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Co–Occurrence of Food Addiction, Obesity, Problematic Substance Use, and Parental History of Problematic Alcohol Use

Lindzey V. Hoover^{1,2}, Hayley P. Yu², Jenna R. Cummings^{3,4}, Stuart G. Ferguson⁵, Ashley N. Gearhardt²

²Department of Psychology, University of Michigan, Ann Arbor

³Social and Behavioral Sciences Branch, Division of Intramural Population Health Research, Eunice Kennedy Shriver National Institute of Child Health and Human Development

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⁵College of Health and Medicine, University of Tasmania, Launceston, TAS, Australia

Abstract

Objective: The current study investigates rates of co-occurrence among food addiction (FA), problematic substance use (alcohol, cannabis, cigarettes, nicotine vaping), parental history of problematic alcohol use, and obesity as an important step to understanding whether an addictivelike eating phenotype exists.

Method: A community sample of 357 US adults (49.7% male, 77.6% white, mean age 40.7) completed the Yale Food Addiction Scale 2.0 (YFAS2.0), the Alcohol Use Disorders Identification Test (AUDIT), the Cannabis Use Disorders Identification Test (CUDIT), the Fagerstrom Test for Nicotine Dependence (FTND), the E–Cigarette Dependence Scale (EDS), the Family Tree Questionnaire (FTQ), and demographic/self-report BMI questions through Amazon Mechanical Turk (Mturk). Risk ratios (unadjusted and adjusted for sociodemographic covariates) were calculated using modified Poisson regression.

Results: Risk of FA was higher in participants with problematic alcohol use (Risk Ratio (RR)=2.13, 99% CI [1.32, 3.45]), smoking (RR=1.86, 99% CI [0.82, 3.36]), cannabis use (unadjusted; RR=2.22, 99% CI [1.17, 4.18]), vaping (RR=2.71, 99% CI [1.75, 4.21]), and parental

¹Correspondence concerning this article should be addressed to Lindzey V. Hoover, 530 Church Street, Ann Arbor, MI 48109, USA, lindzeyh@umich.edu.

Author Contributions

LVH: Conceptualization, data curation, formal analysis, methodology, project administration, supervision, visualization, writing-original draft, writing-review and editing

HPY: Conceptualization, data curation, funding acquisition, project administration, writing – review and editing.

JRC: Conceptualization, writing-original, writing-review and edit

SGF: Conceptualization, writing-original, writing-review and edit

ANG: Conceptualization, data curation, formal analysis, funding acquisition, methodology, project administration, supervision, writing-original draft, writing-review and editing

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history of problematic alcohol use (RR=2.35, 99% CI [1.46, 3.79]). Risk of FA in participants with obesity was only higher in adjusted models (RR=1.87, 99% CI [1.06, 3.27]). Obesity was not significantly associated with problematic substance use and parental history of problematic alcohol use.

Conclusions: FA, but not obesity, co-occurred with problematic substance use and a parental history of problematic alcohol use. Results supports the conceptualization of FA as an addictive disorder. The inclusion of FA as an addictive disorder in diagnostic frameworks is an important area of future consideration.

Keywords

Food Addiction; Substance Use; Obesity; Family History

An ongoing debate surrounds the existence of a phenotype for addiction to rewarding, highly processed (HP) foods (Gearhardt & Hebebrand, 2021). This phenotype, commonly referred to as food addiction, is conceptualized as a substance-based addiction to HP foods containing unnaturally high concentrations of refined carbohydrates and fat (Schulte et al., 2015). The Yale Food Addiction Scale 2.0 (YFAS2.0; (Gearhardt et al., 2016)) is a validated measure that operationalizes food addiction by applying the diagnostic criteria for substance use disorders to highly processed food intake (American Psychiatric Association, 2013). Food addiction as measured by the YFAS 2.0 is a clinically relevant construct associated with lower quality of life (Minhas et al., 2021; Nunes-Neto et al., 2018), increased psychopathology (Burrows et al., 2018; Nunes-Neto et al., 2018), and worse treatment outcomes (e.g., weight loss, disordered eating; Fielding-Singh et al., 2019; Romero et al., 2019).

