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Title : Adulthood and childhood ADHD in patients consulting for obesity is associated with food addiction and binge eating, but not sleep apnea syndrome

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Abstract

Introduction: The exact mechanisms underlying the established association between ADHD and obesity remain unclear. Food addiction and binge eating may contribute to this link. We examined for the first time the association between childhood/adult ADHD and food addiction/ binge eating in patients with obesity, as well as the association between ADHD and sleep apnea syndrome.

Methods: We included 105 obese patients from the Nutrition Department of the University Hospital of Tours (France) between January and December 2014. We assessed categorical diagnoses of childhood/adulthood ADHD (semi-structured interview DIVA 2.0), food addiction (Yale Food Addiction Scale 2.0), binge eating (Binge Eating Scale), obstructive sleep apnea (clinical assessment), and BMI (clinical assessment).

Results: Patients with adult ADHD were at significantly higher risk of food addiction than patients without adult ADHD (28.6% vs. 9.1%; $p=.016$). Adult and childhood ADHD were significantly associated with self-reported food addiction, food addiction scores and binge eating scores, with a larger effect size for adult (ORs: 4.00[1.29-12.40], 1.37[1.14-1.65] and 1.08[1.03-1.14], respectively) than childhood (ORs: 3.32[1.08-10.23], 1.29[1.08-1.55] and 1.06[1.01-1.11], respectively) ADHD. ADHD diagnosis was not significantly correlated to obstructive sleep apnea. Mean age of onset of ADHD preceded mean age of onset of obesity.

Conclusion: ADHD diagnosis is associated with food addiction and binge eating, with a larger effect size for adult than childhood ADHD. Our results provide a strong rationale for further longitudinal research on the link between ADHD, food addiction, binge eating and obesity, paving the way for evidence-based therapeutic interventions for these patients.

Keywords: Attention Deficit Disorder with Hyperactivity; behavior, addictive; impulsive behavior; addictive-like eating; eating addiction; substance-related and addictive disorders.

Highlights:

- Childhood and adulthood ADHD are associated with obesity, but the underlying mechanisms are not well defined

We found that:

- Food addiction/binge eating were significantly associated with both childhood and adult ADHD diagnosis
- This association was stronger for adulthood than childhood ADHD
- ADHD was strongly associated with both food addiction and binge eating, with no difference in effect sizes.
- ADHD was not correlated to obstructive sleep apnea syndrome

1. Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder characterized by impairing levels of inattention and/or hyperactivity-impulsivity, that begins in childhood (by definition before the age of 12) and significantly interfere with the social, academic, or occupational functioning (American Psychiatric Association, 2013). Although some researchers initially asserted that ADHD usually remitted in adulthood (Hill & Schoener, 1996), current evidence indicates that impairing symptoms of the disorder persist in adulthood in up to 65% of the cases (Faraone, Biederman, & Mick, 2006; Kooij et al., 2010). The estimated pooled prevalence of ADHD in children and adults is 5% and 2.5%, respectively (Kessler et al., 2006; Simon, Czobor, Bálint, Mészáros, & Bitter, 2009). These findings led the field to better explore the course of ADHD during adulthood, and to better characterize the comorbidities associated with adult ADHD.

While the comorbidity between ADHD and psychiatric disorders has been extensively documented, the possible association with somatic comorbidities has received less attention (Cortese & Castellanos, 2014; Instanes, Klungsøyr, Halmøy, Fasmer, & Haavik, 2018). One of the most prevalent and disabling comorbidities associated with adult ADHD is obesity. Meta-analytic evidence has shown that the prevalence of obesity is increased by 70% in adults with compared to those without ADHD (Cortese et al., 2016). Although a significant association between ADHD and obesity is established, we currently lack an understanding of the exact neurobiological and psychopathological mechanisms underpinning this link (Cortese & Vincenzi, 2012; Hanć & Cortese, 2018). Assuming that ADHD precedes obesity, as showed by preliminary evidence (Cortese et al., 2016), such an understanding is however crucial to prevent as early as possible obesity in individuals with ADHD, by designing adequate preventive and therapeutic interventions targeting risk factors for obesity.

