

The association between adult ADHD and food addiction: A mediation analysis

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ABSTRACT

Objectives: The aim of this study was to investigate the association between food addiction severity (FA) and ADHD symptomatology. Furthermore, we investigated whether emotional distress, and the mentalization deficits could mediate this relationship.

Methods: Three hundred eighty-five Italian adults (307 women and 78 men) participated in the study and completed the Italian versions of the Modified Yale Food Addiction Scale version 2.0, the Adult ADHD Self-Report Scale, the Depression Anxiety Stress Scales 21 and the Mentalization Questionnaire.

Results: ADHD symptoms severity and general distress were significantly and independently associated with FA. Emotional distress partly mediated the relationship between FA and ADHD symptoms severity. In addition, mentalization deficits partly mediated this relationship only when evaluating the influence of FA on ADHD symptoms severity.

Limitations: This is a cross-sectional study and causal interpretations of the relationships among the variables are speculative.

Conclusions: It is important to assess emotional distress and mentalization deficits in individuals at risk of ADHD and FA because these conditions could increase the risk for the presence of disordered eating patterns.

1. Introduction

Attention deficit/hyperactivity disorder (ADHD) is traditionally conceptualized as a neurodevelopmental condition characterized by symptoms such as inattention, impulsivity and hyperactivity. According to the Diagnostic and Statistical Manual of Mental Disorders 5th Edition (DSM-5) (American Psychology Association, 2013) ADHD is a persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with the functioning or development of the individual. Six or more symptoms of inattention, hyperactivity and impulsivity should be present for a diagnosis of ADHD in those 16 years old or younger, or five or more symptoms in those 17 years and older. Inattentive or hyperactive-impulsive symptoms should be present in two or more settings before age 12 years (APA, 2013), and symptoms of hyperactivity-impulsivity should be present for at least 6 months and considered inappropriate for the person's developmental level.

ADHD is associated with significant social, academic and

professional impairment (American Psychiatric Association, 2013). In the past, ADHD has been considered to be a childhood disorder, whose symptoms decrease during adolescence and disappear in the adulthood (Ross & Ross, 1976). However, several studies have found that ADHD symptoms can persist into adulthood and impact negatively on the quality of life and social relationships (Aizenstros et al., 2019; De Graaf et al., 2008; Mörtstedt et al., 2016; Schoeman et al., 2017a; van Lieshout et al., 2016).

Epidemiological studies that have investigated the prevalence of ADHD in the adult general population reported rates between 2.5% and 5.0% (Bonvicini et al., 2016; Li et al., 2019; Simon et al., 2009). According to the European Consensus Statement on the diagnosis and treatment of adult ADHD (2019), the prevalence of ADHD in adults, across twenty countries, may average 2.8%, with a range between 1.4% and 3.6% (Kooij et al., 2019).

To investigate the persistence of childhood ADHD into adulthood, Bonvicini et al. (2016) analyzed data from follow-up studies of ADHD

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cases published from 1971 to 2000 and found that the rate of persistence of DSM-IV's ADHD definitional criteria was around 15%, but the rate of cases in partial remission was approximately 65%. Davidson (2008) partially confirmed these data, suggesting a prevalence rate of persistence of ADHD into adulthood varies from 40% to 66%. More recently, in a 6-year follow-up study of a large European cohort of children with ADHD, van Lieshout et al. (2016) reported the persistence of a DSM-5 ADHD diagnosis in 86.5% of the sample, with another 8.4% of the sample having subclinical conditions (van Lieshout et al., 2016).

As for adults with ADHD symptoms, it has been noticed that they often present several difficulties in attending to internal and external stimuli, organizing and completing tasks, and managing emotions, impulses and behaviors (American Psychiatric Association, 2013; Martínez de Velasco et al., 2015). Moreover, mentalization capacity appear to be altered in people with ADHD (Rüfenacht et al., 2019). Mentalization consists in the capacity to appreciate own and others mental state (Fonagy et al., 2018). Mentalization deficits seem to be involved in impulsivity, emotional dysregulation and difficulties in targeting one's own and other's emotions. ADHD symptom severity is positively related to mentalization deficits (Perroud et al., 2017).

Furthermore, up to 90% of adults with ADHD symptoms could have comorbid psychiatric and medical disorders (Kaisari et al., 2017; Schoeman et al., 2017b), the most frequent comorbid conditions being major depression, bipolar disorder, anxiety disorders, substance use disorders, sleep disturbances, and eating disorders (Aizenstros et al., 2019; Fayyad et al., 2017; Seymour et al., 2015).

