

# Food addiction is associated with greater objective binge eating and eating disorder psychopathology, and higher body mass index in youth, a meta-analysis

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## ABSTRACT

This meta-analysis quantifies the association between food addiction (FA) and body mass index (BMI), between FA and objective binge eating and between FA and eating disorder psychopathology in youth. We found a small positive significant association between FA and BMI and large positive significant associations between FA and objective binge eating and between FA and eating disorder psychopathology. An exploratory moderation analysis showed that FA may be more strongly correlated with objective binge eating in young adults than in adolescents. While more studies of FA examining its overlap with binge eating and eating disorder psychopathology are needed, the current data provides evidence for the construct of FA as overlapping with higher BMI, greater objective binge eating frequency, and greater self-reported eating disorder psychopathology. Further research to delineate the common and differing behavioral and neurobiological substrates of FA, Binge Eating Disorder, and obesity are needed.

## 1. Introduction

Maladaptive drug use and overconsumption of food are similar in that individuals report loss of control, with both impacting dopamine and opiate systems (Benzerouk et al., 2018; Drownowski, 1995; Gianoulakis et al., 1996; Hoebel et al., 1999; Le Merrer et al., 2009; Lyvers 2000; Volkow and O'Brien, 2007). Symptoms of addiction have also been reported outside the context of drug and alcohol abuse, in the context of addiction for particular food items. Similar to other types of addiction, individuals with food addiction (FA) often consume larger amounts of food for longer than intended and experiencing cravings or urges or a strong desire to eat these foods. Individuals with FA often report spending large amounts of time obtaining food, eating, or recovering from eating, and persistent desire or unsuccessful attempts to cut down. They may report symptoms of tolerance for instance needing to eat more to reduce negative emotions or increase pleasure, and withdrawal symptoms such as experiencing negative affect or physical symptoms when they stop eating. They describe curtailing important social occupational, or recreational activities because of the FA, and continuing to use despite problems arising from the FA. Individuals with FA describe overeating despite knowledge of adverse consequences, it leading to

physically hazardous situations (e.g., such as worsening diabetes), and failures to fulfil major role. As with any addiction, FA causes clinically significant impairment or distress (Brunault et al., 2020; Gearhardt et al., 2009).

FA is not part of the Diagnostic and Statistical Manual of Mental Disorders or the International Classification of Disease systems. In 2009, Gearhardt and Brownell created the Yale Food Addiction Scale (YFAS) using symptoms that reflect the Substance Use Disorders criteria in the Diagnostic and Statistical Manual of Mental Disorders, fourth edition, text version (DSM-IV-TR; American Psychiatric Association, 2000). In 2016, the Yale Food Addiction Scale Version 2.0 (YFAS 2.0) (Gearhardt et al., 2016) was developed to reflect the 12 Substance-related Addictive Disorders criteria in the Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-5) (American Psychiatric Association, 2013). Table 1 summarizes the DSM-5-TR (American Psychiatric Association, 2022) criteria for Substance-Related and Addictive Disorders, and details the YFAS 2.0 (Gearhardt et al., 2016) subscales that map onto these criteria. In Table 1 highlighted sections detail the DSM-IV-TR (American Psychiatric Association, 2000) criteria for Substance Use Disorder and the original YFAS (Gearhardt et al., 2009) subscales that map onto these criteria.

The prevalence of FA is higher in adults and adolescents with

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Acronyms:	
FA	food addiction
BMI	body mass index
BED	Binge Eating Disorder
BN	Bulimia Nervosa

overweight or obesity compared to healthy weight populations (Penzenstadler et al., 2019; Pursey et al., 2014; Yekaninejad et al., 2021). A meta-analysis found that the rate of FA in children and adolescents who were in the overweight or obese weight range was 19%, while the FA rate in the general community was 12% (Yekaninejad et al., 2021). A 2014 meta-analysis of FA in adults has shown that the FA prevalence is twice that in the overweight/obese weight range compared to those in the healthy body mass index (BMI) range (Pursey et al., 2014). However, in contrast to this, a 2021 meta-analysis using studies with clinical and community samples across the lifespan has shown that the overall prevalence of FA is 20%, with 24% of individuals in the overweight BMI range and 28% in the obese BMI range meeting FA criteria (Praxedes et al., 2022).

FA is also associated with disordered eating, especially the objectively large binges characterized by a loss of control observed in Binge Eating Disorder (BED) and Bulimia Nervosa (BN) (Gearhardt et al., 2014; Meadows et al., 2017; Peng-Li et al., 2020; Şengör and Gezer, 2019a). High rates of self-reported FA are often found in BED and BN (Gearhardt et al., 2014; Granero et al., 2014; Peng-Li et al., 2020). The DSM-5 (American Psychiatric Association, 2013) criteria for BED and BN require that individuals report engaging in recurrent objective binge eating episodes (see Table 1). Studies of BED show positive correlations between FA and the number of binge eating symptoms (Rodrigue et al., 2019), between FA and the number of binge eating episodes (Koball et al., 2016), and between FA and objective binge days (Meule et al., 2017).

Though the research indicates there is a relationship between FA and BMI, binge eating, eating disorder psychopathology, effect sizes how strongly these constructs are associated have yet to be quantified. The primary aim of the current meta-analysis is to quantify the strength of the association between FA and BMI, between FA and objective binge eating frequency and between FA and eating disorder psychopathology. The highest prevalence of FA occurs in individuals between 18- and 29-years old (Hauck et al., 2017; Marzilli et al., 2018). At the same time, there is some evidence that the onset of BED is bimodal i.e., occurring immediately after puberty and/or in late adolescence (Hauck et al., 2017; Marzilli et al., 2018). Given this, we explored the question of whether adolescent/young adult age moderates the relationship between FA and BMI, between FA and objective binge eating, and between FA and eating disorder psychopathology, hypothesizing that the relationships with FA will be stronger in young adults than adolescents.

## 2. Methods

This meta-analysis was performed based on the PRISMA 2020 guidelines (Page et al., 2021).