A number of mechanisms/factors have been proposed to explain the association between ADHD and obesity including genetic factors, fetal programming, executive dysfunctions, psychosocial stress, factors directly related to energy balance, and sleep patterns alterations (Hanć & Cortese, 2018). One of the proposed mechanisms includes factors related to energy balance such as reduced physical activity and abnormal eating patterns. More specifically, impulsivity and inattention of ADHD may contribute to weight gain via dysregulated eating patterns. In line with this hypothesis, previous studies demonstrated that childhood ADHD was associated with binge eating, binge eating disorder (Nazar, Bernardes, et al., 2016) and loss of control over eating (Hilbert et al., 2018), and adult ADHD is associated with binge eating severity (Brewerton & Duncan, 2016; Nazar, de Sousa Pinna, et al., 2016) and binge eating disorder (Alfonsson, Parling, & Ghaderi, 2013). With regards to alterations of eating patterns, a possibly relevant but so far unexplored mechanism is represented by food addiction. The concept of food addiction, proposed by Gearhardt et al. by adapting DSM-IV-TR substance dependence criteria (Gearhardt, Corbin, & Brownell, 2009a), and then, DSM-5 criteria for Substance-Related and Addictive Disorders (Gearhardt, Corbin, & Brownell, 2016) to food, postulates that some people may develop addictive-like eating behaviour to highly palatable foods (i.e., foods rich in sugar, fat and/or salt) (Davis & Carter, 2009; Gearhardt et al., 2016). On the one hand, food craving, which is a core feature of food addiction, is associated with weight gain (Contreras-Rodríguez, Martín-Pérez, Vilar-López, & Verdejo-García, 2017). On the other hand, ADHD is indeed a robust risk factor of addictive disorders such as tobacco, alcohol or cannabis use disorder (Luo & Levin, 2017) and gambling disorder (Fatséas et al., 2016; Grall-Bronnec et al., 2011). It is possible that ADHD is associated with a higher risk of food addiction, and this contributes to explain the subsequent weight gain and the higher risk for obesity in this population. Previous research conducted in a non-clinical population of students demonstrated that ADHD symptoms, as

assessed by self-administered questionnaires, were associated to higher prevalence for food addiction (Romo et al., 2018). However, to our knowledge, no study so far has explored the links between ADHD diagnosis, as assessed by a clinical interview, and food addiction in the specific population of individuals with obesity.

Additionally, studies focusing on the links between ADHD and obesity have generally failed to control for a possible important confounder, namely sleep apnea syndrome. Sleep apnea syndrome is indeed far more prevalent in persons with overweight or obesity than in the overall population (30-40% vs. 2-4%) (Schwartz et al., 2008), and this diagnosis may explain the higher prevalence of ADHD in patients with obesity (Hanć & Cortese, 2018). It was indeed proposed that obesity and the factors associated with it (e.g., sleep apnea syndrome and deficits in arousal/alertness) may manifest as ADHD-like symptoms (Cortese & Vincenzi, 2012): patients with sleep apnea syndrome may screen positive for ADHD symptoms, and sleep apnea symptoms may be mistakenly attributed to ADHD (Hanć & Cortese, 2018). To our knowledge, no study tested this hypothesis in patients with obesity.

To fill these gaps, we compared the prevalence for self-reported food addiction between patients with versus without adult ADHD diagnosis in a sample of adults patients with obesity. We hypothesized that obese patients with adult ADHD would have a significantly higher prevalence rate for self-reported food addiction than obese patients without adult ADHD. Our secondary objectives were: (1) to assess whether ADHD diagnosis was significantly related to sleep apnea syndrome; (2) to determine the associations between *childhood* ADHD, *adult* ADHD and food addiction; (3) to compare binge eating score, food addiction score and ADHD diagnosis. We hypothesized that: (1) adult ADHD would not be significantly related to sleep apnea syndrome, because we hypothesized that ADHD inattentive symptoms and sleep apnea symptoms are two different diseases; (2) ADHD status would be associated with both childhood and adult ADHD, with a larger effect size for adult

than childhood ADHD, because we assumed that remission of ADHD symptoms would lead to improved eating behavior; (3) adult ADHD would be associated with both binge eating and food addiction scores, with a larger effect size for food addiction than binge eating, because we assume here that the “food addiction” phenotype may identify a different, potentially more homogeneous, population of patients at-risk for obesity (i.e., binge eating symptoms rely on a certain amount of food eaten, while food addiction symptoms rely on loss of control and maintenance of a behavior despite adverse consequences).

2. Method

This protocol, which was specifically designed for this data collection, was approved by the Institutional Review Board of the University Hospital of Tours, France (IRB number: 2013-A00560-45). The research was conducted in accordance with the Helsinki Declaration as revised in 1989. All participants provided written informed consent after the procedure had been fully explained and prior to their inclusion in the study.