Although patients with ADHD may not meet fully the criteria for a specific eating disorder, ADHD symptoms and dysregulated eating behaviors have frequently been reported to be associated (Kaisari et al., 2017; Mattos et al., 2004; Nazar et al., 2016; Ptacek et al., 2016; Seymour et al., 2015; Sobanski, 2006; Surman et al., 2006). In the past few years, there has been growing interest in the study of the association between ADHD, binge-eating and obesity (Brunault et al., 2019; Cortese et al., 2007; Quesada et al., 2018; Seymour et al., 2015). Adult ADHD symptoms have been found in obese patients with binge eating tendencies (Brunault et al., 2019; Nazar et al., 2014), and obesity and EDs have been found in adult ADHD patients (Reinblatt, 2015). These studies suggested that adult ADHD could be associated with a higher risk of binge eating (Brunault et al., 2019; Cortese et al., 2007; de Velasco et al., 2015; Fernández-Aranda et al., 2013; Kaisari et al., 2017; Martínez; Nazar et al., 2016; Sala et al., 2018; Steadman & Knouse, 2016) and with obesity (Patte et al., 2020; Seymour et al., 2015; Waring & Lapane, 2008). Unfortunately, the exact mechanisms underlying the association between ADHD and disordered eating patterns like Food Addiction (or binge eating, and loss of control overeating) are still unclear (El Archi et al., 2020). Recently, Barker et al. (2021) investigated whether the phenotypes of impulsivity and BMI, and the polygenic risk score of ADHD and Body Mass Index, could share common associations with whole-brain grey matter and the Monetary Incentive Delay Functional Magnetic Resonance Imaging (fMRI) task. The authors concluded that ADHD and BMI phenotypes, and their respective polygenic risk scores, could be significantly associated with a common neural substrate. In particular, both ADHD and obesity share neural correlates of impairments in inhibitory control and reward processing (Barker et al., 2021). Consistent with these results are those from Nazar et al. (2018) who administered the Continuous Performance Test and the Iowa Gambling Task to a sample of individuals with both ADHD and ED and reported that their scores differed from those of individuals with only ADHD and from controls for impaired decision making.

In the last decade, the concept of food addiction (FA) was proposed by Gearhardt et al. (2009) in order to better understand the relationship between psychopathology obesity/overweight. These authors adapted DSM-IV-TR substance dependence criteria to explain addictive behaviors associated with food, and recently they have updated the criteria to the DSM-5 (Gearhardt et al., 2016). According to Gearhardt et al. (2016), some people could develop addictive-like eating behaviors

related to highly palatable foods (i.e., foods rich in refined carbohydrates, sugar, fat and/or salt). Food craving (FC) could be considered to be the core mechanism of FA (Davis & Carter, 2009; Gearhardt et al., 2016). FC is an intense and uncontrollable need to eat highly palatable foods, accompanied by intrusive thoughts, multi-sensory imagery, strong urges to seek and consume the substance of addiction and poor self-control about high palatable food (Taylor, 2019). Nevertheless, some researchers (Hebebrand et al., 2014) have criticized the application of the substance dependence criteria to define the phenomenological aspects of overeating, and they have proposed the replacement of the use of term "food addiction" with the use of "eating addiction" in order to emphasize the behavioral addiction to eating and not the substance-related phenomenon of dependence on specific categories of food.

Although there is only sparse evidence about the relationships, poor inhibitory control at the neurocognitive level seems to be positively associated with FA, loss of control and excessive food intake (Chen et al., 2018; Hardee et al., 2020). According to this preliminary research, higher impulsivity could influence hyper-feeding behaviors and increase the susceptibility of individuals to hyper-palatable foods (Davis et al., 2011). It is known that the perception of loss of control over food, as well as overeating (Gordon et al., 2018), are the main features of FC. FC may be related to the impossibility of imposing inhibitory control on actions and by the inability to delay behavior (Meda et al., 2009) which are the main features of impulsiveness in ADHD and binge eating disorders (Grant & Chamberlain, 2020). Currently, research investigating the association between adult ADHD and FA is rare (Brunault et al., 2019; Davis et al., 2011; Sala et al., 2018). Brunault et al. (2019) investigated the link between FA, binge eating and adult ADHD in 105 obese patients. The authors assessed, among other variables, childhood/adulthood ADHD, FA, and binge eating symptomatology. This research revealed that obese patients with adult ADHD symptomatology were at significantly higher risk of FA and binge eating than obese patients without ADHD symptomatology. Romo et al. (2018) analyzing the possible co-presence of adult ADHD, EDs and addiction-like behaviors, including FA, in a sample of 1517 students, reporting that adult ADHD symptoms were significantly associated with a risk of FA. Sala et al. (2018) investigated the presence of adult ADHD in patients with a longstanding history of EDs and suggested that ADHD comorbidity is more frequent in people with more abnormal eating and attitudes and bulimic symptoms than other people with EDs. Finally, Davis et al. (2011) reported that obese people with FA had more inattentive, hyperactive and impulsive symptoms than obese controls without FA.

As far as we know no previous studies have investigated the relationship between ADHD symptoms and FA in a more heterogeneous sample from the general adult population, taking into account the role of depression, anxiety, stress and mentalization deficit. Past research indicates that the clinical level of emotional distress could be a risk factor in worsening ADHD symptoms and the severity of FA. In addition, some studies have investigated the association between FA and deficits in mentalization. For example, Innamorati et al. (2017), in 322 Italian adults recruited from the general population, reported that people with a high number of FA symptoms (≥ 3 symptoms) were more likely to report severe mentalization deficits than people with fewer or no symptoms of FA.