### 2.1. Literature search strategies

A search was conducted in the PubMed database from March 9, 2021, to April 29, 2021. Search terms related to food addiction, obesity, overweight, BMI, BED, and feeding and eating disorders were used. Table 2 details the search terms and provides the number of records. Resulting articles were hand-filtered to determine if they met our inclusion criteria.

**Table 1**

Mapping of Food Addiction criteria operationalized by the Yale Food Addiction Scale to Diagnostic Statistical Manual criteria and CDC criteria.

#	SRAD (DSM-5-TR) <sup>‡</sup>	FA (YFAS 2.0 based on the DSM-5) <sup>†</sup>	BED (DSM-5-TR).	BN (DSM-5-TR)	Obesity <sup>¶</sup> (CDC)
1	(A1) Consumes the substance in larger amounts or over a longer period than intended.	(1) Consume larger amounts of food for longer than intended	(Ai) recurrent binge eating episodes (i.e., eating a large amount of food in a discrete period of time, with a sense of loss of control),	(Ai) recurrent binge eating episodes (i.e., eating a large amount of food in a discrete period of time, with a sense of loss of control),	<b>Adults:</b> BMI $\geq 30$ kg/m <sup>2</sup> or greater; <b>Adolescent:</b> BMI percentile $\geq 95$ th
2	(A2) Persistent desire to cut down or repeated unsuccessful attempts to quit.	(2) Persistent desire or repeated unsuccessful attempts to quit	(Ai) loss of control over eating	(Ai) loss of control over eating	
3	(A3) Great deal of time spent obtaining/using/recovering from use.	(3) Much time/activity to obtain/use/recover.			
4	(A4) Craving	(11) Craving			
5	(A5) Failure in role obligation (e.g., work, school)	(5) Failure to fulfill major role obligation			
6	(A6) Use despite interpersonal/social consequences	(6) Use despite interpersonal/social consequences			
7	(A7) Important social, occupation, recreational activities given up or reduced	(7) Important social, occupational, or recreational activities given up			
8	(A8) Use in physically hazardous situations	(11) Use in physically hazardous situations			
9	(A9) Use despite recurrent physical or psychological problems.	(8) Use continues despite knowledge of emotional or physical problems			
10	(A10) Tolerance (marked increase in amount; marked decrease in effect)	(6) Tolerance			
11	(A11) Withdrawal (Characteristic withdrawal symptoms;	(7) Withdrawal			

(continued on next page)

**Table 1** (continued)

#	SRAD (DSM-5-TR) <sup>†</sup>	FA (YFAS 2.0 based on the DSM-5) <sup>‡</sup>	BED (DSM-5-TR).	BN (DSM-5-TR).	Obesity <sup>¥</sup> (CDC)
	substance taken to relieve withdrawal)				

NB: Binge Eating Disorder (BED); Bulimia Nervosa (BN); Body Mass Index (BMI); Centers for Disease Control and Prevention (CDC) (CDC, 2021a, 2021b); Diagnostic and Statistical Manual of Mental Disorders Text Version 4 (DSM-IV-TR) (American Psychiatric Association, 2000); DSM-5-TR (American Psychiatric Association, 2022); Food Addiction (FA); Substance-Related and Addictive Disorders (SRAD) from the DSM-5-TR (American Psychiatric Association, 2022); Yale Food Addiction Scale - Version 2 (YFAS 2.0) (Gearhardt et al., 2016).

<sup>†</sup> Gray section in the SRAD (DSM-5-TR) (American Psychiatric Association, 2022) column highlights the 7 of 12 Criterion A criteria of the Substance Use Disorder (DSM-IV-TR) (American Psychiatric Association, 2000) diagnosis. Note that the 12th criterion is the presence of clinical impairment or significant distress is Part B and not denoted here but is part of SRAD (DSM-5-TR) and the Substance Use Disorder criteria (DSM-IV-TR) (American Psychiatric Association, 2000).

<sup>‡</sup> Gray sections in the Food Addiction column highlights the DSM-IV-TR (American Psychiatric Association, 2000) criteria used for the YFAS (Gearhardt et al., 2009).

<sup>¥</sup> Assuming that overconsuming food leads to weight gain over time.

**Table 2**

Search terms used for literature searches conducted in PubMed.

Script
("food addiction" AND "Surveys and Questionnaires") AND ("obesity" OR "overweight" OR "Feeding and Eating Disorders" OR "binge eating disorder" OR "eating disorder psychopathology")
((("Food Addiction"[Mesh]) AND "Surveys and Questionnaires"[Mesh]) AND "Binge Eating Disorder"[Mesh])
"Food Addiction" AND "Surveys and Questionnaires" AND "Binge Eating Disorder" ((("Feeding and Eating Disorders"[Mesh]) AND "Food Addiction"[Mesh]) AND "Surveys and Questionnaires"[Mesh])
"Feeding and Eating Disorders" AND "Food Addiction" AND "Surveys and Questionnaires"
"binge eating" AND "food addiction" AND "food addiction scale"
"Loss of control" AND "food addiction scale"
((("Food Addiction"[Mesh]) AND "Surveys and Questionnaires"[Mesh]) AND "Obesity"[Mesh])
"Food Addiction" AND "Surveys and Questionnaires" AND "Obesity"
"food addiction scale" AND "obesity"
((("Overweight"[Mesh]) AND "Food Addiction"[Mesh]) AND "Surveys and Questionnaires"[Mesh])
"Overweight" AND "Food Addiction" AND "Surveys and Questionnaires"
"food addiction scale" AND "overweight"
"Food Addiction" AND "Surveys and Questionnaires" AND "Eating Disorder Psychopathology"
((("Food Addiction"[Mesh]) AND "Surveys and Questionnaires"[Mesh]) AND "Feeding and Eating Disorders"[Mesh])
"Food Addiction" AND "Surveys and Questionnaires" AND "Feeding and Eating Disorders"