Preliminary evidence supports the conceptualization of food addiction as an addictive phenotype. Similar mechanisms are implicated in food addiction and problematic substance use (i.e., a pattern of intake that may indicate elevated risk for a substance use disorder), including higher impulsivity, reward dysfunction and emotion dysregulation (Gearhardt & Schulte, 2021; Hardy et al., 2018; Minhas et al., 2021). A remaining unanswered question is whether food addiction is associated with a family history of problematic substance use. A family history of problematic substance use is a known risk factor for substance use disorders (Stone et al., 2012). One key piece of evidence supporting the re-classification of gambling disorder as an addictive disorder was its heightened association with a family history of problematic substance use (Grant & Chamberlain, 2015; Hasin et al., 2013). However, no prior study, to our knowledge, has investigated whether a family history of problematic substance use is associated with food addiction.

High rates of co-occurrence between gambling disorder and substance use disorder also supported the re-categorization of gambling disorder as an addictive disorder (Grant & Chamberlain, 2015; Hasin et al., 2013). Existing research investigating whether food addiction and problematic substance use co-occur is mixed (Berenson et al., 2015; Ivezaj et al., 2017; Mies et al., 2017; Müller et al., 2018; Nunes-Neto et al., 2018). One factor that may be contributing to these mixed findings is the nature of the samples used to investigate this question. Some samples have included only individuals with obesity who are seeking

weight-loss surgery (Ivezaj et al., 2017; Müller et al., 2018). Food addiction also occurs in non-obese individuals and, regardless of weight status, this phenotype is associated with heightened impulsivity and worse quality of life (Lerma-Cabrera et al., 2016; Minhas et al., 2021; Schulte & Gearhardt, 2020). Thus, studies that only include individuals with obesity are missing a clinically relevant subset of individuals with food addiction. Other samples in this literature are predominantly female (Berenson et al., 2015; Nunes-Neto et al., 2018). Problematic substance use is more prevalent in male individuals (American Psychiatric Association, 2013; Vasilenko et al., 2017), which may impact the ability to detect associations with food addiction in samples that are mostly female. Thus, studies examining associations among food addiction and problematic substance use with more balanced sex- and weight-distributions are needed.

The current study aims to address these gaps in the literature by utilizing a sample ($n=357$) with an even sex-distribution (49.7% male) that includes participants with and without obesity to investigate rates of co-occurrence among food addiction, parental history of problematic alcohol use, and problematic substance use (i.e., alcohol, cannabis, cigarettes, nicotine vaping). We focused on parental history of problematic alcohol use, as alcohol is a widely used, legal substance. We hypothesized that a parental history of problematic alcohol use and problematic substance use would co-occur with food addiction. Evidence supports conceptualizing food addiction and obesity as distinct constructs (Lerma-Cabrera et al., 2016; Minhas et al., 2021; Schulte & Gearhardt, 2020) and existing literature on obesity and problematic substance use is mixed (Hasin & Kilcoyne, 2012; Le Strat & Le Foll, 2011; Sayon-Orea et al., 2011). Thus, we also investigated the co-occurrence of obesity with a parental history of problematic alcohol use, problematic substance use, and food addiction.

Methods

Transparency and Openness

We report how we determined our sample size, all data exclusions, and all measures in the study. We follow Journal Article Reporting Standards JARS (Appelbaum et al., 2018). Data and analysis code are available at <https://deepblue.lib.umich.edu/data>. Data were analyzed using IBM SPSS Statistics version 27 (IBM Corp, 2020). The study was not pre-registered.

Participants

Participants (Qualifications: U.S. Location, > 95% approval rating by other investigators, Age > 18) were recruited through Amazon Mechanical Turk (MTurk) for a study on how past experiences (parental history, trauma) impact health behaviors (eating, substance use). MTurk provides a platform for collecting high-quality data from demographically varied participants (Berinsky et al., 2012; Hauser et al., 2019). See table 1 for demographic characteristics.