2.1. Participants and procedure

This cross-sectional study included all consecutively referred adult patients with severe obesity (body mass index ≥ 35 kg/m²) seen between January and December 2014 within the Nutrition Department of the University Hospital of Tours, France. Exclusion criteria was: obesity due to an endocrinal disease (e.g., polycystic ovarian syndrome). The study procedure included two phases: (1) all eligible participants received a postal mail inviting to participate in the study completing self-administered questionnaires, including the Yale Food Addiction Scale 2.0 and the Binge Eating Scale); (2) all patients who fully completed the self-

administered questionnaires of the first study phase were invited to participate to a consultation with a psychiatrist that aimed to diagnose ADHD with a semi-structured interview (one systematic recall if no answer after three days). The psychiatric assessments were conducted by psychiatrists trained in the DIVA 2.0 and ADHD assessments: JF, PM and PB.

2.2.Measures

2.2.1. Socio-demographics and medical characteristics

We collected data on demographics (age, gender), current body-mass index (BMI), previous maximal BMI, age of onset of obesity, duration of obesity, and history of previous bariatric surgery based on medical records. BMI was measured using a weighing scale suitable for patients with obesity. We also assessed the presence of a sleep apnea syndrome based on the clinical diagnosis of a physician expert in obstructive sleep apnea syndrome (systematic consultation for all patients). Obstructive sleep apnea syndrome is a common disorder characterized by repetitive episodes of nocturnal breathing cessation due to upper airway collapse; it causes severe symptoms such as excessive daytime somnolence, morning headaches, irritability, memory loss or decreased libido (Spicuzza, Caruso, & Di Maria, 2015).

2.2.2. Attention-deficit/hyperactivity disorder diagnosis

We used the Diagnostic Interview for ADHD in Adults, second edition, (Diagnostische Interview Voor ADHD bij volwassenes, DIVA 2.0). The DIVA 2.0 is a semi-structured diagnostic interview assessing adult ADHD based on the DSM criteria (Kooij & Francken, 2010). It is widely used by health professionals in Europe as well as in clinical research studies (Michielsen et al., 2012; Semeijn et al., 2013). It consists of two parts: the

first assesses the presence of each of the 18 items of the DSM-IV-TR criterion A for ADHD, both in childhood (primary school, before 7 years old (retrospective assessment) and at the current time (assessment at the current time of the interview); the second assesses impairment in five areas of functioning (work, education, family, social/relationships and self-confidence) due to ADHD, both in childhood and at the current time. Respondents were identified as 'cases' if they met the following criteria, in line with the ADHD DSM-IV-TR criteria: (1) six or more symptoms of inattention and/or six or more symptoms of hyperactivity-impulsivity, present for at least six months prior to assessment; (2) six or more symptoms of inattention and/or six or more symptoms of hyperactivity-impulsivity in childhood (before 7 years old); (3) clinically significant impairment in at least two areas of daily life (work, education, family, social & relationships, self-confidence), both during the past 6 months prior to the interview and in childhood (i.e., before the age of 7 years old) (Kooij & Francken, 2010). The DIVA 2.0 is a reliable tool for assessing and diagnosing adult ADHD, with an excellent diagnostic accuracy when compared to the Conners' Adult ADHD Diagnostic Interview for DSM-IV (CAADID) and with a high convergent validity with ADHD self-administered scales such as the Wender Utah Rating Scale and the adult ADHD self-report scale (Ramos-Quiroga et al., 2016).

2.2.3. Food addiction

We assessed food addiction using the French version of the Yale Food Addiction Scale 2.0, which is based on DSM-5 criteria (YFAS 2.0; original version: Gearhardt et al., 2016; French version: Brunault et al., 2017). The YFAS 2.0 is a 35-item self-administered questionnaire that identifies people exhibiting symptoms of addiction regarding specific types of foods (e.g., high fat and/or high sugar) over the past 12 months, relying on the DSM-5 criteria for Substance-Related and Addictive Disorders to food (Gearhardt et al., 2009a; Gearhardt,

Corbin, & Brownell, 2009b). The YFAS 2.0 enables the assessment of a number of DSM-5 symptoms occurred over the past 12 months (ranging from zero to eleven) and the “diagnosis” of food addiction when the participant report at least two food addiction symptoms during the previous 12 months plus clinically significant impairment or distress. Food addiction is then rated as mild, moderate or severe depending on the number of food addiction symptoms associated (cut-off: 2, 4, and 6, respectively) (American Psychiatric Association, 2013; Gearhardt, Corbin, & Brownell, 2016).

The YFAS 2.0 demonstrated good construct validity in both obese and non-obese individuals (Gearhardt et al., 2016; Meule, Müller, Gearhardt, & Blechert, 2017), including excellent good internal consistency ($\alpha=.90$), as well as good convergent and incremental validity (Gearhardt et al., 2016). In the present study, we considered self-reported food addiction (existence of at least two food addiction symptoms plus clinically significant impairment or distress) and food addiction severity (food addiction symptom count).