Thus, the aims of the present study were: (1) to investigate the association between ADHD symptoms and FA severity in a non-clinical sample of adults from the general population; (2) to analyze whether FA and ADHD symptoms were associated with higher presence of general distress (GD; depression, anxiety, perceived stress) and difficulties in mentalization; and (3) to investigate whether the severity of ADHD symptoms is associated with the severity of FA while controlling for sociodemographic and clinical variables. We also analyzed the possible association between FA and groups of ADHD symptoms (i.e., inattentive and hyperactive-impulsive symptoms), considering that they could affect FA severity differently (Martin et al., 2020).

Finally, we tested two mediation models: (1) a model with adult ADHD symptoms as the predictor and FA as the criterion; and (2) an inverse mediation model with FA as the predictor and adult ADHD symptoms as the criterion. In both models GD and mentalization deficits were included as mediators. Models #1 is based on the observation that ADHD is generally a stable and earlier condition than FA, and that impulsive/hyperactive and inattentive symptoms could influence hyperfeeding behaviors and increase FA symptoms, possibly through the distress they cause and the mentalization deficits accompanying them. Model #2 is based on the idea that sometimes FA could have an onset at earlier age and represent a relevant symptom of adult ADHD (i.e., FA could explain a significant portion of variance of adult ADHD) not completely explained by the presence of other psychopathological conditions (i.e., distress and mentalization deficit).

2. Methods

2.1. Participants

Three hundred eighty-five Italian adults (307 women and 78 men) from the general population voluntarily participated in the study and were assessed between October 2019 and January 2020.

Participants were recruited through an online survey (Google form) and through advertisements posted in community groups and university campuses. The mean age of the participants was 29.68 years ($SD = 10.86$; range: 18–65 years). The sociodemographic characteristics of the sample are reported in Table 1.

The inclusion criteria were 18+ years of age and the presence of the ability to understand and complete the battery of tests. The exclusion criteria were the presence of past or current psychiatric disorders, the use of psychoactive medications in the past 12 months, having undergone psychological or psychotherapeutic treatments in the past 12 months, and an inability to complete the assessment for any reason, including denial of informed consent.

All participants received information about the purpose of the study, provided written informed consent, and completed the assessment anonymously. The participants received no honorarium or any benefit from participating to the study. The research was conducted in

Table 1
Sociodemographic characteristics of the sample (N = 385).

Variables	% [M (S.D.)]
Sex	
Women	79.7%
Men	20.3%
Age, M (SD)	29.68 (10.86)
BMI, M (SD)	23.33 (4.58)
BMI <18.5	10.6%
BMI 18.5 to 24.9	59.7%
BMI 25 to 29.9	20%
BMI >30	9.4%
Marital status	
NotMarried	72.5%
Married/Stable partner	24.2%
Divorced	3.4%
Job Status	
Unemployed or Retired	9.6%
Student	51.4%
Employed	39%
School attainment	
High School and lower	42.6%
Bachelor Degree or higher	57.4%
Tobacco use (last 6 months)	33.5%
Alcohol use (last 6 months)	61.3%
Psychoactive substances use (last 6 months)	22.6%
Substance related problems	1.3%
Weight related problems	43.4%
Currently on diet	26.2%

BMI = body mass index; M (SD) = mean (standard deviation).

compliance with the Helsinki Declaration of 1975, as revised in 2008, and it was approved by the European University's ethics review board.

2.2. Instruments

All participants were administered a checklist assessing socio-demographic and health information. Participants self-reported weight and height (in order to calculate BMI), use of tobacco, alcohol, prescribed drugs and illegal or unprescribed drugs (including legal highs) in the last six months. They were asked whether they were currently on a prescribed or unprescribed diet, and whether they had metabolic diseases. The presence of metabolic diseases in family members was also investigated. Participants were also administered Italian versions of the Modified Yale Food Addiction Scale version 2.0 (mYFAS 2.0) (Imperatori et al., 2019), the Adult ADHD Self-Report Scale (ASRS) (Somma et al., 2019), the Depression Anxiety Stress Scales 21 (DASS-21) (Bottesi et al., 2015), and the Mentalization Questionnaire (MZQ).