Procedures

The University of Michigan Institution Review Board approved procedures in accordance with provisions of the World Medical Association Declaration of Helsinki. Our sample size ($n=357$) was determined based on financial considerations. Participants consented and

completed questionnaires through MTurk. Participants reported demographic information and were compensated \$1.25 for their time (approximately 30 minutes).

Measures

Food addiction—The Yale Food Addiction Scale 2.0 (YFAS2.0; (Gearhardt et al., 2016), a 35-item measure of HP food addiction based off substance use disorder diagnostic criteria, was used to determine whether participants met criteria for food addiction (defined as 2+ symptoms and clinical impairment; coded as 0 = not met, 1 = met criteria). Participants responded to each question using frequency response options (0=never to 7=everyday) over the past year and responses were scored to indicate the number of symptoms endorsed (0 to 11). In our sample, 24.1% of participants met criteria for food addiction ($M=3.38$ symptoms, $SD=4.34$, $minmax=0.00-11.00$, $\alpha=.98$).

Problematic Substance Use—The Alcohol Use Disorders Identification Test (AUDIT; (Saunders et al., 1993)) is a brief 10-item self-report measure that screens for problematic alcohol use. Item scoring ranges from 0 to 4 resulting in a possible overall score ranging from 0 to 40. Participants scoring 8 or higher met criteria for problematic use based on established thresholds for hazardous or harmful alcohol consumption (0=not met, 1=met criteria; (Saunders et al., 1993)). In our sample, 19.6% of participants met criteria for problematic alcohol use ($M=5.17$, $SD=8.68$, $min-max=0-35$, $\alpha=.95$).

The Cannabis Use Disorders Identification Test – Revised (CUDIT; (Adamson et al., 2010)) is a brief 8-item self-report measure that screens for problematic cannabis use. Item scoring ranges from 0 to 4 resulting in a possible overall score ranging from 0 to 32. Participants scoring 8 or higher met criteria for problematic cannabis use based on established thresholds for hazardous cannabis use (0=not met, 1=met criteria; (Adamson et al., 2010)). In our sample, 6.3% of participants met criteria for problematic cannabis use ($M=1.24$, $SD=4.16$, $min-max=0-23$, $\alpha=.92$).

The Fagerstrom Test for Nicotine Dependence (FTND; (Heatherton et al., 1991)) is a 6item measure of cigarette dependence. Items are scored 0 or 1 for yes/no questions and 0 to 3 for multiple choice questions resulting in a possible overall score ranging from 0 to 10. A cutoff score of 4 or higher was used to indicate problematic cigarette use based on recommendations from past literature (Huang et al., 2008). In our sample, 13.6% of participants met criteria for problematic cigarette use ($M=0.92$, $SD=2.11$, $min-max=0-9$, $\alpha=.86$).

The E-Cigarette Dependence Scale – Brief Version (EDS; (Morean et al., 2019)) is a 4item measure of e-cigarette dependence. Participants respond to each question on a 5-point scale (0=never to 5=almost always) and responses were summed for an overall score ranging from 0 to 20. No scoring cutoff was indicated. Thus, we used the FTND recommended cutoff score of 4 or higher (Huang et al., 2008) for problematic nicotine vaping (0=not met, 1=met criteria). In our sample, 7.8% of participants met criteria for problematic nicotine vaping ($M=0.82$, $SD=2.96$, $min-max=0-15$, $\alpha=.98$).

Parental History of Problematic Alcohol Use—The Family Tree Questionnaire (FTQ; (Mann et al., 1985)) measured parental history of problematic alcohol use. Participants who indicated one or more biological parent with a possible or definite drinking problem were coded as having a parental history of problematic alcohol use (0=not met, 1=met criteria). In our sample, 33.2% of participants met criteria for a parental history of problematic alcohol use.

