Published in final edited form as:

Appetite. 2020 January 01; 144: 104486. doi:10.1016/j.appet.2019.104486.

Parental Substance Use and Child Reward-Driven Eating Behaviors

Jenna R. Cummings^{a,b}, Julie C. Lumeng^{b,c}, Alison L. Miller^{b,d}, Luke W. Hyde^{a,b}, Ruby Siada^a, Ashley N. Gearhardt^{a,b}

^aDepartment of Psychology, University of Michigan, 530 Church St., Ann Arbor, MI, 48109, United States

^bCenter for Human Growth and Development, University of Michigan, 300 North Ingalls Street, Ann Arbor, MI 48109, United States

^cDepartment of Pediatrics, University of Michigan, 1500 E. Medical Center Drive, Ann Arbor, MI 48109, United States

^dSchool of Public Health, University of Michigan, 1415 Washington Heights, Ann Arbor, MI 48109, United States

Abstract

Family history of substance use is a well-established risk factor for greater substance use in adolescence and adulthood. The biological vulnerability hypothesis proposes that family history of substance use might also confer risk for obesogenic eating behavior because of similar rewarding characteristics between substances and certain foods (e.g., processed foods high in refined carbohydrates and fat). Indeed, preliminary research shows that family history of substance use is linked with sweet liking and obesity in adults; however, it is unknown whether this factor is linked to eating behavior earlier in development. The present study ($n = 52$) tested the association of severity of parental nicotine dependence and alcohol use (drinking frequency, drinking quantity, binge drinking, and number of annual drinks consumed) with two types of child [$M_{\text{age}} = 10.18$ (0.83) years] eating behavior: *homeostatic eating*, or eating regulated by internal satiety cues, and *reward-driven eating*, or eating motivated by pleasure. Results indicated that—over and above the influence of child age, child biological sex, and family income—more severe parental nicotine dependence and frequent and/or heavy, frequent parental alcohol use were associated with significantly greater child reward-driven eating as indexed by the Food Responsiveness and Enjoyment of Food subscales on the Child Eating Behavior Questionnaire. Parental substance use was not associated with child homeostatic eating as indexed by the Satiety Responsiveness subscale. Family history of substance use may be an important transdiagnostic risk factor that identifies children at risk for obesogenic, reward-driven eating behaviors.

Corresponding author: Jenna R. Cummings, Department of Psychology, University of Michigan, 1465 East Hall, 530 Church St., Ann Arbor, MI, 48109, United States jennarc@umich.edu.

Declarations of interest: None.

Keywords

child eating behavior; parental alcohol use; parental nicotine dependence; reward-driven eating

Long-standing research establishes that family history of substance use is a robust risk factor for greater substance use. For example, adults with substance use disorder are eight times more likely to have a family member with substance use disorder (Merikangas et al., 1998). Parental substance use, in particular, is linked with substance use in offspring with effects emerging as early as adolescence [$M_{\text{age}} = 15.9 (\pm 4.7)$ years], the developmental period when most people first have access to substances such as alcohol and tobacco (Biederman, Faraone, Monuteaux, & Feighner, 2000). Although family history of substance use is well established as a risk factor for greater substance use, there is little research on how this factor may more broadly relate to mental and/or physical health.

The shared biological vulnerability hypothesis proposes that the biological factors that predispose individuals towards substance use also predispose them towards obesogenic eating behavior (Gearhardt & Corbin, 2009). This would occur because the rewarding characteristics of certain foods—namely, processed foods high in refined carbohydrates and fats (e.g., chocolate, ice cream, pizza)—overlap with those of substances of abuse (e.g., alcohol, cocaine). For instance, individuals report similar behavioral reactions (e.g., loss of control, persistent craving) in response to processed foods high in refined carbohydrates and fats and in response to other substances of abuse (Schulte, Avena, & Gearhardt, 2015; Schulte, Smeal, & Gearhardt, 2017). Also, when individuals attempt to remove processed foods from their diet, that attempt may trigger psychological (i.e., depressed mood, irritability) and behavioral (i.e., increased craving, inability to concentrate) withdrawal symptoms similar to psychological and behavioral substance withdrawal symptoms (Falbe, Thompson, Patel, & Madsen, 2018; Schulte, Smeal, Lewis, & Gearhardt, 2018). Furthermore, similar neural reward circuitry activates when individuals consume processed foods and when individuals use substances of abuse (Volkow, Wang, Fowler, & Telang, 2008). However, it should be noted that neural reward circuitry activates in response to a variety of stimuli (e.g., money, sexual images, music; Volkow et al., 2013) and does not alone indicate that the rewarding characteristics of substances of abuse overlap with those of highly processed food (Heinz, Daedelow, Wackerhagen, & Di Chiara, 2019). Nevertheless, individuals with a family history of substance use might be vulnerable to processed foods high in refined carbohydrates and fat because of these overlapping rewarding characteristics and, thereby, might be vulnerable to obesogenic eating behavior.