2.2.4. Binge eating

We assessed binge eating symptoms with the French version of the Binge Eating Scale (BES) total score, a 16-item questionnaire designed to assess severity of binge eating using behavioural, affective, and cognitive symptoms (original version: Gormally, Black, Daston, & Rardin, 1982; French version: Brunault et al., 2016). It is a reliable tool for assessing binge eating disorder symptoms and has been validated in French (Brunault et al., 2016).

2.3. *Statistical analyses*

Descriptive statistics included percentages for ordinal variables, and mean and standard deviation for continuous variables. To answer our primary objective, we used univariate logistic regression analysis to determine the links between self-reported food

addiction (YFAS 2.0, independent variable) and adult ADHD diagnosis (DIVA 2.0; dependent variable). To answer our secondary objectives, we used univariate logistic regression analyses to factors associated with childhood/adult ADHD (DIVA 2.0; dependent variable): food addiction score (YFAS 2.0; independent variable), binge eating score (BES total score; independent variable), sleep apnea syndrome (clinical assessment; independent variable). Results were presented as Odds-Ratio, its Confidence Interval at 95% [95% CI] and its associated *p*-value. Analyses were conducted using SPSS version 22 (IBM Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp.). All analyses were two-tailed; *p*-values $\leq .05$ were considered statistically significant.

3. Results

3.1. Study flow diagram

The study flow diagram is presented in Figure 1. Out of the 287 initial patients who were initially eligible, one was excluded due to a polycystic ovary syndrome, and 125 returned fully usable questionnaires (no missing data for the self-administered questionnaires of the first step of the study). Out of these 125 patients, 105 (final study population) accepted to undergo the DIVA assessment. The 105 patients who completed the study were significantly older than the 182 who did not (43.9 ± 10.8 years vs. 41.2 ± 11.4 years; $p=.045$), but there no significant difference in terms of gender (men were respectively 19% vs. 24.2%; $p=.32$), current BMI (47.4 ± 7.4 vs. 47.8 ± 7.6 ; $p=.70$), nor previous maximal BMI (50.1 ± 8.2 vs. 50.0 ± 8.2 ; $p=.90$).

3.2. Descriptive statistics and correlations between the variables

Table 1 presents the descriptive data of the study sample. Prevalence rate for adult ADHD, childhood ADHD and self-reported food addiction were respectively 26.7%, 35.2% and

14.3%.-Mean age in the whole sample was 46.4 ± 10.7 years (range: 27-69). In our study, the YFAS 2.0 had an excellent internal consistency ($\alpha=.91$), with a one-factor structure explaining 30.1% of the total variance. Cronbach's α for the BES was .89. The correlations between food addiction score, binge eating score, mean number of adult and childhood ADHD symptoms, as well as current BMI and age are presented in Supplemental Table 1. Food addiction score was correlated to binge eating symptoms, but 68.8% of the patients with significant binge eating had no food addiction, and 66.6% of the patients with food addiction had no significant binge eating.

3.3.Factors associated with adult ADHD diagnosis

As presented in Table 2, patients with adult ADHD were at significantly higher risk of food addiction than patients without adult ADHD (28.6% vs. 9.1%; $p=.016$), but they did not report higher prevalence of sleep apnea syndrome. Food addiction and binge eating scores were both significantly associated with adult ADHD status (respective ORs were 1.37[1.14-1.65], $p<.001$ and 1.08[1.03-1.14], $p=.004$). Although the 95% CI for food addiction was higher than the one for binge eating, the comparison between the two odds ratio indicated no significant differences ($p=.12$). Adult ADHD diagnosis was not related to age ($p=.94$), gender ($p=.86$), current body mass index ($p=.63$), previous maximal body mass index ($p=.61$), nor age of onset of obesity ($p=.09$).

3.4.Factors associated with childhood ADHD diagnosis

As presented in Table 3, patients with childhood ADHD were at higher risk for food addiction (24.3% vs. 8.8%) and binge eating than patients without childhood ADHD, but they did not report higher prevalence of sleep apnea syndrome. Food addiction and binge eating scores were both significantly associated with childhood ADHD status. Childhood ADHD diagnosis

was not related to age ($p=.92$), gender ($p=.52$), current body mass index ($p=.75$), previous maximal body mass index ($p=.98$), nor age of onset of obesity ($p=.17$). Overall, self-reported food addiction, food addiction score and binge eating scores were more strongly associated with adult ADHD than childhood ADHD.