2.2.1. Modified Yale Food Addiction Scale version 2.0 (mYFAS 2.0)

The mYFAS 2.0 (Schulte & Gearhardt, 2017) is a short version of the YFAS 2.0 (Gearhardt et al., 2016). It is composed of 13 items, rated on an eight-point Likert-type scale (from 0 = never to 7 = every day) assessing addictive eating behaviors (e.g., "I had such strong urges to eat certain foods that I could not think of anything else"). The mYFAS 2.0 provides two scoring options: a symptom count version (scores ranging from 0 to 11) and a diagnostic version based on the last edition of the DSM criteria (American Psychiatric Association, 2013) for substance related and addictive disorders (i.e., a categorical cut off diagnosis is met when at least two symptoms and clinically significant impairment/distress from eating are present). In the mYFAS 2.0 each symptom is scored based on one item except for the "impairment and distress" criterion, which is assessed by two items. For the "diagnosis" scoring option, a participant can meet the criteria for mild FA (two to three symptoms), moderate FA (four to five symptoms) or severe FA (six or more symptoms). In the present sample Cronbach's α was 0.88.

2.2.2. Adult ADHD self-report scale (ASRS)

The ADHD Self-Report Scale (ASRS) (Kessler et al., 2005) is an 18 item self-report questionnaire based on the DSM-IV-TR (American Psychiatric Association, 2000) criteria of ADHD, adapted for use in adults. The ASRS was developed in conjunction with the revision of the WHO Composite International Diagnostic Interview (CIDI) (Kessler et al., 2005) for the WHO World Mental Health (WMH) Survey Initiative (Demyttenaere et al., 2004). The ASRS was constructed as a screening tool to facilitate identification of probable ADHD in adults. Each ASRS question asks respondents how often a particular symptom of ADHD occurred over the past six months on a five-point response Likert scale (from 0 = never, to 4 = very often). The ASRS includes six questions in Part A (considered most predictive of adult ADHD; ASRS-A) and 12 questions in Part B (specific symptom profile questions; ASRS-B). Total ASRS score can be obtained by the sum of the Part A and Part B scores (ASRS-AB). To assess ADHD risk, we used a cutoff based on the ASRS-AB (Kessler et al., 2005). A cut-off score on the ASRS-AB ≥ 46 was reported to be associated with clinical-level ADHD (Somma et al., 2019). In the present sample the Cronbach's α 's for the Part A scale and the Part B scale were 0.74 and 0.83, respectively. It is also possible to calculate separate scores for severity of inattentive symptoms (including items #1, #2, #3, #4, #7, #8, #9, #10, and #11) and hyperactive-impulsive symptoms (including items #5, #6, #12, #13, #14, #15, #16, #17, and #18). Cronbach's α s were 0.76 and 0.65, respectively for Inattention and Hyperactivity-Impulsivity. To assess the relationship between each ASRS subscale and FA symptoms count score, we performed partial correlations, controlling for the effect of the other group of symptoms.

2.2.3. Depression Anxiety Stress Scales 21(DASS-21)

The Depression Anxiety Stress Scales 21 (DASS-21) (Henry &

Crawford, 2005) is a short form of the Lovibond and Lovibond (1995), p. 42-item self-report measure of depression, anxiety, and stress (DASS). The DASS-21 questionnaire measures the negative emotional states of depression, anxiety and stress by the sum of the 7 corresponding questions (Henry & Crawford, 2005). The questionnaire had 21 items which were rated on a four-point Likert scale (from 0 = did not apply to me at all, to 3 = applied to me very much or most of the time). The GD score is calculated as the sum score of all the 21 items. A higher score is related to a higher level of distress for each subscale, and a higher score on the GD dimension is associated with more severe emotional distress. The DASS-21 does not have a cut-off score, but it is possible to determine five degrees of severity (from “normal” to “extremely severe”). In this study, the Italian version of DASS-21 questionnaire was used (Bottesi et al., 2015), and the Cronbach α 's in the present sample were 0.91, 0.86, 0.88, respectively for the depression, anxiety and stress dimensions.

2.2.4. The Mentalization Questionnaire (MZQ)

The Mentalization Questionnaire (MZQ) (Hausberg et al., 2012) is a 15-item self-report scale measuring mentalization, that is the ability to represent and understand inner mental states in oneself and others. Respondents are asked to rate each item on a five-point Likert scale (from 0 = I disagree, to 4 = I agree). MZQ scores are: (1) Refusing Self-Reflection, (2) Emotional Awareness, (3) Psychic Equivalence Mode, and (4) Regulation of Affect, as well as a total score. An exploratory analysis of the factor structure of the Italian version of the MZQ indicated a one-factor structure with satisfactory internal consistency (McDonald's omega = 0.87) (Innamorati et al., 2017). Lower scores on the MZQ indicate more difficulties in mentalization. Cronbach's α in the present sample was 0.86 for the total score.

2.3. Statistical analysis

All statistical analyses were performed with SPSS 25 (IBM SPSS Statistics for Windows, Version 25.0). Pearson r coefficients were reported as measures of association. Pearson r coefficients <0.10 are considered to be a negligible or null effect, r between 0.10 and 0.30 a small effect, r between 0.30 and 0.50 a medium effect, and $r \geq 0.50$ a large effect (Cohen, 2013). In order to avoid type-1 errors, a Bonferroni correction was used.