Obesity—Body mass index (BMI) was calculated using self-report height and weight. Obesity was indicated by a BMI of 30.0 or above based on the Center for Disease Control cut-off ((Center for Disease Control and Prevention, 2021); Not Obese, BMI <30.0=0, Obese, BMI ≥30.0=1). In our sample, 20.0% of participants met criteria for obesity.

Demographics—Participants completed self-report demographic questions (e.g., age, sex at birth, gender; see table 1) as open text or multiple-choice responses. Subjective socioeconomic status (SES) was measured by asking participants to indicate their self-ranking on a ladder representing people in the US (10=most money, most education, most respected jobs and 1=least money, least education, least respected jobs; (Adler et al., 2000)).

Data analytic plan

Analyses presented were not pre-registered and thus are considered exploratory. Analyses were conducted in IBM SPSS Statistics version 27 (IBM Corp, 2020). Data were reviewed for outliers ($\pm 3SD$) and skewness (>2). To improve data quality (Buhrmester et al., 2018) we excluded data from 29 participants who failed quality control checks (incorrectly answered 2+ quality control questions ($n=15$); preferred not to answer 10% of questions ($n=7$); completed questionnaires in <10 minutes ($n=7$)). BMI data were also excluded for participants with improbable values (BMI <15 or >50 ($n=22$)). Participant's ability to skip individual questions resulted in some missing data ($n=2$ to $n=25$), which were removed using pairwise deletion.

Pearson zero-order correlational analyses were conducted to identify potential sociodemographic covariates (see table S1 in Supplemental Materials). Age, sex at birth, and SES were included as covariates in adjusted analyses. Modified Poisson regression (with robust standard error estimations; (Zou, 2004)) were used to estimate risk ratios among food addiction, parental history of problematic alcohol use, personal problematic substance use (i.e., alcohol, cannabis, cigarettes, nicotine vaping), and obesity. Unadjusted and adjusted results are presented. Significance was set at $p<.05$. However, given multiple testing, 99% CI estimates are reported instead of 95% CI estimates.

Results

Table 2 presents the unadjusted and adjusted risk ratios (RR) with 99% confidence intervals among food addiction, parental history of problematic alcohol use, personal problematic substance use, and obesity. Risk of food addiction was significantly higher in participants with personal problematic alcohol use, smoking, and vaping and with parental history of problematic alcohol use. Risk of food addiction was significantly higher in participants with personal problematic cannabis use in the unadjusted model, but associations were

nonsignificant in the adjusted model. Risk of food addiction was significantly higher in participants with obesity only in the adjusted model. Unlike food addiction, risk of obesity was not significantly higher in participants with personal problematic substance use or parental history of problematic alcohol use.

Discussion

In a sex-balanced community sample ($n=357$) of individuals with and without obesity, a parental history of problematic alcohol use was associated with food addiction. Problematic alcohol use, smoking, and vaping were also more likely to co-occur with food addiction. The cooccurrence of food addiction with a parental history of problematic alcohol use and personal problematic substance use is consistent with shared mechanisms (e.g., impulsivity, reward dysfunction, emotion dysregulation) contributing to both types of compulsive intake (Gearhardt & Schulte, 2021; Hardy et al., 2018; Minhas et al., 2021). Evidence that gambling was associated with a familial history of problematic substance use and co-occurred with other substance use disorders supported the reconceptualization of gambling as an addictive disorder (Grant & Chamberlain, 2015; Hasin et al., 2013). While evidence that food addiction was associated with a parental history of problematic alcohol use and co-occurred with other problematic substance use is not sufficient to confirm the appropriateness of an addictive conceptualization, the absence of these associations would challenge an addictive conceptualization.