4. Discussion

This study is the first study to demonstrate that obese patients with adult ADHD, as assessed by a clinical interview, have significantly higher prevalence of food addiction (categorical disorder) as well as of food addiction symptoms than patients without adult ADHD. In addition, ADHD diagnosis was associated with both binge eating symptoms and food addiction score, with a larger effect size for adult ADHD than childhood ADHD. Furthermore, ADHD diagnosis was not related to obstructive sleep apnea syndrome (OSA), ruling out the possibility that what we measured was a phenocopy of ADHD symptoms due to OSA.

According to Hanć & Cortese (Hanć & Cortese, 2018), the proposed mechanism underlying the association between obesity and ADHD include genetic factors, fetal programming, executive dysfunctions, psychosocial stress, factors directly related to energy balance, and sleep patterns alterations. Our study allowed testing some of these hypothesis associations, including the notion that the ADHD-related impulsivity and the ADHD-related inattention may lead to obesity via dysregulated eating patterns (factors related to energy imbalance). To our knowledge, no study investigated the prevalence of food addiction in patients diagnosed with adult ADHD. Studies on the association between ADHD and obesity have been conducted more frequently in children than in adults (Cortese et al., 2016). Studies in children/adolescents found a significant association between ADHD and binge eating, as well as loss of control over eating (Hilbert et al., 2018; Nazar, Bernardes, et al., 2016).

Studies in adults showed that ADHD was related to binge eating (Alfonsson et al., 2013; Brewerton & Duncan, 2016; Nazar, de Sousa Pinna, et al., 2016), but the majority assessed ADHD using self-administered questionnaires rather than a clinical interview. To our knowledge, the study assessing eating behaviors in adults with ADHD using a clinical interview is the one by Nazar et al. (2016). Interestingly, in our study, ADHD was associated with both food addiction symptoms and binge eating score. Given that there was no strict overlap between binge eating and food addiction, we can assume that these two measures may identify different sub-populations of ADHD patients at-risk for with obesity. Future research should determine the added value of food addiction over binge eating alone. Finally, we found that food addiction and binge eating were more strongly associated with adult ADHD than childhood ADHD, which lends support to the notion that remitted ADHD is associated with a lower risk of food addiction and to better outcome. To our knowledge, this has not been reported in previous research.

Our study also allowed testing another mechanism underlying the association between obesity and ADHD: obesity and/or the factors associated with it may manifest as ADHD-like symptoms. Although one previous study found no association between adult ADHD symptoms and sleep apnea syndrome (Oğuztürk, Ekici, Çimen, Ekici, & Senturk, 2013), there are no studies assessing ADHD using a clinical interview rather than a self-report questionnaire. Filling this gap, our study found no evidence for an association between sleep apnea syndrome and ADHD.

Our data also confirm that adult ADHD is highly prevalent in obese patients, and that these disorders are highly comorbid. The mean prevalence of adult ADHD was 26.7%, which is slightly higher than expected according to Cortese et al. meta-analysis (Cortese et al., 2016). Indeed, it should be noted that the meta-analysis by Cortese et al. excluded studies based on morbid obese seen in bariatric clinics, to avoid inflating the estimated prevalence of

ADHD. Our study population, constituted of severely obese patients, could explain this higher prevalence rate and our results are in line with those reported in a bariatric sample by Altfas, which indeed found a prevalence rate of 27.4% in obese patients, but 42.6% in morbidly obese patients ($\text{BMI} \geq 40 \text{ kg/m}^2$) (Altfas, 2002). Mean age of onset of obesity was lower (although not significantly), in adult ADHD than non-ADHD patients (14.9 vs. 18.9 years), and lower in adult ADHD than childhood ADHD (14.9 years vs. 15.9 years). In addition, mean age of onset of ADHD (before 12 years) preceded mean age of onset of obesity (14.9 years). Although one can assume that ADHD patients may represent, when untreated, a specific population with a higher risk of obesity and for an earlier obesity (B. S. Schwartz et al., 2014), our results failed to demonstrate such an association, probably because of lack of statistical power and/or because of our sample included only severely obese patients.

Our results should be seen in the light of study strengths and limitations. One of our study's strength includes the use of a clinical interview rather than self-administered questionnaires: we used the DIVA 2.0, which is a reliable tool for assessing and diagnosing adult ADHD (Ramos-Quiroga et al., 2016). Many researches in adults used self-administered questionnaires to assess ADHD instead of clinical interview, and this tends to overestimate the prevalence rate for childhood and adult ADHD, as well as late-onset ADHD, with many false positives. Other strengths were the assessment of both childhood and adult ADHD diagnoses, the assessment of both binge eating and food addiction (that are two different and complementary measures of eating behaviour), and the use of the YFAS 2.0 that is based on the extrapolation of the most recent DSM-5 criteria for addictive disorders.