## 2.2. Eligibility criteria

We included studies that examined the relationships between FA and BMI, obesity and/or overweight, objective binge eating, or eating disorder psychopathology. We only included: (1) empirical studies with samples with a range of BMI or studies with samples in the overweight or obese BMI-class groups and a control group, (2) studies where the mean BMI of the total sample was within the healthy, overweight, or obese range based on Centers for Disease Control and Prevention BMI criteria for children and teenagers, and for adults (Centers for Disease Control and Prevention, 2021; June 7; Centers for Disease Control and Prevention, 2021; June 21). We included studies: (3) that described the sample as young adults or emerging adults or adolescents or university students recruited from universities or colleges, and (4) which used the YFAS to assess FA. YFAS versions used in these studies were the Yale Food

Addiction Scale (YFAS), Yale Food Addiction Scale Version 2.0 (YFAS 2.0), Yale Food Addiction Scale for Children (YFAS - C), and the Modified Yale Food Addiction Scale (mYFAS) (Flint et al., 2014; Gearhardt et al., 2013, 2016).

We excluded reports where data was duplicated, or that did not include our target age groups, e.g. reports with samples of older adults (e.g., older adults, children, pre-adolescents). Reports with samples of patients with disorders or conditions that may affect appetite or weight, for instance Type II diabetes, individuals with substance dependence, individuals on medication for schizophrenia, and individuals with depression, (n = 28) were excluded. We were concerned that these medical conditions and disorders would confound the attempts to examine the relationship between FA and the constructs of BMI, objective binge eating, and eating disorders psychopathology. We also excluded reports where insufficient data was reported to allow for calculation of an effect size, for instance reports where no index of variance such as a standard deviation or standard error was reported. We excluded reports not written in English. We excluded studies that were qualitative such as reviews.

## 2.3. Selection and data collection process

After removing 877 duplicates, 452 papers remained. Titles and abstracts of these studies were evaluated by one reviewer (KB), and 98 records were excluded during this stage. In the next phase, 354 full articles were assessed for eligibility by four independent reviewers for the initial screening (KB, SK, MF, BS) with another reviewer examining the papers for the data-analysis criteria (MF). 35 studies were included in the meta-analysis. We extracted information about participants (sample size, age, age group, sex, college students or not, BMI, race, ethnicity, country), the FA measure used, how objective and/or subjective binge eating, eating disorder psychopathology, and the type of effect size quantified. During both the selection and data collection phases, we checked that there was agreement in including and excluding studies. Questions regarding the extraction of outcomes were resolved in meetings with EYC (51.02% of studies). We emailed authors (2 papers) where there were concerns that the data sample was duplicated and we could not establish this from the manuscripts. In this situation, we did not receive a response but made the decision to include the larger more recent sample where the constructs of interest were assessed. We emailed authors (3 papers) where it was clear that authors had the data that we were interested in but did not report this data in the manuscript, receiving response to 1 of 3 of these inquiries.

## 2.4. Data synthesis and meta-analysis

### 2.4.1. Effect size calculations

In the current study, the construct of food addiction was operationalized by "food addiction symptom count," "YFAS score," and "food addiction diagnosis (yes or no)" based on any version of the YFAS. We analyzed the association between FA and these constructs: BMI (27 papers), binge eating (10 papers), and eating disorder psychopathology (9 papers), yielding a total of 35 papers (note some papers yielded data for multiple associations with FA).

First, for the FA and BMI association, we included papers that reported mean BMI for groups with or without FA (i.e., Cohen's d), correlations between FA and BMI (correlation, r), or prevalence of FA in different BMI groups (e.g., healthy weight, overweight, and obese groups) using odds ratio.

Second, for examining the association between FA and objective binge eating, we used papers that reported mean objective binge eating days for groups with or without FA, correlations between combined objective and subjective binge eating episodes and FA (Cohen's d), or correlations between Binge Eating Scale scores and FA (correlation, r). Measures for binge eating included the Binge Eating Scale (BES), Eating Disorder Examination - Questionnaire (EDEQ) (objective binge days),

and Youth Eating Disorder Examination-Questionnaire (YEDE-Q) (Fairburn and Beglin, 1994; Goldschmidt et al., 2007; Gormally et al., 1982).

Last, we analyzed the association between FA and eating disorder psychopathology using papers that reported the prevalence of eating disorder diagnosis in groups with and without FA (i.e., Cohen's *d*), the correlations between eating disorder scale scores and FA (i.e., correlation, *r*), or prevalence of eating disorder diagnoses for those with and without FA (i.e., odds ratio). Assessments used to assess eating disorder psychopathology include Eating Attitudes Test (EAT-26) and eating disorder diagnosis based on clinical records (Garner et al., 1982).

Cohen's *d* was calculated using means, standard deviations, and group sizes for studies using between-group comparisons between individuals with and without FA (Wilson, 2001). For studies reporting a correlation coefficient, *r*, an odds-ratio (OR), or proportion values/binary data, these were converted to Cohen's *d* in order to combine the effect sizes across studies (Borenstein et al., 2009; Friedman, 1968; Lin, 2020; Wilson, 2001). To avoid publication bias, publications reporting multiple outcomes in a given analysis were averaged and included as one entry for the overall effect size per study (Field, 2013). For all papers, the standard error of each averaged effect size was calculated using the sample size (Hedges and Olkin, 1985). Finally, a weighted value was assigned based on the standard error, using the inverse variance method (Hedges and Vevea, 1998; Marín-Martínez and Sánchez-Meca, 2010).

Some papers reported their numbers of FA participants separately in different BMI classes: healthy weight, overweight, and obesity. For papers reporting binary/proportion data, we calculated Cohen's *d* based on the healthy weight group versus the overweight, obese, and Class 3 obesity groups.