There are several factors that could be contributing to the association between a parental history of problematic alcohol use and food addiction. A parental history of problematic substance use has been associated with individual differences in a number of biopsychosocial mechanisms (e.g., reward dysfunction, inhibitory control difficulties; (Andrews et al., 2011)) that are also associated with food addiction (Gearhardt & Schulte, 2021). One possibility is that risk for food addiction is transferred, in part, through the inheritance of biological vulnerabilities in neural reward circuitry (e.g., mesolimbic dopamine and endogenous opioid pathways;(Oberlin et al., 2013; Wand et al., 1998)) implicated in both substance use (Herz, 1998) and reward-driven eating (Berridge et al., 2010). It is also possible that a parental history of substance use increases the risk of food addiction through indirect pathways, such as environmental factors (e.g., reduced parental monitoring, increased family stress (Chassin et al., 1993)). In animal models, prenatal exposure to addictive substances is associated with reward-driven eating in offspring (Malanga & Kosofsky, 2003; Pinheiro et al., 2015), which may explain associations between parental history of substance use and food addiction. Future research is needed to identify which of these (or other) potential factors contributes to the association between food addiction and a parental history of problematic substance use.

The association of parental history of problematic alcohol use with food addiction is consistent with previous findings that parents with a history of problematic substance use have children with increased sweet preferences and reward-driven eating (Cummings et al., 2020; Fortuna, 2010). Notably, parental history of problematic alcohol use was more strongly associated with food addiction than personal problematic substance use (including personal alcohol use). One possible explanation is that unlike with other substances, almost

everyone is repeatedly exposed to HP foods beginning early in development (Herrick et al., 2020; Wang et al., 2021). This repeated, widespread exposure to HP foods may contribute to stronger associations between familial risk and food addiction (relative to addiction to other substances, which may never be consumed by those with increased familial risk). Children with a parental history of problematic substance use may be particularly vulnerable to developing food addiction especially in combination with a food environment where highly rewarding HP foods are cheap, accessible, and heavily marketed (Moodie et al., 2013). Like adults, children can exhibit signs of food addiction, which is associated with poorer mental and physical health (Skinner et al., 2021), and recent estimates of food addiction in children and adolescence are comparable to rates seen in adults (15%; (Yekaninejad et al., 2021)). Investigating whether a parental history of problematic substance use is associated food addiction in children is an important next step and may be important in informing prevention efforts.

In addition to parental history of substance use, food addiction also co-occurred with personal problematic substance use across multiple different substance classes (alcohol, nicotine, vaping). Such findings suggest that screening and treatment planning for food addiction may be an important consideration when treating substance use disorders. Importantly, food addiction was associated with problematic substance use across multiple substances which vary in their impact on satiety and hunger. Indeed, both alcohol, a substance known to increase appetite and impair self-regulatory processes that might disinhibit consumption (Hofmann et al., 2011), and nicotine, an appetite suppressant (Mineur et al., 2011) co-occurred with food addiction.

Interestingly, cannabis, a substance that is strongly associated with increased appetitive drive for food (Patel & Cone, 2015), was the only substance non-significantly associated with food addiction in adjusted models. Thus, the current findings appear to be not solely attributable to the impact of a specific drugs impact on food intake or satiety. Future research should investigate factors that may be contributing to the co-occurrence between specific substances and food addiction, as well as the time course for the development of co-occurring food addiction and problematic substance use.

Unlike food addiction, obesity was not associated with either a parental history of problematic alcohol use or with a co-occurrence of problematic substance use. This is consistent with prior work showing that obesity and food addiction are distinct constructs (Lerma-Cabrera et al., 2016; Minhas et al., 2021; Schulte & Gearhardt, 2020). Individuals can consume HP food in an addictive manner, but this does not always result in excess adiposity due to a multitude of factors (e.g., dieting, excessive exercise; (Wright & Aronne, 2012)). Further, obesity is the result of a multitude of factors (e.g., genetics, medication side effects, physical inactivity (Grundy, 1998)). While food addiction is more likely to occur in individuals with obesity, a systematic review of the prevalence of food addiction suggests that only about 15-25% of individuals with obesity meet food addiction criteria (Oliveira et al., 2021). Thus, the current study suggests that the food addiction behavioral phenotype is more closely associated with an addiction profile than obese weight status.