Our study has also some limitations. Our sample was relatively small, included obese patients who consulted for obesity, and completers were slightly younger than non-completers, although not different in terms of gender or current BMI. Future studies should be conducted in patients with less severe obesity (i.e., non-seeking obese patients) to determine

whether we could generalize our results to the overall population of either obese and/or ADHD patients. In the current international diagnostic classifications, food addiction is not recognized as a standalone disorder, and some authors have questioned the validity of the concept and/or of its assessment, as it is currently assessed (Ziauddeen & Fletcher, 2013). Results from the YFAS have been found to correlate highly with other measures of disordered eating, and future studies should determine whether high scores are a marker of disordered eating problems generally or a specific assessment of “food addiction” (Price, Higgs, & Lee, 2015). Before potentially considering it as a standalone disorder, future studies should also determine the added value of food addiction over binge eating alone. Another important point is the need to account for psychiatric disorders because they could mediate the association between ADHD symptomatology and food addiction/binge eating (Kaisari, Dourish, Rotshtein, & Higgs, 2018): future studies should assess both ADHD and other psychiatric disorders using clinical interviews. We diagnosed sleep apnea syndrome using a clinical approach, and this may have lowered prevalence for sleep apnea syndrome when compared to polysomnography, which is the gold standard; our results should be confirmed using polysomnography to assess sleep apnea syndrome. Additionally, the cross-sectional design of our study precludes us from inferring a causal relationship between ADHD and obesity, but chronology of both disorders suggests that ADHD may precede obesity rather than the opposite. Finally, to demonstrate the validity of our theoretical model (i.e., food addiction may explain the transition between ADHD and obesity), one should first demonstrate using a longitudinal design that children with ADHD have a higher risk of food addiction and obesity than children without ADHD, and that treatment for childhood or adult ADHD would prevent obesity or delay its age of onset.

Despite these limitations, this study has important practical implications. First, obese patients diagnosed with food addiction should be systematically screened for a comorbid

ADHD, notably because such a dual diagnosis implies a more complex care plan. Screening and diagnosing ADHD in adults is a challenging area, as already stated in other populations (Grall-Bronnec et al., 2011; Kooij et al., 2010). Self-reported impulsivity is positively associated with BMI (Emery & Levine, 2017), and thus higher in obese patients (especially those with food addiction; Ouellette et al., 2017), and it is not always easy to disentangle between ADHD impulsive-hyperactivity symptoms and overall impulsivity symptoms (as would assess a self-administered questionnaire) without a specific training in ADHD. However, use a semi-structured interview enables a robust ADHD assessment. Another practical implication of our study is that obese patients with ADHD should be systematically screened for food addiction in addition to binge eating. Child and adolescent psychiatrists should systematically bear in mind that their patients with childhood ADHD might be at greater risk for later food addiction, even when ADHD remits, and clinicians should systematically follow up their patient's eating behaviour over a long-term period. Since ADHD children treated with stimulants had slower early BMI growth than non treated ADHD children (B. S. Schwartz et al., 2014), we might assume that adequate treatment for ADHD in childhood might prevent weight gain in adulthood. Future studies should determine whether children with persistent ADHD and adequate psychosocial and pharmacological management may develop a lower risk for obesity in adulthood.

In conclusion, we found evidence that obese patients with a diagnosis of adult ADHD have higher risk for food addiction and binge eating than patients without ADHD. Food addiction and binge eating may be important variables implicated in the association between ADHD and obesity. Future longitudinal studies should determine whether food addiction and/or binge eating could best explain the over-representation of obesity in ADHD patients. Such longitudinal studies should also assess whether an adequate and early management of

ADHD could prevent obesity or delay the onset of obesity in these patients, paving the way for evidence-based therapeutic interventions for these patients.

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The authors' contribution:

Study design: PB, JF, NB

Analysis: PB

Writing the manuscript: PB, JF, PM, AdL, RH, PHD, SC, NB

All authors have approved the final article.

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TABLES

Table 1. Descriptive data of the study sample (n=105)