Concerning the ASRS, we reported scores for the part A (items #1 to #6), part B (items #6 to #18), and a total score for the whole scale (parts A + B). Furthermore, we also reported scores for dimensions of Hyperactivity-Impulsivity and Inattention. Higher ASRS scores were suggestive of more severe ADHD symptomatology. Considering the low internal consistencies (Cronbach's $\alpha < 0.70$) for most MZQ dimensions and a previous study suggesting unidimensionality for the Italian MZQ, we only reported MZQ total scores as indices of mentalization deficits.

All variables significant at the bivariate analyses after correction for multi-testing were included as independent variables in a regression model, with FA as the criterion. Beta coefficients and their significance were reported as measures of association.

We used moderated-mediation analysis to evaluate the mediating role of GD and mentalization deficit in the relationship between FA and ADHD. Two models were analyzed: (1) a model with adult ADHD symptoms as predictor and FA as criterion; and (2) a model with FA as predictor and adult ADHD symptoms as criterion.

The PROCESS V3.5 macro for SPSS (Hayes, 2013) was used for the analysis (simple mediation model with two independent mediators, model no. 4). We report unstandardized coefficients (b), and their 95% bootstrap confidence intervals (95% CI) (with 5000 bootstrap samples) (Preacher & Hayes, 2008). According to Preacher and Hayes, confidence intervals that do not include zero could provide evidence of a significant indirect effect. Even though mediation models cannot identify causal relationships between variables when data from correlational studies are used (Maxwell et al., 2011), these techniques can be effective in analyzing whether a third variable could mediate the relationship

between two other variables (Salthouse, 2011).

All statistics were considered significant for $p < 0.05$.

3. Results

Forty-five participants had probable clinical-level ADHD (11.7%) based on the 18-item version of the ASRS, and 26.7% met the criteria for FA. Furthermore, 11.9% of the sample reported a clinical-level of anxiety, 12.2% a clinical-level of depression, and 33.8% a clinical-level of stress. Finally, 9.4% of the sample were obese (BMI ≥ 30), 20.0% were overweight (BMI ≥ 25), 59.0% were normal weight (BMI between 18.5 and 24.99), and 10.6% were underweight (BMI < 18.5).

3.1. Relationship between FA, ADHD severity, and other variables

Correlations between variables, after correction for multi-testing, are reported in Table 2. FA symptom severity was significantly and moderately associated with ADHD ($r = 0.44$, $p < 0.001$), Inattention ($r = 0.43$, $p < 0.001$), Hyperactivity-Impulsivity ($r = 0.40$, $p < 0.001$), and GD ($r = 0.37$, $p < 0.001$). FA symptom severity was weakly and significantly associated with mentalization ($r = -0.26$, $p < 0.001$), and BMI ($r = 0.27$, $p < 0.001$). FA symptom severity was not significantly associated with age, and sex. Partial correlation between ASRS Inattention and Hyperactivity-Impulsivity subscale and FA severity (controlling for the effect of the other subscale) resulted in lower effects for both variables (Inattention subscale $\times$ FA severity, controlling for Hyperactivity-Impulsivity: $r = 0.24$, $p < 0.001$; Hyperactivity-Impulsivity subscale $\times$ FA severity, controlling for Inattention: $r = 0.25$, $p < 0.001$).

All variables significantly associated with FA at the bivariate level were included in a regression model with FA as dependent variable. The model explained the data variance well ($R^2 = 0.26$, F -test = 19.43, $p < 0.001$). ADHD symptoms severity ($\beta = 0.29$, $p < 0.001$), GD ($\beta = 0.13$, $p = 0.03$), age ($\beta = -0.12$, $p = 0.01$), and BMI ($\beta = 0.25$, $p < 0.001$) were independently and significantly associated with FA symptoms. Mentalization ($\beta = -0.06$, $p = 0.25$), and sex ($\beta = 0.06$, $p = 0.24$) were not independently associated with FA symptoms.

At the bivariate analyses, ADHD severity was significantly and moderately associated with GD ($r = 0.57$, $p < 0.001$) and mentalization ($r = -0.41$, $p < 0.001$). ADHD severity was not significantly associated with age and BMI. Furthermore, Inattention was significantly and moderately correlated with GD ($r = 0.49$, $p < 0.001$), and mentalization ($r = -0.35$, $p < 0.001$). Hyperactivity-Impulsivity was significantly and moderately correlated with GD ($r = 0.49$, $p < 0.001$), and it was weakly associated with mentalization ($r = -0.38$, $p < 0.001$).

All variables significantly associated with ADHD symptom severity at the bivariate level were included in a regression model with ADHD symptom severity as the dependent variable. The model explained the data variance well ($R^2 = 0.40$, F -test = 35.84, $p < 0.001$). FA severity ($\beta = 0.24$, $p < 0.001$), GD ($\beta = 0.40$, $p < 0.001$), mentalization ($\beta = -0.12$, $p = 0.002$), and sex ($\beta = -0.10$, $p = 0.01$) were independently and significantly associated with ADHD symptom severity. Age ($\beta = 0.02$, $p = 0.74$) and BMI ($\beta = 0.003$, $p = 0.78$) were not significantly associated with ADHD symptom severity.