We made some adjustments when calculating effect sizes, given that the included studies reported different types of data. Some studies calculated Spearman rank correlation coefficient,  $\rho$  instead of Pearson's correlation coefficient *r*. To include these studies in our analysis, we regarded  $\rho$  as *r* (Bourdier et al., 2018; Schulte et al., 2018). In cases where a study only reported the Pearson correlation between FA and BMI *z* score (Lin et al., 2020), we converted this correlation to Cohen's *d* directly and analyzed it with other effect sizes obtained from the associations between FA and BMI raw scores. When raw BMI means and standard deviations for the FA group and non-FA group were needed to compute Cohen's *d*, we converted BMI *z* scores to raw scores first (Vidmar et al., 2021).

#### 2.4.2. Main analysis calculations

The main analysis was conducted with R 4.0.5 using the metafor package. We used a random effects model, as this model does not assume uniform variability in samples across studies (Field, 2013). Specifically, we used the method developed by Hedges and colleague's (Hedge's estimator, HE; (Hedges and Olkin, 1985). Pooled effects between 0.2 and 0.49 were considered small; pooled effects between 0.50 and 0.79, medium, and 0.80 or greater, large (Cohen, 1988). Heterogeneity was assessed using  $I^2$  (Higgins and Thompson, 2002) and Cochran's *Q*-test (Cochran, 1954). Tests were 2-tailed and significance was defined as  $p < 0.05$ .

#### 2.4.3. Moderator analysis

A moderator analysis was conducted separately for sample age (young adulthood or adolescence). When a sample in a paper was described as 'emerging adults', 'university students', 'young adults', or as a 'college sample', we categorized this as a study with young adults. When a sample in paper was described as 'adolescents', we categorized this as a study of adolescents. We also checked if these descriptors matched the mean or median age of the study sample. If the moderator analysis yielded significant differences across the subgroups, we calculated the effect sizes for subgroups separately.

#### 2.4.4. Risk of bias assessment across studies

We ran a sensitivity analysis to identify possible outliers using a

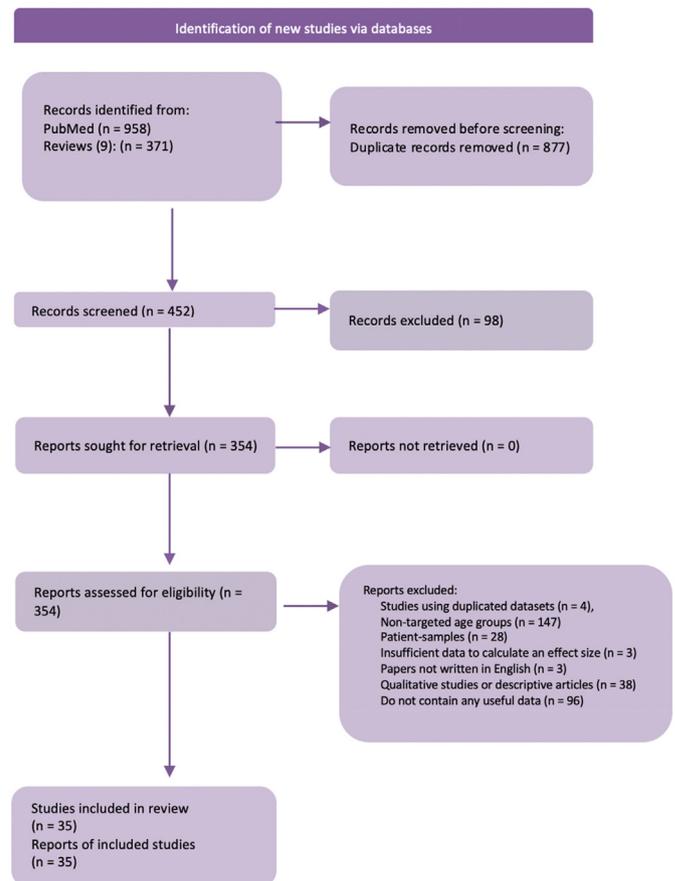


Fig. 1. PRISMA 2020 flowchart.

Forest plot. Publication bias was assessed using Egger's funnel plot (Egger et al., 1997). A leave-one-out diagnostic, where the analysis was rerun leaving out one study at a time, was conducted where there were concerns that outlier studies may have undue influence on the findings (Viechtbauer and Cheung, 2010).

#### 2.4.5. Power analysis

A power analysis was calculated using the R metapower package (Viechtbauer, 2010), which calculates power using the summary effect size, the average number of participants per study, the number of effect sizes, and the  $I^2$  value to calculate power.

### 3. Results

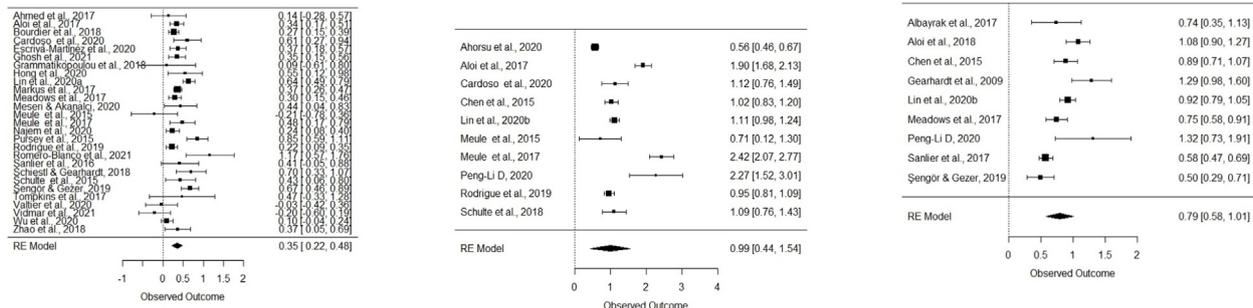
#### 3.1. Identified studies for meta-analysis

