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Adult attention-deficit/hyperactivity disorder among alcohol use disorder inpatients is associated with food addiction and binge eating, but not BMI

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ABSTRACT

Introduction: Attention-deficit/hyperactivity disorder (ADHD) is associated with binge eating (BE), food addiction (FA), and obesity/higher BMI in individuals without alcohol use disorder (AUD). ADHD is highly prevalent in patients with AUD, but it is unknown whether the presence of comorbid AUD might change the nature of the association between ADHD, BE, FA and BMI (food and alcohol may either compete for the same brain neurocircuitry or share vulnerability risk factors). Here, we filled this gap by testing the association between ADHD and FA/BE in adult patients hospitalized for AUD, with the strength of simultaneously assessing childhood and adult ADHD. We also investigated the association between ADHD and BMI, and the other factors associated with BMI (FA/BE, AUD severity).

Methods: We included 149 AUD inpatients between November 2018 and April 2019. We assessed both childhood and adulthood ADHD (Wender Utah Render Scale and Adult ADHD Self-Report Scale), FA (modified Yale Food Addiction Scale 2.0), BE (Binge Eating Scale), and BMI and AUD (clinical assessment).

Results: In multivariable analyses adjusted for age, adult ADHD was associated with higher BE scores ($p = .048$), but not significant BE (9% vs. 7%; $p = .70$). ADHD was also associated with FA diagnosis and the number or FA symptoms, with larger effect size for adult (ORs: 9.45[95%CI: 2.82–31.74] and 1.38[1.13–1.69], respectively) than childhood ADHD (ORs: 4.45[1.37–14.46] and 1.40[1.13–1.75], respectively). In multivariable analysis, BMI was associated with both significant BE ($p < .001$) and FA diagnosis ($p = .014$), but not adult ADHD nor AUD severity.

Conclusion: In patients hospitalized for AUD, self-reported adult ADHD was associated with FA and BE, but not BMI. Our results set the groundwork for longitudinal research on the link between ADHD, FA, BE, and BMI in AUD inpatients.

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1. Introduction

Adult attention-Deficit/Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder characterized by impairing levels of inattention and/or hyperactivity-impulsivity, that usually begins in childhood (before the age of 12) and significantly interferes with social, academic, and/or occupational functioning (American Psychiatric Association, 2013). Current evidence indicates that impairing symptoms of the disorder persist in up to 70% of cases in adulthood (Faraone et al., 2006). Pharmacotherapy (stimulants and non-stimulants) (Cortese et al., 2018) and psychotherapies, including parent training (for children /adolescents) and other behavioral approaches (Kooij et al., 2019), are important components of the multimodal treatment strategy for ADHD. In the general population, ADHD is significantly associated with abnormal eating patterns such as binge eating (BE, i.e., recurrent consumption of unusually large amounts of food during a discrete period of time while experiencing loss of control over food intake) (Cortese et al., 2008), binge eating disorder (i.e., recurrent episodes of binge eating without use of compensatory behaviors), loss of control over food intake (Nazar et al., 2016), and food addiction (FA; i.e., existence of addictive-like eating symptoms towards specific foods high in fat and/or refined carbohydrates, including craving, loss of control over eating, harms related to the behavior, and maintenance of the behavior despite negative consequences) (Gearhardt et al., 2009; Romo et al., 2018). ADHD is also associated with higher prevalence of obesity, with an increase by 70% in obesity prevalence in adults with ADHD compared to the overall population, and by 40% in children with ADHD (Cortese et al., 2016; Nigg et al., 2016). In patients with ADHD, eating disorders or obesity, adult ADHD was significantly associated with loss of control over eating (Hilbert et al., 2018), binge eating behavior/disorder (Nazar et al., 2016), and FA (Brunault et al., 2019). According to these studies, ADHD was thus proposed to be a factor that may, in some patients, explain weight gain and obesity through these abnormal eating patterns (Brunault et al., 2019; Cortese et al., 2016; Nazar et al., 2016). However, we do not know whether these associations might persist in other populations, especially among patients with addictive disorders that are at higher risk for ADHD (Grall-Bronnec et al., 2011; van Emmerik-van Oortmerssen et al., 2012).

Alcohol use disorder (AUD), which is clinically defined by loss of control over alcohol use, craving, at-risk alcohol use, maintenance of use despite negative consequences, social impairment, and pharmacological dependence, is the second most prevalent substance use disorder in the overall population (after tobacco use disorder) (American Psychiatric Association, 2013). Its twelve-month and lifetime prevalence in the overall population are 13.9% and 29.1%, respectively (Grant et al., 2015). AUD is very frequently associated with medical, psychiatric, and addictive comorbidities, including other substance use disorders, affective disorders, anxiety disorders, and personality disorders, with odds ratios (ORs) ranging from 1.20 (95% CI 1.01–1.43) to 6.40 (95% CI 5.76–7.22) (Grant et al., 2015). The prevalence of adult ADHD in patients with AUD is estimated between 19.9% and 28.6% (van Emmerik-van Oortmerssen et al., 2012), and this comorbidity is associated with poorer clinical outcomes such as development of substance use disorders (Klein et al., 2012) and higher SUD severity (Fatséas et al., 2016).