In the mediation model #1, we included ADHD symptom severity as the independent variable, FA severity as the dependent variable, and MZQ scores and GD as independent mediators (Fig. 1). The model accounted for 18% of the variance in FA severity ($F_{1,367} = 81.73$, $p < 0.001$). Both the direct ($c = 0.25$, $se = 0.04$, $t = 5.68$, $p < 0.001$) and total indirect ($c' = 0.09$, $boot\ se = 0.02$, $boot\ CI = 0.04/0.14$) effects of ADHD on FA were significant. However, ADHD had indirect effect on FA only through GD ($b = 0.06$, $boot\ se = 0.03$, $boot\ CI = 0.008/0.12$), but not through MZQ ($b = 0.03$, $boot\ se = 0.02$, $boot\ CI = -0.01/0.06$).

In the mediation model #2, we included ADHD symptom severity as the dependent variable, FA severity as the independent variable, and GD and mentalization as independent mediators (Fig. 2). The model accounted for 38% of the variance in probable ADHD severity ($F_{3,365} =$

Table 2

Associations between variables after correction for multitesting (N = 385).

	ASRS-A	ASRS-B	ADHD	Inatt	Hyper	GD	Stress	Anx	Depr	MZQ	Age	BMI
FA	0.40**	0.40**	0.44**	0.43**	0.40**	0.37**	0.33**	0.27**	0.40**	-0.26**	-0.14	0.27**
ASRS-A		0.62**	0.85**	0.81**	0.58**	0.44**	0.42**	0.25**	0.48**	-0.33**	-0.15	0.09
ASRS-B			0.94**	0.75**	0.82**	0.55**	0.54**	0.41**	0.51**	-0.38**	-0.09	0.14
ADHD				0.86**	0.80**	0.56**	0.54**	0.38**	0.55**	-0.41**	-0.12	0.13
Inatt					0.60**	0.51**	0.46**	0.31**	0.55**	-0.38**	-0.16**	0.06
Hyper						0.52**	0.52**	0.42**	0.47**	-0.43**	-0.05	0.13
GD							0.90**	0.86**	0.90**	-0.54**	-0.24**	0.03
Stress								0.68**	0.73**	-0.45**	-0.23**	0.03
Anx									0.64**	-0.42**	-0.19**	0.007
Depr										-0.52**	-0.19**	0.07
MZQ											0.17**	-0.03
Age												0.25**
												0.09

FA = Modified Yale Food Addiction Scale 2.0 symptom count subscale, ASRS-A = Adult ADHD Self Report Scale part A, ASRS-B = Adult ADHD Self Report Scale part B, ADHD = Adult ADHD Self Report Scale total score, Hyper = ASRS Hyperactivity subdimension; Inatt = ASRS Inattentivity subdimension; GD = General Distress, Anx = Depression Anxiety Stress Scale - Anxiety Subscale, Stress = Depression Anxiety Stress Scale - Stress Subscale, Depr = Depression Anxiety Stress Scale - Depression Scale, MZQ = Mentalization Questionnaire Total Score, BMI = Body Mass Index. Bonferroni's correction $0.05/13 = 0.003$. *Significant after correction for multitesting.

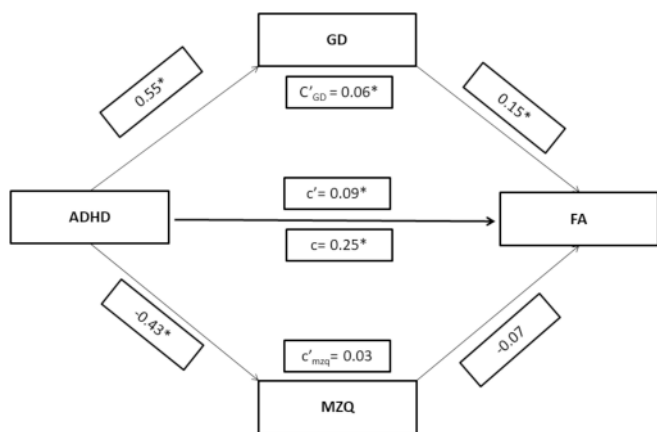


Fig. 1. Mediation model with ADHD severity as IV, FA severity as DV, general distress (GD) and mentalization capacity as mediators. Path values represent standardized regression coefficients. c' represents indirect effects of FA on ADHD (reported are confidence interval from 5000 bootstrapping analysis). " c " represents the effect of Food Addiction on ADHD symptomatology before the inclusion of the mediating variables. * significant effect.