1329 entries were identified through our search, with 371 identified from reviews. 354 were assessed after screening the abstract and title. Of these, 35 publications met the full inclusion criteria, see Fig. 1 for the PRISMA 2020 flowchart (Page et al., 2021). Reports using duplicated datasets were excluded ( $n = 4$ ). Reports that did not include our target age groups, e.g. reports with samples of older adults ( $n = 147$ ) were also excluded. Reports with samples of patients with disorders or conditions that may affect appetite or weight, for instance Type II diabetes, individuals with substance dependence, individuals on medication for schizophrenia, and individuals with depression, ( $n = 28$ ) were excluded. Reports that did not provide sufficient data to calculate an effect size ( $n = 3$ ) were excluded, e.g. reports where a crude odds ratio or adjusted odds ratio was presented that could not be converted to a Cohen's *d*. We excluded reports not written in English ( $n = 3$ ), or that were qualitative studies ( $n = 38$ ). This included book chapters describing models or

**Table 3**  
Characteristics of included studies.

#	Citation	Age	OBE sample	ES calculation	Outcome type(s)	ES BMI	ES BE	ES ED path
1	Ahmed & Sayed (2017)	adolescent	no	OR to d	BMI	0.14	--	--
2	Ahorsu et al. (2020)	adolescent	yes	r to d	BE	.-	0.56	--
3	Albayrak et al. (2017)	adolescent	no	not converted (d) & OR to d	ED path	.-	--	0.74
4	Aloi et al. (2017)	young adult	no	r to d	BMI, BE, ED Path	0.34	1.90	1.08
5	Bourdier et al. (2018)	young adult	no	r to d	BMI	0.27	--	--
6	Cardoso et al. (2020)	young adult	no	r to d	BMI, BE	0.61	1.12	--
7	Chen et al. (2015)	adolescent	no	r to d	BE, ED Path	.-	1.02	0.89
8	Escrivá-Martínez et al. (2020)	young adult	no	r to d	BMI	0.18	--	--
9	Gearhardt et al. (2009)	young adult	no	r to d	ED path	.-	--	1.29
10	Ghosh et al. (2021)	young adult	no	r to d	BMI	0.35	--	--
11	Grammatikopoulou et al. (2018)	young adult	no	not converted (d)	BMI	0.09	--	--
12	Hong et al. (2020)	young adult	no	not converted (d)	BMI	0.55	--	--
13	Lin et al. (2020)	adolescent	yes	r to d	BMI	0.64	--	--
14	Lin et al. (2021)	adolescent	yes	r to d	BE, ED Path	.-	1.11	0.86
15	Markus et al. (2017)	young adult	no	r to d	BMI	0.37	--	--
16	Meadows et al. (2017)	young adult	no	r to d	BMI, ED Path	0.30	--	0.75
17	Meseri & Akanalci (2020)	adolescent	no	OR to d	BMI	0.44	--	--
18	Meule et al. (2015)	adolescent	yes	not converted (d)	BMI, BE	-0.21	0.71	--
19	Meule et al. (2017)	young adult	no	not converted (d)	BMI, BE	0.48	2.42	--
20	Najem et al. (2020)	young adult	no	r to d	BMI	0.12	--	--
21	Peng-Li et al. (2020)	young adult	no	r to d	BE, ED Path	.-	2.27	1.32
22	Pursey et al. (2015)	young adult	no	not converted (d)	BMI	0.85	--	--
23	Rodrigue et al. (2019)	adolescent	no	r to d	BMI, BE	0.22	0.95	--
24	Romero-Blanco et al., 2021b	young adult	no	OR to d	BMI	1.17	--	--
25	Sanlier et al. (2016)	young adult	no	OR to d	BMI	-0.41	--	--
26	Sanlier et al. (2017)	young adult	no	r to d	ED Path	--	--	0.28
27	Schiestl & Gearhardt (2018)	adolescent	no	r to d	BMI	0.70	--	--
28	Schulte et al. (2015)	young adult	no	r to d	BMI	0.43	--	--
29	Schulte et al. (2018)	adolescent	yes	r to d	BE	.-	0.84	--
30	Şengör and Gezer, 2019b	young adult	no	r to d	BMI, ED Path	0.67	--	0.50
31	Tompkins et al. (2017)	adolescent	yes	r to d	BMI	0.47	--	--
32	Valtier et al. (2020)	adolescent	no	OR to d	BMI	-0.03	--	--
33	Vidmar et al. (2021)	adolescent	yes	not converted (d)	BMI	-0.20	--	--
34	Wu et al. (2020)	young adult	no	r to d	BMI	0.10	--	--
35	Zhao et al. (2018)	adolescent	no	not converted (d)	BMI	0.37	--	--
1	Ahmed and Sayed (2017), 2017	adolescent	No	OR to d	BMI	0.14	--	--
2	Ahorsu et al. (2020)	adolescent	Yes	r to d	BE	.-	0.56	--
3	Albayrak et al. (2017)	adolescent	No	d*& OR to d	ED path	.-	--	0.74
4	Aloi et al. (2017)	young adult	No	r to d	BMI, BE, ED Path	0.34	1.9	1.08
5	Bourdier et al. (2018)	young adult	No	r to d	BMI	0.27	--	--
6	Cardoso et al. (2020)	young adult	No	r to d	BMI, BE	0.61	1.12	--
7	Chen et al. (2015)	adolescent	No	r to d	BE, ED Path	.-	1.02	0.89
8	Escrivá-Martínez et al. (2020)	young adult	No	r to d	BMI	0.18	--	--
9	Gearhardt et al. (2009)	young adult	No	r to d	ED path	.-	--	1.29
10	Ghosh et al. (2021)	young adult	No	r to d	BMI	0.35	--	--
11	Grammatikopoulou et al. (2018)	young adult	No	d*	BMI	0.09	--	--
12	Hong et al. (2020)	young adult	No	d*	BMI	0.55	--	--
13	Lin et al. (2020)	adolescent	Yes	r to d	BMI	0.64	--	--
14	Lin et al. (2021)	adolescent	Yes	r to d	BE, ED Path	.-	1.11	0.86
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25	Sanlier et al. (2016)	young adult	No	OR to d	BMI	0.41	--	--
26	Sanlier et al. (2017)	young adult	No	r to d	ED Path	--	--	0.28
27	Schiestl & Gearhardt (2018)	adolescent	No	r to d	BMI	0.7	--	--
28	Schulte et al. (2015)	young adult	No	r to d	BMI	0.43	--	--
29	Schulte et al. (2018)	adolescent	Yes	r to d	BE	.-	0.84	--
30	Şengör and Gezer, 2019b	young adult	No	r to d	BMI, ED Path	0.67	--	0.5
31	Tompkins et al. (2017)	adolescent	Yes	r to d	BMI	0.47	--	--
32	Valtier et al. (2020)	adolescent	No	OR to d	BMI	-0.03	--	--
33	Vidmar et al. (2021)	adolescent	Yes	d*	BMI	-0.2	--	--
34	Wu et al. (2020)	young adult	No	r to d	BMI	0.1	--	--
35	Zhao et al. (2018)	adolescent	No	d*	BMI	0.37	--	--