This study has several strengths, including the use of a community sample with a balanced sex-distribution (49.7% male), the inclusion of individuals with and without obesity, and the investigation of multiple types of problematic substance use. To our knowledge, food addiction is not currently assessed in large, nationally representative samples, which prevents the investigation of these questions in existing datasets. Prevalence of obesity and parental history of alcohol use in this study were comparable to those seen in large, nationally representative studies (Grucza et al., 2010). Thus, the current study has strengths that increase confidence in the ability to investigate these associations in the current sample and highlights the importance of including measures of food addiction in future epidemiological samples with substance use disorders to allow for further investigation of these associations. Limitations of this study included the use of self-report questionnaires and BMI. The racial/ethnic distribution of the sample was primarily white (77.6%), which limits generalizability of findings to non-White samples. The current study focused only on parental history of problematic alcohol use, which did not allow for exploration of associations between food addiction and family history of problematic use across multiple substances (e.g., tobacco, cannabis) and for other family members (e.g., grandparents, siblings).

Addressing these questions is an important next step. Finally, the use of Mturk is a limitation given recent concerns about the use of Mturk data in eating disorder research (Burnette et al., 2021). Future research should utilize clinical interviews and objective measures of BMI with larger, nationally representative samples. This would also allow for sufficient power to investigate potentially important moderators, including developmental stage, sex, race/ethnicity, and SES.

In sum, food addiction co-occurred with parental history of problematic alcohol use and personal problematic substance use in a community sample of adults. Obesity did not co-occur with parental history of problematic alcohol use and personal problematic substance use, which highlights food addiction as a distinct phenotype from obesity and one that is more strongly associated with an addiction profile. The identification of parental history of problematic substance use as a shared risk factor with gambling and the co-occurrence of gambling with problematic substance use were key in identifying gambling as an addictive disorder. The current study results similarly support the conceptualization of food addiction as an addictive disorder. Parental history of problematic alcohol use may be an important risk factor for food addiction that allows for earlier detection and intervention for this high-risk group. If HP foods can trigger an addictive-like eating phenotype, public health approaches (e.g., marketing restriction, taxation) may be important to consider. The inclusion of food addiction as an addictive disorder in diagnostic frameworks is an important area of future consideration.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Public Health Significance Statements

The co-occurrence between food addiction and problematic substance use detected in this study indicates that treatment approaches may need to simultaneously consider addictive eating and problematic substance use. Parental history of problematic alcohol use appears to be a risk factor for addictive eating and may identify individuals for targeted prevention efforts. Public health approaches that have successfully reduced the negative impact of other addictive substances (e.g., marketing restrictions, taxation) may be important to consider for highly processed foods.

Table 1

Demographic Characteristics

	<i>n</i> (%)
Age (<i>M</i> = 40.7, <i>SD</i> = 12.1, <i>min-max</i> = 20-73)	
20 - 29	66 (18.8%)
30 - 39	124 (35.2%)
40 - 49	78 (22.1%)
50 - 59	51 (14.5%)
60 - 69	29 (8.2%)
70 - 73	4 (1.2%)
Sex at Birth	
Male	176 (49.7%)
Female	178 (50.3%)
Gender Identity	
Man	176 (49.7%)
Woman	177 (50.0%)
Non-binary	1 (0.3%)
Sexual Orientation	
Heterosexual	321 (90.4%)
Gay / Lesbian	9 (2.5%)
Bisexual	20 (5.6%)
Pansexual	2 (0.6%)
Asexual	2 (0.6%)
Queer	1 (0.3%)
Racial Identity [±]	
American Indian / Alaskan Native	3 (0.8%)
Hispanic / Latino	17 (4.8%)
Asian	29 (8.1%)
Native Hawaiian / Other Pacific Islander	1 (0.3%)
Black / African American	40 (11.2%)
White	277 (77.6%)
Education	
Less than high school	1 (0.3%)
High school degree	33 (9.3%)
Some college	41 (11.5%)
Associates degree	39 (11.0%)
Bachelor's degree	190 (53.5%)
Advanced degree	51 (14.4%)
Income	
Less than \$10,000	10 (2.8%)
\$10,000 - \$19,999	21 (6.0%)
\$20,000 - \$29,999	35 (10.0%)