	Mean \pm SD Percentage (n)
<i>Socio-demographic and weight-related characteristics</i>	
Age (years)	46.5 \pm 10.7
Gender (female)	86.7% (91)
Current Body Mass Index (BMI; kg/m ²)	46.9 \pm 7.8
Previous maximal BMI (kg/m ²)	50.1 \pm 7.9
Previous bariatric surgery (yes)	81% (85)
Age of onset of obesity (years)	17.8 \pm 10.4
<i>Presence of sleep-apnea syndrome</i>	28.6% (30)
<i>Adult ADHD (as per the DIVA 2.0)</i>	
Prevalence of adult ADHD (all subtypes)	26.7% (28)
Prevalence of mixed-subtype	17.1% (18)
Prevalence of hyperactive impulsive subtype	6.7% (7)
Prevalence of inattentive subtype	2.9% (3)
Number of hyperactive impulsive symptoms (adult)	4.0 \pm 2.9
Number of inattentive symptoms (adult)	3.9 \pm 3.3
<i>Childhood ADHD (as per the DIVA 2.0)</i>	
Prevalence of childhood ADHD (all subtypes)	35.2% (37)
Prevalence of mixed-subtype	15.2% (16)
Prevalence of hyperactive impulsive subtype	5.7% (6)
Prevalence of inattentive subtype	14.3% (15)
Number of hyperactive impulsive symptoms (childhood)	2.9 \pm 2.9
Number of inattentive symptoms (childhood)	3.6 \pm 3.3
<i>Food addiction (as per the YFAS 2.0)</i>	
Prevalence of food addiction	14.3% (15)
Number of food addiction symptoms	1.7 \pm 2.4
Significant distress in relation to food	17.1% (18)
<i>Binge eating severity (BES total score)</i>	8.9 \pm 8.9

Note. Descriptive data are presented as mean \pm standard deviation (SD) or percentage (number). ADHD: Attention-Deficit/Hyperactivity Disorder; BES: Binge Eating Scale; BMI: Body Mass Index; DIVA: Diagnostisch Interview Voor ADHD bij volwassenen; YFAS 2.0: Yale Food Addiction Scale version 2.0 (based on DSM-5 criteria for addictive disorders).

Table 2. Factors associated with adult ADHD diagnosis in the whole sample (logistic regression analyses).

	Obese patients with adult ADHD (n=28)	Obese patients without adult ADHD (n=77)	OR [95% CI]	p-value
<i>Socio-demographic and weight-related characteristics</i>				
Age (years)	46.6 ± 11.7	46.7 ± 10.3	1.00 [.96-1.04]	.94
Gender (female)	85.7% (24)	87% (67)	1.12 [.32-3.90]	.86
Current Body Mass Index (BMI; kg/m ²)	46.3 ± 8.2	47.1 ± 7.7	.99 [.93-1.05]	.63
Previous maximal BMI (kg/m ²)	49.5 ± 8.2	50.3 ± 7.8	.99 [.94-1.04]	.61
Previous bariatric surgery (yes)	78.6% (22)	81.8% (63)	1.23 [.42-3.59]	.71
Age of onset of obesity (years)	14.9 ± 9.5	18.9 ± 10.5	.96 [.92-1.01]	.09
<i>Presence of sleep-apnea syndrome</i>	32.1% (9)	27.3% (21)	1.26 [.49-3.23]	.63
<i>Adult ADHD (as per the DIVA 2.0)</i>				
Prevalence	100% (28)	0% (0)	-	-
Number of hyperactive impulsive symptoms (adult)	6.9 ± 1.5	2.9 ± 2.5	2.07 [1.55-2.75]	<.001
Number of inattentive symptoms (adult)	6.6 ± 2.5	3.0 ± 3.0	1.49 [1.25-1.77]	<.001
<i>Childhood ADHD (as per the DIVA 2.0)</i>				
Prevalence	100% (28)	11.7% (9)	-	-
Number of hyperactive impulsive symptoms (childhood)	6.3 ± 2.3	1.7 ± 2.0	2.00 [1.56-2.58]	<.001
Number of inattentive symptoms (childhood)	6.8 ± 2.6	2.5 ± 2.7	1.66 [1.36-2.04]	<.001
<i>Food addiction (as per the YFAS 2.0)</i>				
Prevalence of food addiction	28.6% (8)	9.1% (7)	4.00 [1.29-12.40]	.016
Number of food addiction symptoms	3.1 ± 3.1	1.2 ± 1.8	1.37 [1.14-1.65]	<.001
Significant distress in relation to food	32.1% (9)	11.7% (9)	3.58 [1.25-10.30]	.018
<i>Binge eating severity (BES total score)</i>	13.4 ± 11.1	7.2 ± 7.3	1.08 [1.03-1.14]	.004

Note. Descriptive data are presented as mean ± standard deviation (SD) or percentage (number). We compared patients with versus without adult ADHD using univariate logistic regression analyses: results are presented as Odds-Ratio, its Confidence Interval at 95% [95% CI] and its associated p-value (two-tailed). ADHD: Attention-Deficit/Hyperactivity Disorder; BES: Binge Eating Scale; BMI: Body Mass Index; DIVA: Diagnostisch Interview Voor ADHD bij volwassenen; YFAS 2.0: Yale Food Addiction Scale version 2.0 (based on DSM-5 criteria for addictive disorders).