As previously shown, ADHD is in general associated with BE, FA and obesity/elevated BMI, but it is unknown whether the presence of a co-occurrent ADHD and AUD might change the nature of the association between, on the one hand, ADHD, and, on the other hand, BE, FA, and obesity/elevated BMI. One hypothesis may be that the existence of an AUD among patients with ADHD may increase the risk for another addictive disorder (i.e., addictive-like eating behavior) because of shared vulnerabilities (Frascella et al., 2010). Another hypothesis, in line with the evidence that food and alcohol may compete on the same dopamine receptors sites in the brain, may explain the fact that the presence of AUD among ADHD patients may decrease the risk for addictive-like eating behavior (i.e., food consumption may make alcohol ingestion less reinforcing) (Brownell & Gold, 2012). In addition, although we know that prevalence of obesity among patients with an AUD is high (38.3% [95% CI 30.2%–47.0%] according to a meta-analysis) (Vancampfort et al., 2016), it is unclear whether the mechanisms underlying obesity/elevated BMI in patients with ADHD may be different between patients with versus without a co-occurrent AUD. To our knowledge, the links between ADHD and BE or FA, as well as between ADHD and BMI, have not specifically been tested in patients with AUD. Our study strengths include the simultaneous assessment of childhood and adult ADHD, and the focus on a population of patients with AUD diagnosed using a DSM-5 based interview. In line with the studies conducted in patients with obesity, we may assume that the higher BMI observed in patients with a co-morbid AUD and ADHD might be accounted for by abnormal eating behaviors such as BE, binge eating disorder, and/or FA (Brunault et al., 2019; Gearhardt et al., 2009). Given that adult ADHD is highly prevalent in patients with AUD and strongly associated with abnormal eating patterns such as BE or loss of control over food intake (Cortese et al., 2008), we hypothesized here that FA, BE and obesity/BMI would be all associated with adult ADHD in this population.

Another focus of the present study was to study more specifically the association between BE/FA and BMI in patients with an AUD. Although AUD was found to be significantly associated with eating disorders (i.e., anorexia nervosa, bulimia nervosa or binge eating disorder) (Fouladi et al., 2015), especially in populations at higher risk for eating disorders (i.e., women, college students) (Buchholz et al., 2018; Piran & Robinson, 2006), not all eating disorders are associated with weight gain (i.e., only binge eating disorder is associated with overweight/obesity). Given their preferential association with obesity, we may assume that BE and addictive-like eating might be specifically implicated in weight gain. This study could also improve our understanding of the association between both BE and FA and BMI in patients with an AUD.

To fill this gap, the primary objective of this study was to explore the association between adult ADHD and abnormal eating patterns (i.e., FA and BE) in patients hospitalized for an AUD. We hypothesized that patients with an AUD and a co-occurrent adult ADHD would have a significantly higher prevalence rate of FA and of significant BE than AUD patients without adult ADHD. The secondary objectives were: (1) to explore the association between adult ADHD and the number of FA symptoms/BE symptoms in patients with an AUD; (2) to determine whether self-reported *childhood ADHD, as opposed to adulthood ADHD*, was also associated with FA and BE symptoms; (3) to determine the factors associated with BMI in AUD inpatients, including FA, BE, ADHD, and AUD severity. We hypothesized that: (1) self-reported adult ADHD would be significantly associated with both BE and FA, with a larger effect size for FA than BE, because we assumed that the "FA" phenotype may be more specific to ADHD patients given that this association is found in other types of populations (Brunault et al., 2019) (i.e., BE symptoms rely on a certain amount of food eaten, while FA symptoms rely on loss of control and maintenance of a behavior despite adverse consequences) and given that food may play a role as an exogenous activation of reward systems especially in ADHD patients (Blum et al., 2014; Weissenberger et al., 2017); (2) Self-reported ADHD status would be significantly associated with both FA and BE symptom scores, 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) FA, BE and ADHD would be significantly associated with higher BMI.

2. Method

2.1. Participants and procedure

This cross-sectional study included all consecutively hospitalized patients with AUD for an alcohol abstinence 3-month rehabilitation program within the addiction rehabilitation center named "Centre

Hospitalier Louis Sevestre" (La Membrolle-sur-Choisille, France) between November 2018 and April 2019. Patients were cross-sectionally assessed after appropriate treatment of the withdrawal symptoms (second week of their stay). This center, which is one of the biggest French addiction rehabilitation centers (140 patients can be hospitalized at the same time), mainly receives AUD patients, but it is also opened for patients with all types of addictive disorders (i.e., substance-related disorders and behavioral addictions). Exclusion criteria were: hospitalization only for a short detoxification period (seven to fifteen days), hospitalization for an illicit drug use disorder rather than AUD, and patients refusing to participate to the study.