	<i>n</i> (%)
\$30,000 – \$39,999	45 (12.8%)
\$40,000 – \$49,999	41 (11.7%)
\$50,000 – \$59,999	49 (14.0%)
\$60,000 – \$69,999	29 (8.3%)
\$70,000 – \$79,999	39 (11.1%)
\$80,000 – \$89,999	18 (5.1%)
\$90,000 – \$99,999	16 (4.6%)
\$100,000 – \$149,999	28 (8.0%)
More than \$150,000	20 (5.7%)
Subjective Socioeconomic Status [¥]	
1	8 (2.2%)
2	20 (5.6%)
3	36 (10.1%)
4	43 (12.1%)
5	102 (28.7%)
6	58 (16.3%)
7	51 (14.3%)
8	24 (6.7%)
9	11 (3.1%)
10	3 (0.8%)
BMI ^Φ (<i>M</i> = 26.0, <i>SD</i> = 5.8, <i>min-max</i> = 15.3-47.8)	
Underweight (BMI < 18.5)	14 (4.5%)
Normal Weight (BMI 18.5 – 24.9)	148 (47.7%)
Overweight (BMI 25.0 – 29.9)	86 (27.7%)
Obese (BMI > 30)	62 (20.0%)

Notes. Differences in *n*s are due to “prefer not to answer” responses.

[±] Percentages for Race / Ethnicity exceed 100% because of option to select multiple response options

[¥] Subjective Socioeconomic Status indicates participants self – ranking on a ladder representing people in the US with 10 = people who are best off (most money, most education, most respected jobs) and 1 = worst off (least money, least education, least respected jobs)

^Φ Improbable BMI values (i.e., BMI <15 or >50, *n* = 22 were excluded).

