptoms in obese patients may improve their quality of life, leading to better family, school and general adaptive functioning.

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