2.2. Measures

2.2.1. Socio-demographics and medical characteristics

We collected data on demographics (age, sex, marital status, employment status) based on medical records. We assessed current body mass index (BMI) using height and weight that were systematically measured by a clinician using a measuring tape and a weighting scale upon admission. Assessment of AUD, as *per* DSM-5 criteria, was systematically performed by one of the five addiction rehabilitation center practitioners, all being expert in addiction medicine (American Psychiatric Association, 2013). We assessed each of the eleven DSM-5 criteria that occurred during the previous 12-month period, as well as significant distress, enabling the calculation of a number of AUD criteria ranging from 0 to 11. AUD severity was rated as mild, moderate, or severe depending on the number of positive criteria (cut-offs respectively at 2, 4 and 6).

2.2.2. Self-reported attention-deficit/hyperactivity disorder

Self-reported adult ADHD was assessed using both the 6-item Adult ADHD Self-report Scale (ASRS) version 1.1 (Kessler et al., 2005) and the 25-item Wender Utah Rating scale (WURS) (Ward et al., 1993; French version:; Caci et al., 2010). The 6-item ASRS, which consists of six out of these 18 questions that were selected based on stepwise logistic regression to optimize concordance with the clinical classification, is the short version of the 18-items ASRS, based on DSM-IV-TR criteria. The 6-item ASRS includes items with Likert-type scale ranging from 1 (never) to 5 (very often) with a specific cut-off for each item. When participants met at least four positive items (on a total of 6), the ASRS questionnaire was considered significant (Kessler et al., 2005). According to Brevik et al., 2020, the 6-item ASRS performs equally well as the full ASRS. Self-reported childhood ADHD was assessed with the WURS-25, which is a 25-item questionnaire with a Likert-type scale ranging from 0 (not at all) to 4 (a lot) for each item (total score ranges from 0 to 100). A score of 46 or more indicates probable childhood ADHD (Ward et al., 1993). In line with previous studies (Brevik et al., 2020; Daigre et al., 2015), we used the combination of the WURS and the ASRS to assess adult ADHD. The combination of both the ASRS and the WURS has indeed high diagnosis accuracy to discriminate between patients with versus without ADHD, with an Area Under Curve of 0.964 (95% CI: 0.955–0.973) (Brevik et al., 2020).

Participants were considered as having "self-reported adult ADHD" when they had a significant score on both the ASRS (cutoff$\geq$4) and the WURS (cutoff$\geq$46). Participants were considered as having "self-reported childhood ADHD" when the WURS was significant ($\geq$46) no matter the ASRS score. Participants with an ASRS score <4 and a WURS score <46 were considered as having "no childhood or adult ADHD". For better clarity, we used the term "self-reported ADHD" for "probable ADHD as assessed by a self-administered questionnaire" throughout the manuscript.

2.2.3. Food addiction

The concept of food addiction (FA) relied on Gearhardt et al.'s proposal of adapting DSM-IV-TR substance dependence criteria (Gearhardt et al., 2009), and then, DSM-5 criteria for Substance-Related and Addictive Disorders (Gearhardt et al., 2016) to food. We assessed FA using the French version of the modified Yale Food Addiction Scale 2.0 (mYFAS 2.0) (original version: Schulte & Gearhardt, 2017), which is the short version of the Yale Food Addiction Scale 2.0 (YFAS 2.0).

The mYFAS 2.0 and the YFAS 2.0 are based on DSM-5 criteria (YFAS 2.0; original version: Gearhardt et al., 2016; French version: Brunault et al., 2017). The mYFAS 2.0 is a 15-item self-administered questionnaire that identified people exhibiting addictive-like eating symptoms over the previous 12 months, relying on the DSM-5 criteria for Substance-Related and Addictive Disorders extrapolated to food (Schulte & Gearhardt, 2017). The mYFAS 2.0 enabled the assessment of a number of FA symptoms that occurred over the past 12 months (ranging from zero to eleven) and the "diagnosis" of FA when the participant report at least two FA symptoms during the previous 12 months associated with a self-reported significant clinical impairment and/or distress. FA was then rated as mild, moderate or severe depending on the number of FA symptoms associated (cut-off: 2, 4, and 6, respectively) (American Psychiatric Association, 2013; Gearhardt et al., 2016).

The mYFAS 2.0 as well as the YFAS 2.0 demonstrated good construct validity in both obese and non-obese individuals (Gearhardt et al., 2016; Meule et al., 2017; Schulte & Gearhardt, 2017) including excellent internal consistency (α=.90), as well as good convergent and incremental validity (Gearhardt et al., 2016) with BE, emotional eating, and weight-related variables (i.e., current BMI, past maximal BMI, history of weight cycling). In the present study, we considered self-reported FA 'diagnosis' was met when the patient reported at least two FA symptoms plus clinically significant impairment or distress, and we assessed FA severity using the number of FA symptoms (symptom count).