Table 2

Adjusted and Unadjusted Risk Ratios

	RR	Unadjusted [99% CI]	p	RR	Adjusted [99% CI]	p
Food Addiction (24.1%)						
<i>Alcohol</i>	3.10	[1.80, 5.33]	<.001	2.40	[1.35, 4.27]	<.001
<i>Cannabis</i>	3.13	[1.04, 9.46]	.008	2.46	[0.70, 8.58]	.064
<i>Smoking</i>	2.68	[1.34, 5.33]	<.001	2.51	[1.26, 5.04]	<.001
<i>Vaping</i>	6.38	[2.33, 17.47]	<.001	5.63	[2.35, 13.50]	<.001
<i>Parent Alcohol Hx</i>	2.03	[1.398, 2.99]	<.001	2.18	[1.47, 3.25]	<.001
<i>Obesity</i>	1.50	[0.79, 2.87]	.106	1.87	[1.06, 3.27]	.005
Alcohol (19.6%)						
<i>Food Addiction</i>	2.77	[1.75, 4.40]	<.001	2.13	[1.32, 3.45]	<.001
<i>Cannabis</i>	13.60	[3.82, 48.44]	<.001	12.12	[3.43, 42.86]	<.001
<i>Smoking</i>	3.75	[1.88, 7.49]	<.001	3.58	[1.69, 7.58]	<.001
<i>Vaping</i>	4.40	[1.72, 11.31]	<.001	3.78	[1.45, 9.86]	<.001
<i>Parent Alcohol Hx</i>	1.55	[1.01, 2.39]	.009	1.60	[1.02, 2.49]	.007
<i>Obesity</i>	0.40	[0.11, 1.42]	.062	0.48	[0.14, 1.69]	.134
Cannabis (6.3%)						
<i>Food Addiction</i>	2.22	[1.17, 4.18]	.001	1.66	[0.82, 3.36]	.063
<i>Alcohol</i>	5.00	[3.14, 7.96]	<.001	3.98	[2.34, 6.78]	<.001
<i>Smoking</i>	4.75	[2.38, 9.47]	<.001	4.04	[1.99, 8.21]	<.001
<i>Vaping</i>	3.14	[0.90, 10.94]	.018	2.20	[0.61, 7.92]	.113
<i>Parent Alcohol Hx</i>	1.36	[0.69, 2.70]	.247	1.45	[0.70, 2.98]	.185
<i>Obesity</i>	0.40	[0.03, 4.76]	.339	0.46	[0.04, 5.39]	.419
Smoking (13.6%)						
<i>Food Addiction</i>	2.23	[1.34, 3.71]	<.001	1.86	[1.13, 3.07]	.001
<i>Alcohol</i>	3.11	[1.81, 5.32]	<.001	2.65	[1.48, 4.76]	<.001
<i>Cannabis</i>	6.67	[2.41, 18.46]	<.001	5.47	[1.97, 15.19]	<.001
<i>Vaping</i>	12.12	[5.91, 24.85]	<.001	11.03	[4.20, 28.92]	<.001
<i>Parent Alcohol Hx</i>	1.41	[0.87, 2.30]	.069	1.56	[0.96, 2.51]	.018
<i>Obesity</i>	0.89	[0.33, 2.41]	.756	0.86	[0.31, 2.36]	.691
Vaping (7.8%)						
<i>Food Addiction</i>	3.39	[2.11, 5.44]	<.001	2.71	[1.75, 4.21]	<.001
<i>Alcohol</i>	3.15	[1.72, 5.79]	<.001	2.56	[1.38, 4.73]	<.001
<i>Cannabis</i>	3.34	[0.85, 13.11]	.023	2.32	[0.57, 9.41]	.122
<i>Smoking</i>	8.13	[4.72, 31.14]	<.001	7.79	[3.91, 15.55]	<.001
<i>Parent Alcohol Hx</i>	1.41	[0.78, 2.55]	.139	1.48	[0.81, 2.72]	.094
<i>Obesity</i>	0.57	[0.10, 3.23]	.402	0.64	[0.10, 4.11]	.539
Parent Alcohol Hx (33.2%)						
						1
<i>Food Addiction</i>	2.33	[1.42, 3.83]	<.001	2.35	[1.46, 3.79]	<.001
<i>Alcohol</i>	1.73	[0.98, 3.06]	.013	1.74	[0.99, 3.06]	.012
<i>Cannabis</i>	1.58	[0.53, 4.71]	.284	1.70	[0.55, 5.23]	.223

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	RR	Unadjusted [99% CI]	p	RR	Adjusted [99% CI]	p
<i>Smoking</i>	1.60	[0.79, 3.23]	.088	1.80	[0.89, 3.65]	.032
<i>Vaping</i>	1.65	[0.64, 4.28]	.174	1.74	[0.68, 4.46]	.132
<i>Obesity</i>	1.31	[0.72, 2.39]	.251	1.35	[0.75, 2.45]	.193
Obesity (20.0%)						
<i>Food Addiction</i>	1.51	[0.78, 2.93]	.107	2.11	[1.09, 4.08]	.003
<i>Alcohol</i>	0.39	[0.11, 1.41]	.058	0.45	[0.13, 1.64]	.112
<i>Cannabis</i>	0.35	[0.03, 5.08]	.315	0.42	[0.03, 6.23]	.404
<i>Smoking</i>	0.88	[0.29, 2.64]	.875	0.82	[0.25, 2.69]	.661
<i>Vaping</i>	0.53	[0.08, 3.54]	.386	0.59	[0.07, 5.26]	.530
<i>Parent Alcohol Hx</i>	1.25	[0.77, 2.05]	.241	1.31	[0.78, 2.21]	.185

Notes. Parent Alcohol Hx = Parental History of Problematic Alcohol Use.

Significant p-value set at $p < .05$; due to multiple analyses, 99% CIs are presented.

Bold in 1st column indicates predictor variable and *italicized* indicates outcome variable. Percentages in parentheses indicate the percent of participants categorized as meeting criteria for food addiction, obesity, problematic substance use, and parental problematic alcohol use as described in methods.

Adjusted Risk Ratios include sex at birth, age, and subjective socioeconomic status as sociodemographic covariates.