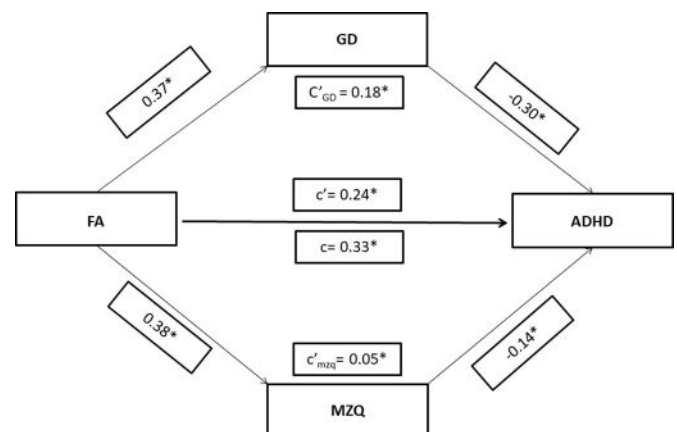


Fig. 2. Mediation model with FA severity as IV, ADHD severity as DV, general distress (GD) and mentalization capacity as mediators. Path values represent standardized regression coefficients. c' represents indirect effects of FA on ADHD (reported are confidence interval from 5000 bootstrapping analysis). " c " represents the effect of Food Addiction on ADHD symptomatology before the inclusion of the mediating variables. * significant effect.

75.08, $p < 0.001$). Both the direct ($c = 0.33$, $se = 0.06$, $t = 5.67$, $p < 0.001$) and total indirect ($c' = 0.23$, $boot\ se = 0.04$, $boot\ CI = 0.16/0.32$) effects were significant. In this inverse mediation model, both GD ($b = 0.18$, $boot\ se = 0.04$, $boot\ CI = 0.12/0.26$) and mentalization ($b = 0.05$, $boot\ se = 0.02$, $boot\ CI = 0.01/0.10$) partly mediated the relationship between FA and ADHD.

4. Discussion

In line with our starting hypothesis, FA was significantly and independently associated with ADHD symptom severity. This is in line with the results of several previous studies which investigated the possible association between FA, eating disorders and adult ADHD (Brunault et al., 2019; Carlucci et al., 2017), suggesting that ADHD symptoms are common in many individuals with an eating disorder (Nolen-Hoeksema & Rector, 2011; Ziobrowski et al., 2018). Davis et al. (2011) also noted that higher impulsivity could influence hyper-feeding behaviors and increase the susceptibility of individuals for hyper-palatable foods. Thus, it was conceivable that hyperactive-impulsive symptoms could be associated more strongly with FA severity than inattentive symptoms. Nevertheless, our results seem to suggest that there could be a combined

effect of inattentive and hyperactive-impulsive symptoms on FA (e.g., Kaisari et al., 2017; Martin et al., 2020). Supporting this hypothesis, Weiss et al. (2003) pointed out that an increase in the levels of inattention could lead to higher risk of internalizing disorders (i.e., anxiety and depression) and, in turn, the presence of these symptoms might increase severity of Food Addiction (Burrows et al., 2018). Also Martin et al. (2020), in a normative cohort of 642 young adults, reported that sex, depression and inattentive symptoms could be associated with higher disordered eating risk.

We should note that the mechanisms underlying the associations between ADHD, obesity, and patterns of disordered eating are not clear (Karaca et al., 2017), and the question concerning the order of succession of these associations is still unresolved. Preliminary studies suggested that binge eating could contribute to ADHD (Brunault et al., 2019). Conversely in many cases ADHD could precede obesity and disordered eating (Cortese et al., 2016; Erhart et al., 2012). Our mediation models indicated that the variables considered were able to explain FA or ADHD with different efficacy. For example, only 18% of the variance in FA severity was explained by adult ADHD, emotional distress, and mentalization deficits. Although, the association between ADHD and FA was not mediated by the effect of the presence of

mentalization deficits. ADHD symptoms were associated directly with FA severity and through emotional distress, so that the presence of probable adult ADHD could increase the severity of perceived distress in the individual and, in turn, the severity of FA. In this sense, ADHD, through impulsivity and inattentiveness, could directly or indirectly increase FA. This is in line with results from the recent study of [Martin et al. \(2020\)](#). The authors presented two mediation analyses, with groups of ADHD symptoms as predictors of disordered eating, and reported that, while the effect of inattentive symptoms was partly mediated by depression, the effect of hyperactive-impulsive symptoms was totally mediated by depression severity.

The inverse mediation model (compared to the ADHD → FA model), analyzing the mediated effect of FA on ADHD, was more effective in explaining ADHD (the model accounted for 38% of ADHD variance). The fact that the models were effective differently and that different paths were indicated by the models was supportive of their validity. The model indicated that emotional distress and mentalization partly mediate the relationship between FA and adult ADHD, and it supports the hypothesis that maladaptive eating patterns and FA could represent a relevant characteristic of adult ADHD. Our results lead us to hypothesize that FA symptoms (e.g., craving) could cause emotional distress and inhibit mentalization temporarily. In turn, the presence of emotional distress and the impaired ability to think about mental states could limit the ability to regulate emotional states and increase the presence of inattentive and hyperactive-impulsive symptoms. It is also possible that FA symptoms, in the presence of mentalization deficits, could limit the ability to inhibit unwanted behaviour ([Silverstein et al., 2020](#); [Tatar & Cansiz, 2020](#)).