2.2.4. Binge eating

BE symptoms were assessed using the French version of the Binge Eating Scale (BES), a 16-item questionnaire designed to assess severity of BE using behavioral, affective, and cognitive symptoms (original version: Gormally et al., 1982; French version: Brunault et al., 2016). It has been validated in French and is a reliable tool for assessing binge eating disorder and BE symptoms (Brunault et al., 2016). In this study, the BES total score was used to estimate BE behavior severity. Significant BE behavior was met when the BES score was 18 or higher (Gormally et al., 1982).

2.3. Statistical analyses

Descriptive statistics included percentages for ordinal variables, and means and standard deviations for continuous variables. FA and BE were considered as different entities because these two variables were not associated ($p = .08$). We used Spearman's correlation tests to determine the association between FA symptoms and BE scores. All results were adjusted for age because patients with self-reported ADHD were younger than other patients ($p < .001$).

The analyses of the primary and secondary hypotheses were prespecified. To address the primary objective, multivariate logistic regression analyses were used after adjustment for age (independent variable) to assess the link between self-reported adult ADHD (having both a significant score at both the ASRS and the WURS with respective cut-offs at 4 and 46; dependent variable) and self-reported FA diagnosis (mYFAS 2.0, independent variable), as well as between self-reported adult ADHD and significant BE (BES, independent variable). To account for a possible gender bias, we computed the same analyses in the male-only sample. We used the same procedure to determine what were the factors associated with self-reported *childhood* ADHD. In relation to the secondary objectives, multivariable linear regression analyses were used to determine the factors associated with BMI at the beginning of the hospitalization (dependent variable): age (independent variable), sex (independent variable), AUD severity (DSM-5 symptom count; independent variable), FA diagnosis (mYFAS 2.0; independent variable), and

significant BE behavior (BES≥18; independent variable). We also tested whether there was an interaction between self-reported ADHD and FA in predicting BMI, and between self-reported ADHD and BE in predicting BMI. Results were presented as OR, 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 <.05 were considered statistically significant.

2.4. Ethics

This protocol was approved by the Institutional Review Board of the University Hospital of Tours, France (IRB number: 2018-084), prior to the study. 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.

3. Results

3.1. Study flow diagram

The study flow chart is presented in Fig. 1. Out of the 221 initial patients who were admitted for a 3-month abstinence program during the inclusion period, 20 patients were treated for a substance-use disorder but did not meet DSM-5 criteria for an AUD, 25 patients refused to participate in this study, and 27 patients returned questionnaires with missing data. One hundred forty-nine patients returned fully useable questionnaires (no missing data for the self-administered questionnaires).

3.2. Descriptive statistics and correlations between the variables

Table 1 presents the descriptive data of the study sample. Prevalence rate for self-reported adult ADHD, self-reported FA and significant BE behavior were 22%, 10%, and 7%, respectively. Almost 80% (n = 118) of the sample met severe AUD diagnosis; mean number of DSM-5 AUD symptoms was 8 ± 3. The mean age in the whole sample was 43.5 ± 10.6 years (range: 18–68). A significant correlation was found between FA score and BE score (ρ_s=.31; $p < .01$), but 73% of the patients with significant BE had no FA, and 80% of the patients with FA had no significant BE.

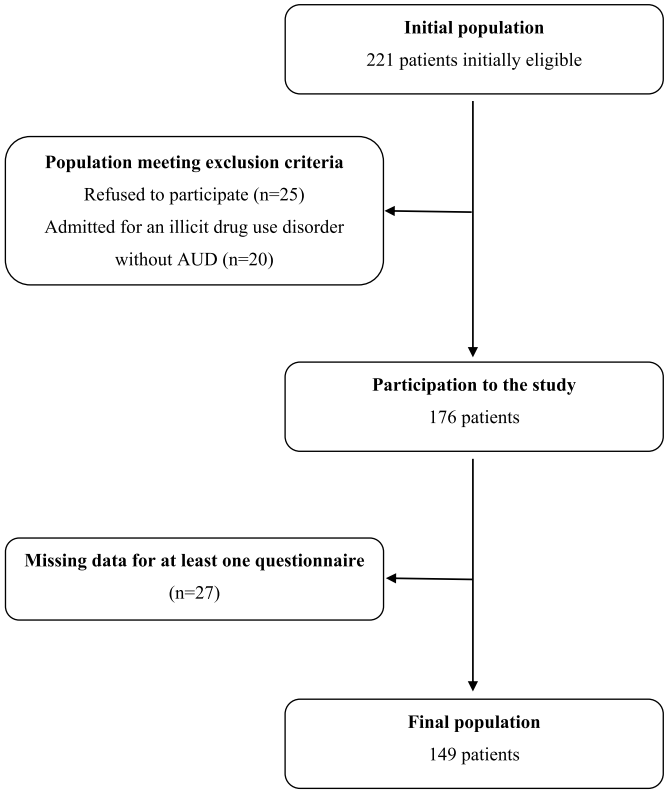

Fig. 1. Study flow chart.