Table 3. Factors associated with childhood ADHD diagnosis in the study sample (logistic regression analyses).

	Obese patients with childhood ADHD (n=37)	Obese patients without childhood ADHD (n=68)	OR [95% CI]	p-value
<i>Socio-demographic and weight-related characteristics</i>				
Age (years)	46.4 ± 11.6	46.6 ± 10.2	1.00 [.96-1.04]	.92
Gender (female)	83.8% (31)	88.2% (60)	1.45 [.46-4.56]	.52
Current Body Mass Index (BMI; kg/m ²)	46.6 ± 8.2	47.1 ± 7.6	.99 [.94-1.05]	.75
Previous maximal BMI (kg/m ²)	50.1 ± 8.3	50.1 ± 7.7	1.00 [.95-1.05]	.98
Previous bariatric surgery (yes)	81.1% (30)	80.9% (55)	1.01 [.37-2.81]	.98
Age of onset of obesity (years)	15.9 ± 9.7	18.9 ± 10.6	.97 [.93-1.01]	.17
<i>Presence of sleep-apnea syndrome</i>	32.4% (12)	26.5% (18)	1.33 [.56-3.20]	.52
<i>Adult ADHD (as per the DIVA 2.0)</i>				
Prevalence	75.7% (28)	0% (0)	-	-
Number of hyperactive impulsive symptoms (adult)	5.9 ± 2.4	2.9 ± 2.6	1.54 [1.28-1.85]	<.001
Number of inattentive symptoms (adult)	5.8 ± 2.8	2.9 ± 3.1	1.35 [1.17-1.56]	<.001
<i>Childhood ADHD (as per the DIVA 2.0)</i>				
Prevalence	100% (37)	0% (0)	-	-
Number of hyperactive impulsive symptoms (childhood)	5.6 ± 2.8	1.4 ± 1.7	1.95 [1.54-2.46]	<.001
Number of inattentive symptoms (childhood)	6.7 ± 2.5	1.9 ± 2.3	1.90 [1.51-2.37]	<.001
<i>Food addiction (as per the YFAS 2.0)</i>				
Prevalence of food addiction	24.3% (9)	8.8% (6)	3.32 [1.08-10.23]	.034
Number of food addiction symptoms	2.7 ± 2.9	1.2 ± 1.9	1.29 [1.08-1.55]	.006
Significant distress in relation to food	17.1% (10)	11.8% (8)	2.78 [.99-10.30]	.053
<i>Binge eating severity (BES total score)</i>	11.6 ± 10.5	7.4 ± 7.5	1.06 [1.01-1.11]	.025

Note. Descriptive data are presented as mean ± standard deviation (SD) or percentage (number). We compared patients with versus without adult ADHD using univariate logistic regression analyses: results are presented as Odds-Ratio, its Confidence Interval at 95% [95% CI] and its associated p-value (two-tailed). ADHD: Attention-Deficit/Hyperactivity Disorder; BES: Binge Eating Scale; BMI: Body Mass Index; DIVA: Diagnostisch Interview Voor ADHD bij volwassenen; YFAS 2.0: Yale Food Addiction Scale version 2.0 (based on DSM-5 criteria for addictive disorders).

SUPPLEMENTAL TABLE 1. Correlations between food addiction, binge eating, mean number of adult and childhood ADHD symptoms, current BMI and age in the whole sample (n=105).

	1	2	3	4	5	6	7	8
1. Food addiction score (YFAS 2.0)	-							
2. Binge eating score (BES)	.64 ^{***}	-						
3. Adult ADHD inattentive symptoms (DIVA 2.0)	.36 ^{***}	.40 ^{***}	-					
4. Adult ADHD hyperactivity/impulsivity symptoms (DIVA 2.0)	.33 ^{***}	.32 ^{***}	.70 ^{***}	-				
5. Childhood ADHD inattentive symptoms (DIVA 2.0)	.25 ^{**}	.22 ^{**}	.67 ^{***}	.58 ^{***}	-			
6. Childhood ADHD hyperactivity/impulsivity symptoms (DIVA 2.0)	.29 ^{**}	.26 ^{**}	.58 ^{***}	.68 ^{***}	.66 ^{***}	-		
7. Age	.15	.11	.20 [*]	.04	.01	-.07	-	
8. Current BMI	-.02	-.08	.05	-.01	.15	.07	.12	-

Note. We used Spearman's correlation tests because some variables did not meet normality assumptions. BES: Binge Eating Scale; YFAS 2.0: Yale Food Addiction Scale 2.0; DIVA 2.0: Diagnostische Interview Voor ADHD bij volwassenes.

* p<.05; ** p<.01; *** p<.001

FIGURES

Figure 1. Study flow chart