Abbreviations. OBE = objective binge eating; BMI = Body Mass Index (kg/m<sup>2</sup>); d = Cohen's d; ED path = eating disorder psychopathology; ES = effect size; FA = food addiction; \* = effect size d used was reported in the paper and not converted; Obesity = yes/no meets criteria for obesity; OR = odd ratio coefficient; r = correlation, r, coefficient; YFAS = Yale Food Addiction scale.

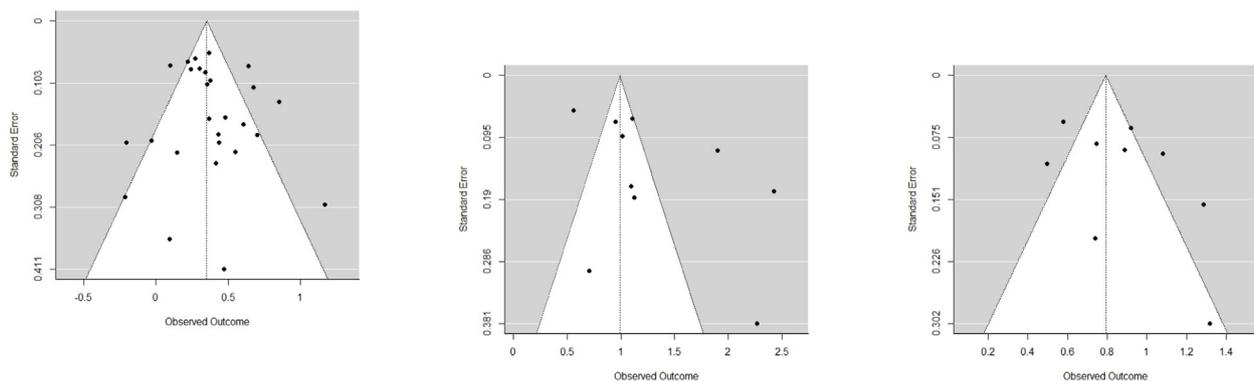


**Fig. 2.** Forest plot of effect sizes for each outcome.

Figure a. Forest plot of effect sizes of each study (N = 27) for the relationship between Food Addiction and Body Mass Index.

Figure b. Forest plot of effect sizes of each study (N = 10) for the relationship between Food Addiction and Objective Binge Eating.

Figure c. Forest plot of effect sizes of each study (N = 9) for the relationship between Food Addiction and Eating Disorders Psychopathology.



**Fig. 3.** Egger's Funnel Plot by each outcome type.

Figure a. Eggers plot of each study (N = 27) for the relationship between Food Addiction and Body Mass Index.

Figure b. Eggers plot of each study (N = 10) for the relationship between Food Addiction and Objective Binge Eating.

Figure c. Eggers plot of each study (N = 9) for the relationship between Food Addiction and Eating Disorder Psychopathology.

theories, journal reviews, and opinion pieces. This resulted in the inclusion of 27 reports that had a BMI outcome measure, 10 studies using objective binge eating outcomes, and 9 reports that included eating disorder psychopathology measures. See Table 3 and Supplement Table 1 for descriptive details of included studies.

### 3.2. Effect sizes and moderator analyses

**3.2.1. BMI.** FA status had a small positive and significant association with BMI,  $d = 0.35$ ,  $p < 0.0001$ , CI: 0.22 to 0.48. There was significant heterogeneity ( $I^2 = 81.54\%$ ;  $Q(26) = 88.19$ ,  $p < 0.0001$ ). The moderator for age ( $p = 0.97$ ) was not significant.

**3.2.2. Objective Binge eating.** There was a large and significant association between FA and objective binge eating,  $d = 0.99$ ,  $p < 0.0004$ , CI: 0.44 to 1.54. Heterogeneity was significant ( $I^2 = 97.31\%$ ;  $Q(9) = 207.66$ ,  $p < 0.0001$ ). Age moderated the relationship between FA and objective binge eating, and this was significant,  $QM(1) = 12.43$ ,  $p < 0.0004$ , with a stronger association found during young adulthood ( $n = 4$ ) compared to adolescence ( $n = 6$ ). For young adults, there was a large, significant effect size for the association between FA and objective binge eating,  $d = 1.88$ ,  $p < 0.0001$ , CI: 1.23 to 2.53, with significant heterogeneity ( $I^2 = 89.25\%$ ;  $Q(3) = 26.54$ ,  $p < 0.0001$ ). For adolescents, there was a significant large effect size, demonstrating a positive association between FA and objective binge eating,  $d = 0.85$ ,  $p < 0.0001$ , CI: 0.68 to 1.03 with significant heterogeneity ( $I^2 = 78.42\%$ ;  $Q(5) = 51.41$ ,  $p < 0.0001$ ).

**3.2.3. Eating Disorder Psychopathology.** FA status had a large positive significant association with eating disorder psychopathology,  $d$

$= 0.79$ ,  $p < 0.0001$ , CI: 0.58 to 1.01 with high heterogeneity  $I^2 = 87.48\%$ ;  $Q(8) = 49.64$ ,  $p < 0.0001$ . The moderator for age was not significant ( $p = 0.50$ ).