3.3. Factors associated with self-reported adult ADHD

Table 2 presents the factors associated with self-reported adult ADHD after adjusting for age (because it was associated with adult ADHD in univariate analysis; $p = .035$). Patients with self-reported adult ADHD had significantly higher rates of FA than patients without adult ADHD (27% vs. 5%), but not higher prevalence for significant BE (7% vs. 9%). BMI was not higher in patients with adult ADHD. FA symptom count and BE scores were both significantly associated with adult ADHD status. Self-reported adult ADHD was not related to sex, marital status, employment status, nor number of AUD symptoms. We computed the same analyses in the male-only sample and we obtained results close to the ones observed in the whole sample,[1] except for the number of AUD symptoms that was associated with having a self-reported ADHD in the male-only sample but not in the whole sample.

Table 1
Descriptive data of the study sample (n = 149).

	Mean ± SD Percentage (n)
Socio-demographic and weight-related characteristics	
Age (years)	43.5 ± 10.6
Sex (male)	83% (124)
Single (yes)	28% (41)
Employment (yes)	30% (44)
Current Body Mass Index (BMI; kg/m^2)	24.4 ± 3.8
Self-reported ADHD (as per the ASRS and the WURS)	
WURS total score	37 ± 20
Number of positive ASRS items	4 ± 2
Prevalence of self-reported childhood ADHD (WURS≥ 46)	30% (44)
Significant ASRS score (ASRS≥4)	54% (81)
Prevalence of self-reported adult ADHD (ASRS; WURS)	22% (33)
Food addiction (as per the mYFAS 2.0)	
Prevalence of food addiction	10% (15)
Mild food addiction	4% (7)
Moderate food addiction	2% (3)
Severe food addiction	3% (5)
Number of food addiction symptoms	1 ± 2
Binge eating (as per the BES)	
Binge eating severity (BES total score)	6 ± 6
Significant binge eating behavior (BES≥18)	7% (11)
Alcohol use disorder (clinical interview)	
Number of DSM-5 criteria	8 ± 3
Mild alcohol use disorder	10% (15)
Moderate alcohol use disorder	11% (16)
Severe alcohol use disorder	79% (118)

Note. Descriptive data are presented as mean ± standard deviation (SD) or percentage (number). ADHD: Attention-Deficit/Hyperactivity Disorder; BES: Binge Eating Scale; BMI: Body Mass Index; ASRS: Adult ADHD self-report scale, 6 items; WURS: Wender Utah Rating Scale, 25 items; mYFAS 2.0: modified version of the Yale Food Addiction Scale version 2.0 (based on DSM-5 criteria for addictive disorders).

[1] In the male-only sample, having a self-reported adult ADHD was not associated with marital status (p = .51), current employment (p = .43), current BMI (p = .90), or the BES total score (p = .074). Having a self-reported adult ADHD was however associated with a higher number of FA symptoms (p < .003) and a higher number of AUD symptoms (p = .012).

Table 2
Factors associated with self-reported *adult* ADHD in the whole AUD inpatient sample (multivariate logistic regression analyses adjusted for age).

	Patients with self-reported adult ADHD ($n = 33$)	Patients without self-reported adult ADHD ($n = 116$)	OR [95% CI]	*p-value*
Socio-demographic and weight-related characteristics				
Sex (male)	91% (30)	81% (94)	.40 [.11–1.47]	.17
Single (yes)	18% (6)	30% (35)	.54 [.20–1.44]	.22
Current employment (yes)	18% (6)	33% (38)	.53 [.20–1.42]	.21
Current Body Mass Index (BMI; kg/m^2)	24.1 ± 4.0	24.5 ± 3.7	1.01 [.91–1.13]	.81
Self-reported adult ADHD symptoms (as per the ASRS)				
Significant ASRS score (ASRS≥4)	100% (33)	41% (48)	-	–
Number of adult ADHD symptoms (ASRS positive items)	5 ± 1	3 ± 2	2.08 [1.46–2.95]	<.001***
Self-reported childhood ADHD (as per the WURS)				
Significant score (WURS ≥46)	100% (33)	10% (11)	–	–
Childhood symptom severity (WURS total score)	62 ± 11	29 ± 16	1.17 [1.11–1.23]	<.001***
Food addiction (as per the mYFAS 2.0)				
Prevalence of food addiction	27% (9)	5% (6)	9.45 [2.82–31.74]	<.001***
Number of food addiction symptoms	2 ± 2	1 ± 1	1.38 [1.13–1.69]	.002**
Significant distress in relation to food	30% (10)	8% (9)	3.58 [1.65–13.46]	.004**
Binge eating (as per the BES)				
Binge eating severity (BES total score)	8 ± 6	5 ± 6	1.05 [1.00–1.11]	.048*
Significant binge eating behavior	9% (3)	7% (8)	1.32 [.33–5.37]	.70
Alcohol use disorder (clinical interview)				
Number of DSM-5 criteria	9 ± 3	7 ± 3	1.14 [.98–1.33]	.08
Mild AUD	6% (2)	11% (13)	3.99 [.33–48.53]	.28
Moderate AUD	6% (2)	12% (14)	1.00 [.99–1.01]	.99
Severe AUD	88% (29)	77% (89)	.54 [.11–2.61]	.44