Emotional distress and deficit in mentalization could be aspects that patients with FA and those with ADHD have in common ([Perroud et al., 2017](#)), and it may be possible that deficits in emotional processing and regulation could mediate the relationships between the deficits in response inhibition that are frequently observed in adult ADHD patients, in obese people, and in those with disordered eating patterns ([Seymour et al., 2015](#); [Nazar et al., 2018](#)). Although, highly speculative and based on correlational data, it seems that distress should be considered an important piece of the puzzle in the circular relationship between ADHD and FA (↑ADHD → ↑FA → ↑ADHD), while mentalization deficits seem to be involved only when analyzing the effect that FA has on ADHD symptoms.

In our study the prevalence of FA was at 26.7%. This is comparable to figures reported in a past study by [Gearhardt et al. \(2014\)](#), although prevalences reported in the literature are highly variable ([Imperatori et al., 2019](#); [Innamorati et al., 2017](#); [Pursey et al., 2014](#)). Our results also suggested that 11.7% of the participants may have probable adult ADHD. In previous studies investigating the prevalence of adult ADHD in the European general population, the estimates vary widely, from 1.2% to 7.3% ([Ramos-Quiroga et al., 2013](#)). Our figures could be explained partly by the fact that we used a self-report measure generally used as screening tool and characterized by suboptimal specificity. We also should point out that the ASRS does not assess the presence of ADHD symptoms in childhood or whether their onset was before the age of 12. Nevertheless, it is also possible that ADHD may be underdiagnosed in the European adult population ([Kooij et al., 2019](#); [Valsecchi et al.,](#)).

5. Limitations

It is important to note some issues limiting the generalizability of our findings. First, the sample was composed mostly of women, and our sample was not representative of the Italian general population. Second, our sample was nonclinical in nature, and we did not include adult patients with a diagnosis of ADHD. However, investigating the possible association between probable ADHD symptoms and FA could be important in a nonclinical sample because of the implication of the presence of ADHD symptoms in worsening FA severity or dysregulated

eating patterns, even in people without a diagnosis of ADHD.

Third, even though we excluded from the study people reporting the presence of psychiatric diagnosis, we cannot rule out that adults with probable ADHD symptoms also have psychiatric symptomatology.

Fourth, our sample included few people with obesity (only 2.3% of the participants had BMI ≥ 30, and 0.5% BMI ≥ 40), limiting the possibility of generalizing our results to this population. Fifth, we did not screen participants for the possible presence of other eating disorders, and it may be possible that the high FA prevalence observed in our sample could be associated with the presence of other eating disorders (i.e., bulimia nervosa and binge eating disorder) whose symptomatology partially overlaps with that of FA. Moreover, even if the mYFAS 2.0 is a useful screening tool for FA in non clinical population, it can underestimate the number of FA symptoms.

Sixth, we used only self-report measures, potentially affected by social desirability and other biases in reporting one's own health status ([Arnold & Feldman, 1981](#)), and self-reported height and weight were used to calculate the BMI. Finally, the cross-sectional nature of the study, which precludes causal interpretations of the variables, should also be mentioned. Although, we used moderated-mediation models, their results cannot be interpreted in causal terms but only as a way of variance partitioning. Nevertheless, our results demonstrated that severity of probable ADHD could be considered to be related to FA even in our nonclinical sample.

Our study differed from previous studies and added to previous findings by investigating the possible association between ADHD and FA, while controlling for potential confounding variables and for considering the role of emotional distress and mentalization in this association. Furthermore, investigating the associations between ADHD, FA, and difficulties in mentalization in a nonclinical sample may draw attention to populations at risk of psychopathology that need preventive interventions.

6. Conclusion

In conclusion, it seems important to assess emotional distress (ED) and mentalization in individuals at risk of ADHD and FA because these factors could impact negatively on their quality of life, decrease their ability to inhibit negative responses, and increase the risk for the presence of disordered eating patterns. Moreover, clinicians should plan specific treatments with the goal of decreasing both the emotional burden of ADHD and the comorbid eating disorders. The role of treatments targeting mentalization deficits should be investigated in adult ADHD patients who present FA and other disordered eating patterns. Future studies should investigate this topic also in clinical samples with a longitudinal design to shed light on the causal relationships between adult ADHD and FA.

Authors' contributions to the manuscript

Marco Innamorati, Mariantonietta Fabbricatore and Tonia Samela designed the research; Tonia Samela take part in data acquisition; Marco Innamorati, Claudio Imperatori and Tonia Samela analyzed the data; Tonia Samela and Marco Innamorati wrote the first draft; Tonia Samela, Marco Innamorati and Giulia Raimondi wrote the second version of the draft; Tonia Samela and Marco Innamorati has primary responsibility for the final content. All authors read and approved the final manuscript.

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Declaration of competing interest

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