### 3.3. Power analysis

Power measurements for the BMI, binge eating, and the eating disorder psychopathology were all  $>99.9\%$ , indicating we had significant power to detect our main results.

### 3.4. Risk of bias across studies

A qualitative review of the Forest plots for each outcome (see Fig. 2) identified possible outliers for the objective binge eating outcome (Meule et al., 2017; Romero-Blanco et al., 2021a). An Egger's funnel plot identified the same potential outliers for binge eating (see Fig. 3), introducing potential publication bias. Nonetheless, in a leave-out-one diagnostic analysis for the association between FA and objective binge eating, effect sizes ranged from 0.92 to 1.19, and  $p$ 's  $< 0.004$ . This indicated the effect sizes for the association between FA and binge eating were consistently large and significant.

## 4. Discussion

### 4.1. Summary of results

In the 35 included studies (N = 17,774), we found that greater FA symptoms are more strongly and significantly associated with greater

objective binge eating and with higher levels of eating disorders psychopathology in youth; these effects sizes were both large. There is also a small and significant positive association between FA symptoms and greater BMI in youth. Exploratory analyses showed that age significantly moderated the positive association between FA and objective binge eating but not with BMI or eating disorder psychopathology. While our exploratory results show that the relationship between FA and objective binge eating was stronger in young adulthood than in adolescence, this finding must be taken with caution given the small sample size of studies.

#### 4.2. Relevance of results to previous research

Our results support previous systematic reviews and meta-analyses showing a positive significant relationship between FA and eating and weight disorders throughout the lifespan (Albayrak et al., 2017; di Giacomo et al., 2022; Meadows et al., 2017; Peng-Li et al., 2020; Penzenstadler et al., 2019; Praxedes et al., 2022; Şengör and Gezer, 2019a; Yekaninejad et al., 2021). Our finding of a stronger association between FA and objective binge eating and between FA and eating disorders psychopathology, relative to a relationship with BMI may reflect the younger age of our included study samples. However, other meta-analyses conducted in samples at different ages across the lifespan also show stronger associations between FA and BED relative to FA and obesity. Another meta-analysis of 6 controlled studies, using different search and analytic methods, showed that FA has higher odds of co-occurrence with BED than obesity (di Giacomo et al., 2022). Another 2022 meta-analysis of clinical and community samples across the lifespan showed that the prevalence of FA was approximately 55% in individuals meeting criteria for BED, while 24% of individuals in the overweight BMI range and 28% in the obese BMI range met FA criteria, with 20% across all samples meeting FA criteria (Praxedes et al., 2022). FA appears more strongly related to eating disorders involving objective binge eating than to the chronic medical condition of obesity and this appears to be across the lifespan. However, large scale epidemiological studies that use random or representative sampling to examine the relationship between FA, BED, and obesity are needed.

#### 4.3. Overlapping neurobiological mechanisms of FA, BED and obesity

Significant associations between FA with binge eating, eating disorder psychopathology, and higher BMI suggest that these problems may have common underlying behavioral and neurobiological mechanisms and risk factors. For instance, overeating, and binge eating in the context of FA, BED, and obesity may in part be driven by systems common to hedonic and homeostatic processes in response to highly palatable food rewards over time. Studies of individuals with FA, BED, and obesity relative to those without these problems, in response to highly palatable foods show functional and structural differences in the medial orbitofrontal cortex, a region key in monitoring the reward value of food including sensory-specific satiety, and in reward decision-making (Beyer et al., 2019; Chen et al., 2017; Gearhardt et al., 2011; Guzzardi et al., 2018; Schafer 2010; Schienle et al., 2009; Murdaugh et al., 2012; Neseliler et al., 2019). In responding to food stimuli, the medial orbitofrontal cortex acts in concert with other brain regions that are important in taste response (insula, thalamus), reward response (amygdala, putamen, caudate, nucleus accumbens) and in regulating homeostatis (hypothalamus, and cerebellar regions) (Chen et al., 2020; Chen and Zeffiro, 2020; Murray and Fellows, 2022; Rolls, 2019). Research measuring hormone levels involved in hunger and satiety also support shared mechanisms between individuals with FA, BED, and obesity. Leptin is a hormone that signals satiety and acts on receptors particularly in the hypothalamus. Studies show that there is dysregulated leptin response in FA, BED, and obesity (Caldas et al., 2022; Graßmann et al., 2017; Peters et al., 2018; Yagin et al., 2020). Genetic research, also provide evidence for neurobiological similarities in hunger and satiety cues related to FA, BED, and obesity, although more research in this area is needed to fully