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 adjusted for age: results are presented as Odds-Ratio, its Confidence Interval at 95% [95% CI] and its associated *p*-value (two-tailed). AUD: alcohol use disorder; ADHD: Attention-Deficit/Hyperactivity Disorder; BES: Binge Eating Scale; BMI: Body Mass Index; ASRS: 6-item adult ADHD self-report scale; WURS: 25-item Wender Utah Rating Scale; YFAS 2.0: Yale Food Addiction Scale version 2.0 (based on DSM-5 criteria for addictive disorders). *p < .05; **p < .01; ***p < .001.

3.4. Factors associated with self-reported childhood ADHD

As presented in Table 3, after adjusting for age (because it was associated with childhood ADHD in univariate analysis; $p < .001$), patients with self-reported childhood ADHD had higher rates of FA (21% vs. 6%) and a higher number of FA symptoms than patients without self-reported childhood ADHD, but they did not report significantly higher BE scores nor higher prevalence of significant BE behavior. Self-reported childhood ADHD was not related to sex, marital status, nor employment status.

3.5. Factors associated with elevated BMI

Table 4 shows the results of a multivariable analysis investigating the association between, on one hand, BMI (dependent variable) and, on the other hand, age, FA diagnosis, significant BE behavior, adult ADHD and AUD severity (independent variables). BMI was significantly associated with both FA, significant BE behavior and older age, but not self-reported adult ADHD nor AUD severity.

The strength of the association between BMI and significant BE behavior did not differ significantly between patients with and without adult ADHD (95% CI [-1.40; 1.05]; $p = .78$). This model accounted for 5.6% of the variance in BMI (adjusted R-squared=.179; $F(7; 141) = 5.60$; $p < .001$). Likewise, no difference was found in patients with and without adult ADHD when testing the association between BMI and FA (95% CI [-0.63-1.33]; $p = .48$), and this model accounted for 6% of the variance in BMI (adjusted R-squared =.181; $F(7; 141) = 5.678$; $p < .001$).

4. Discussion

This study demonstrates that among patients with severe AUD, self-reported adult ADHD was significantly associated with a higher prevalence of FA diagnosis, higher FA scores, and higher BE severity. In the whole sample, BMI was not associated with adult ADHD, but it was independently associated with both significant BE behavior and FA diagnosis. Both childhood and adult self-reported ADHD were associated with FA, with a larger effect size for self-reported *adult* ADHD than *childhood* ADHD. Furthermore, self-reported ADHD was not related to AUD symptoms.

The association between ADHD and AUD is well described in the literature (Bernardi et al., 2012; Ercan et al., 2003), but, to the best of our knowledge, no study has investigated the prevalence of FA in patients with adult self-reported ADHD amongst an AUD clinical sample. Studies on the association between ADHD and FA have been mainly been conducted in patients with obesity or have assessed the prevalence of obesity amongst patients with ADHD (Brunault et al., 2019; Cortese & Tessari, 2017), highlighting the role of underlying mechanisms such as abnormal eating patterns such as loss of control over eating (Hilbert et al., 2018) and BE behavior (Brewerton & Duncan, 2016; Nazar et al., 2016). We found here that the association between adult ADHD and FA/BE was also demonstrated outside the obesity field. As described by Weissenber et al., for patients with ADHD, food may help in regulating emotion dysregulation, by soothing an unpleasant state of mind or by self-medicating some symptoms such as anxiety and/or sadness (Weissenberger et al., 2017). On top of this psychopathological hypothesis, Blum et al. bring a neurobiological hypothesis to explain the increased risk for addictive disorders in persons with ADHD (Blum et al., 2014). According to these authors, ADHD may be a behavioral subtype of a reward deficiency syndrome, and patients with ADHD may cope with this reward deficiency syndrome using food and alcohol (Blum et al., 2008). Interestingly, AUD patients with a co-occurrent adult ADHD had a FA prevalence that was high and close to the ones found in bariatric surgery candidates (27% vs. 28.6%) (Brunault et al., 2019). Given its legal status and accessibility during childhood, we may hypothesize that food may in fact be the first substance/behavior used by patients with

Table 3
Factors associated with self-reported *childhood* ADHD in the whole AUD inpatient sample (univariate logistic regression analyses adjusted for age).

	Patients with self-reported childhood ADHD ($n = 44$)	Patients without self-reported childhood ADHD ($n = 105$)	OR [95% CI]	*p-value*
Socio-demographic and weight-related characteristics				
Sex (male)	89% (39)	81% (85)	.50 [.17–1.48]	.21
Single (yes)	18% (8)	31% (33)	1.94 [.79–4.73]	.15
Current employment (yes)	16% (7)	35% (37)	.71 [.93–5.99]	.07
Current Body Mass Index (BMI; kg/m^2)	24.1 ± 3.7	24.5 ± 3.8	1.00 [.90–1.10]	.93
Self-reported Adult ADHD symptoms				
Significant ASRS score (ASRS ≥4)	75% (33)	46% (48)	-	–
Number of adult ADHD symptoms (ASRS positive items)	4 ± 1	3 ± 2	.80 [.62–1.03]	.08
Self-reported childhood ADHD (WURS)				
Prevalence (WURS ≥46)	100% (44)	0% (0)	–	–
Childhood symptom severity (WURS total score)	62 ± 10	26 ± 12	–	–
Food addiction (as per the YFAS 2.0)				
Prevalence of food addiction	21% (9)	6% (6)	4.45 [1.37–14.46]	.01*
Number of food addiction symptoms	2 ± 3	1 ± 2	1.40 [1.13–1.75]	.003**
Significant distress in relation to food	33% (11)	8% (8)	2.30 [.81–6.53]	.12
Binge eating (as per the BES)				
Binge eating severity (BES total score)	7 ± 7	5 ± 6	1.04 [.98–1.09]	.23
Significant binge eating behavior (BES ≥18)	9% (4)	7% (7)	.73 [.20–2.73]	.64
Alcohol use disorder (clinical interview)				
Number of DSM-5 criteria	8 ± 3	7 ± 3	1.11 [.97–1.27]	.12
Mild AUD	9% (4)	10% (11)	.47 [.04–5.87]	.56
Moderate AUD	2% (1)	14% (15)	2.92 [.58–14.62]	.19
Severe AUD	89% (39)	75% (79)	6.14 [.74–51.38]	.09

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 adjusted for age: results are presented as Odds-Ratio, its Confidence Interval at 95% [95% CI] and its associated *p*-value (two-tailed). AUD: Alcohol use disorder; ADHD: Attention-Deficit/Hyperactivity Disorder; BES: Binge Eating Scale; BMI: Body Mass Index; ASRS: 6-item adult ADHD self-report scale; WURS: 25-item Wender Utah Rating Scale; YFAS 2.0: Yale Food Addiction Scale version 2.0 (based on DSM-5 criteria for addictive disorders). *p < .05; **p < .01; ***p < .001.

childhood ADHD. The severity of AUD may also explain this high FA prevalence, because alcohol intake is associated with increased food intake through enhancing short-term rewarding effects of food (Yeomans, 2010).

Adding to existing data showing over-representation of ADHD and high prevalence for BE/FA in patients with AUD (van Emmerik-van Oortmerssen et al., 2012), this study demonstrated that FA and BE severity were more strongly associated with self-reported *adult* ADHD than self-reported *childhood* ADHD. This is in line with previous studies indicating that remitted adult ADHD (when compared to non-remitted adult ADHD) was associated with a lower risk of FA in obese populations (Brunault et al., 2019). On a similar note, remitted ADHD in adulthood was also associated with a shorter duration of substance use disorder and higher substance use disorder remission rates (Wilens et al., 1998). This result is consistent with the idea that BE/FA and ADHD may be causally related, as improvement in ADHD symptoms is associated with lower FA/BE.

Another interesting result from our study was the lack of association between BMI and ADHD. BMI was however independently associated with BE and FA, supporting the hypothesis that these two measures may in fact assess different and complementary measures of abnormal eating patterns (Gearhardt, Corbin, & Brownell, 2016a,b). BE, as assessed *per* the Binge Eating Scale, refers to a quantity of food intake, with items relying also on emotional distress related to weight, whereas FA, as assessed *per* the mYFAS 2.0, focuses mainly on loss of control over food intake and on the harms associated with this loss of control. These two complementary measures of abnormal eating patterns may thus be differentially associated with outcomes (i.e., BE was more strongly associated with BMI, while FA was more strongly associated with adult and childhood ADHD).

These results should be seen in light of study strengths and limitations. Study strengths include the clinical assessment of DSM-5 AUD criteria by addiction medicine clinicians, the assessment of both childhood and adult ADHD, the assessment of both BE and FA (that are two different and complementary measures of eating behavior), and the use of the mYFAS 2.0 that is based on the extrapolation to food of the DSM-5 criteria for addictive disorders. This study also has some limitations. We used self-administered questionnaires to assess ADHD instead of a clinical interview, and this tends to overestimate the prevalence rate for childhood and adult ADHD (Dakwar et al., 2012). This study was conducted in a relatively small sample, and concentrated mainly male patients with severe AUD, which may in turn explain the high prevalence

Table 4
Factors associated with Body Mass Index in multiple linear regression analysis.