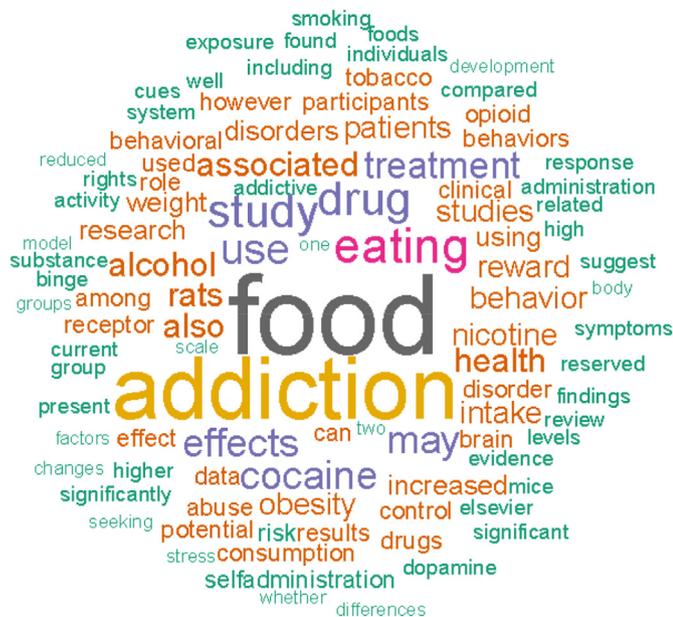
elucidate the genetic underpinnings of these constructs. For example, genome-wide association studies show that obesity and possibly FA are both associated with Fat mass and obesity-associated (FTO) gene variants (Cornelis et al., 2016; Frayling et al., 2007; Dina et al., 2007; Vasan et al., 2012; Gong et al., 2013). Other studies show that FTO may be associated with impaired satiety response (Benedict et al., 2012; Melhorn et al., 2018). In contrast, BED, when controlling for BMI, in the one genome-wide association study available, appears associated with Human homeostatic iron regulator protein, Melanin-concentrating hormone receptor 2, and Low density lipoprotein receptor-related protein 11, genes which are associated with impaired iron metabolism (Burstein et al., 2022). It may be the overconsumption high-fat, high-sugar, overly processed food that is common to FA, BED, and obesity may interact with homeostatic and hedonic systems to promote similar neural, hormonal and genetic responses. BED and obesity appear associated with poorer reward-related decision-making, working memory, and adaptive emotional functioning/emotion regulation skills, and reduced inhibitory control (Amlung et al., 2016; Cury et al., 2020; Fernandes et al., 2018; Lavagnino et al., 2016; Leeher et al., 2015; Rotge et al., 2017; Iceta et al., 2021; Voon, 2015; Wu et al., 2013; Yang et al., 2018). These are constructs that are also associated with the neural networks of which the medial orbitofrontal cortex is part of such as the executive functioning, salience, and default mode networks. Hunger and satiety processing, reward-related decision-making, executive functioning, and emotional functioning/emotion regulation skills, and reduced inhibitory control are constructs that as yet to be delineated more systematically in FA. These differences in executive functioning, reward decision-making, and emotion regulation may result from and/or interact with the overconsumption of poor quality foods, and this may maintain the overeating or binge eating in not only FA but also BED and obesity.

#### 4.4. Limitations and strengths

Our findings are limited by the heterogeneity of measures used to assess binge eating, eating disorders psychopathology, and BMI. Heterogeneity in measures leads to variability in effect size estimates across studies. For instance, in the studies included in the meta-analysis, BMI may have been self-reported or assessed in-person, and in the case of adolescents or young adults, scaled given normative data. We also included studies that used different versions of the YFAS measure assessing FA, and this may also have led to greater variability in effect size estimates. More broadly, FA is assessed using a self-reported measure, which may result in biased findings. We noticed that studies varied in the correlation indices used for the calculation of effect size - some reported using Spearman's  $r$  and others, Pearson's  $r$ , others did not report what correlation index was used. Because Spearman and Pearson's  $r$  estimates are not equivalent, this may have also led to error in our estimates. We focused on objective binge eating as a construct of interest in relation to FA. A limitation of including only studies of objective binge-eating as opposed to subjective binge-eating is that the latter is more frequently seen in AN. High rates of FA in restricting AN have been reported (Praxedes et al., 2022), but this may be indicative of the distress in overeating and loss of control experienced during subjective binges in AN. The current meta-analysis adds to the literature in assessing the strength of the association between FA and BMI, FA and objective binge eating, and FA and eating disorders psychopathology particularly in youth in studies conducted in multiple countries. Studying FA in youth is important as the highest prevalence of FA occurs in individuals between 18- and 29-years, with evidence that the onset of BED may occur immediately after puberty or in late adolescence (Hauck et al., 2017; Marzilli et al., 2018). Our meta-analysis also extends the literature by assessing eating disorder behaviors rather than focusing on diagnoses.

#### 4.5. Future directions

The number of publications on 'food addiction' have increased four-



**Fig. 4.** Wordcloud of the words in Pubmed abstracts that are frequently associated with the term ‘food addiction’.

fold since 2009 to 2021 (PubMed search, 5/11/2022). Meta-analyses on ‘food addiction’ have more than doubled from 2013 to 2021 (2013–2021). Construction of a Wordcloud of words from PubMed abstracts that depicts the frequency of the occurrence of the term ‘food addiction’ with other terms such as ‘binge’ and ‘obesity’, suggest by the size of these words in the wordcloud that these terms do not frequently occur together in this database, see Fig. 4. This stands in contrast to our current findings that suggests that FA, overlaps with higher BMI and BED.

More epidemiological studies using random sampling or nationally representative samples are needed to understand the rate of co-occurrence in FA, obesity and BED, as well as the overlap between FA and objective binge eating, and eating disorders psychopathology. More studies are needed to examine the relationship between FA, BED, and obesity, to delineate common and differing behavioral and neurobiological substrates of these disorders. This is important given the broader implications for understanding where FA stands in the context of the current DSM-5 nosology of problems that include binge eating and overeating. FA may more parsimoniously account for the binge eating found in Feeding and Eating Disorders such as BED as well as the overeating observed in the medical condition of obesity. If FA is to be regarded as a substance use disorder, it may be similar to a behavioral addiction such as gambling. It is unclear what the substance of abuse or addiction in FA is, whether this is poor diet quality, high-fat, high-sugar foods, ultraprocessed or highly processed foods, or foods with a high glycemic index, with future research needed to understand if these foods result in similar overeating and binge eating behaviors.

#### CRedit author statement

**Kehan Bao:** Writing- Original draft preparation, Writing- Reviewing and Editing, Visualization, Data Curation, **Elan French:** Methodology, Writing- Reviewing and Editing, Visualization, Data Curation, **Brooke Schleyer:** Writing- Reviewing and Editing, Data Curation, **Shely Khai-kin:** Writing- Reviewing and Editing, Data Curation, **Eunice Y Chen:** Conceptualization, Writing- Reviewing and Editing, Supervision, Visualization.

#### Declaration of competing interest

The authors declare that they have no known competing financial

interests or personal relationships that could have appeared to influence the work reported in this paper.

#### Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.psychom.2022.100067>.

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