	Body Mass Index (kg/m^2)			
	Total effect			
	B ± *SE*	[95% CI]	*t*	*p*
Age	0.07 ± 0.03	[0.02; 0.13]	2.62	0.01
Sex				
Female				
Male	−0.53 ± 0.77	[-2.07; 0.95]	−0.73	0.47
Alcohol use disorder severity	−0.07 ± 0.10	[-0.26; 0.13]	−0.73	0.49
Adult ADHD				
No adult ADHD (reference)	0.23 ± 0.38	[-0.51; 0.97]	0.62	0.54
Adult ADHD				
Binge eating (BE)				
No Significant BE (reference)				
Significant BE	3.07 ± 0.55	[1.99; 4.15]	5.63	<0.001
Food addiction (FA)				
No FA (reference)				
FA	2.53 ± 1.02	[0.51; 4.55]	2.48	0.014

Note. We used a multiple linear regression analysis to predict body mass index based on age, sex, alcohol use disorder severity (number of DSM-5 criteria), self-reported adult ADHD, food addiction and significant binge eating behavior ($F(6,142) = 6.566$; R^2=.22; $p < .001$). Results are presented as unstandardized beta (B), its standard error (SE), its 95% confidence interval (95% CI), and its associated t- and p-values. ADHD: attention-deficit/hyperactivity disorder; BE: Binge Eating behavior; FA: Food addiction.

rates for ADHD and FA/BE observed here. The over-representation of male in this sample should also be taken into account as eating disorders such as anorexia nervosa, bulimia and, to a lesser extent, binge eating disorder are more prevalent in females. In the male-only sample, we found however close results from those obtained in the whole sample (association with the number of FA symptoms), and this could be due to the fact that in patient with an AUD, FA may be more related to binge eating disorder symptoms rather than to bulimia nervosa or anorexia nervosa symptoms (binge eating disorder being more strongly associated with ADHD than bulimia or anorexia nervosa). Future studies should determine whether we could generalize these results to the overall population of patients with AUD with a less severe disorder. Another important point is the need to account for psychiatric disorders because they could mediate the association between ADHD symptomatology and FA/BE (Kaisari et al., 2018). Finally, the cross-sectional design of this study precludes us from inferring a causal relationship between ADHD, FA/BE, and BMI.

Despite these limitations, this study has interesting clinical and research implications. Firstly, patients with AUD diagnosed with FA or BE behavior should be systematically screened for comorbid childhood and/or adult ADHD. Another practical implication of this study is that patients with AUD and ADHD should be systematically screened for FA in addition to BE, because FA may be a more valid abnormal eating pattern in the AUD population than BE. Professionals in the field of child and adolescent mental health should systematically bear in mind that their patients with childhood ADHD might be at greater risk for FA and BE, and they should systematically follow up on their patients' eating behaviours over a long period of time.

In conclusion, patients with a co-occurrent AUD and adult ADHD were at a higher risk for FA and BE symptoms than patients without ADHD, which replicates previous findings in the specific population of patients with an AUD. However, we failed to demonstrate an association between BMI and adult ADHD, BMI being independently associated with both FA and BE, but not ADHD status. Systematic screening for FA and BE should be conducted among patients with a co-occurrent AUD and adult ADHD. Future longitudinal studies should determine whether ADHD, FA and/or BE could best explain the over-representation of obesity in alcohol abstinent patients (Gottfredson & Sokol, 2019; Jeynes & Gibson, 2017), and test whether an adequate screening for and early management of ADHD could impact FA and BE, and possibly also BMI and post-abstinence weight evolution.

Research data

All the data are available on request.

Declaration of competing interest

PB reports personal fees from Lundbeck, Astra-Zeneca and DNA Pharma, unrelated to the submitted work. NB reports personal fees and non-financial support from Lundbeck, personal fees from Astra-Zeneca and DNA Pharma, unrelated to the submitted work. SC declares reimbursement for travel and accommodation expenses from the Association for Child and Adolescent Central Health (ACAMH), Canadian ADHD Alliance Resource (CADDRA), Healthcare Convention, and British Psychopharmacology Association (BPA) in relation to lectures delivered on ADHD. MGB declares that the Addictology and Psychiatry Department has received funding directly from the University Hospital of Nantes and gambling industry operators (FDJ and PMU); scientific independence in relation to gambling industry operators is assured; there were no constraints on publishing. MGB reports no financial or other relationship relevant to the subject of this article. HEA, SB, AG, JF, EM, FBB: none.

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Appendix A. Supplementary data

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Author contributions

Conceptualization: NB, PB. Methodology: HEA, SB, PB, NB. Formal analysis: HEA, SB, AG, PB. Investigation and Data Curation: HEA, JF, EM. Writing – original draft preparation: HEA, SB, AG, PB. Writing – review and editing: HEA, SB, AG, SC, JF, EM, FBB, MGB, NB, PB. Supervision and project administration: SB, NB, PB. All authors approved the final version of the manuscript for submission.

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Ethical statement

This protocol was approved by the Institutional Review Board of the University Hospital of Tours, France (IRB number: 2018-084), prior to the study. 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.

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