A few studies have found preliminary support for the shared biological vulnerability hypothesis. In particular, adults with a family history of alcoholism have a greater affinity for sweet tastes in drinking solution taste tests (Kampov-Polevoy, Garbutt, & Khalitov, 2003; Kampov-Polevoy, Tsoi, Zvartau, Neznanov, & Khalitov, 2001). In two U.S. nationally representative samples of more than 39,000 participants, there were greater odds of obesity among adults with family history of alcoholism compared to those without family history (Gruza et al., 2010). Furthermore, the link between family history of alcoholism and adult obesity appears to be strengthening over time. In these nationally representative samples, the

association between family history of alcoholism and adult obesity was stronger in those surveyed in 2001–2002 relative to those surveyed in 1991–1992 (Grucza et al., 2010). This may be due to the rapidly changing nature of the food environment, which has grown more “toxic” with greater availability of processed foods high in refined carbohydrates and fat in the past few decades (Brownell, 2002).

These findings are compelling; however, there are multiple research gaps. Foremost, prior research is limited to testing associations of family history of alcoholism with sweet liking and with adult obesity. It therefore remains unknown whether family history of substance use is directly linked with *obesogenic eating behavior*, and, if so, what types of obesogenic eating behavior. *Homeostatic eating*, or eating regulated by internal satiety cues, has been extensively studied in the eating behavior literature because dysregulated homeostatic eating (i.e., inability to regulate eating based on these cues) may contribute to the development of obesity (Gale, Castracane, & Mantzoros, 2004). In empirical studies, homeostatic eating has been operationalized as Satiety Responsiveness, or reported sensitivities to internal satiety signals (Wardle et al., 2001).

Additionally, *reward-driven eating*, or eating motivated by pleasure, might contribute to the development of obesity (Lowe & Butryn, 2007). In empirical studies, reward-driven eating has been operationalized as (a) Food Responsiveness, or appetitive drive and responsiveness to food cues; (b) Enjoyment of Food, or interest in food; and/or (c) Desire to Drink, or drive and responsiveness to non-alcoholic beverage cues (Gearhardt, 2018). Dysregulated homeostatic eating and reward-driven eating have been correlated in prior work; however, the two behaviors demonstrate incremental utility in predicting obesity risk (Carnell & Wardle, 2007). Indeed, even someone who is able to regulate eating based on internal satiety cues may be susceptible to reward-driven eating because of persistent desires for food or because they experience pleasure from food, and this may be especially true when highly processed foods are readily available (Lowe & Butryn, 2007). Moreover, neural reward circuitry including the mesolimbic dopamine and endogenous opioid pathways are implicated in reward-driven eating (Berridge, Ho, Richard, & DiFeliceantonio, 2010), and these same circuitries are implicated in substance use (Volkow et al., 2008). Since family history of substance use confers risk for substance use in part through inheritance, likely via neural vulnerabilities (Oberlin et al., 2013; Wand, Mangold, & El Deiry, 1998), family history of substance use might confer risk specifically for reward-driven rather than dysregulated homeostatic eating behavior.

Another limitation of the prior research is that it predominately focuses on adults. From a developmental and prevention perspective, however, investigating whether family history of substance use confers risk for obesogenic eating behavior earlier in life is essential. First, childhood provides a unique context wherein individuals have not yet had abundant/legal access to substances of abuse (such as alcohol) but have abundant access to processed foods high in refined carbohydrates and fat—especially in light of the increasingly “toxic” food environment (Brownell, 2002). Thus, in childhood, individuals with a family history of substance use may engage in greater obesogenic eating behavior because the most rewarding substance available to them is processed food high in refined carbohydrates and fat. Second, childhood is a critical developmental period for preventing adult obesity because it is very

difficult for individuals to lose weight once obesity is established (Wing & Phelan, 2005). Child reward-driven eating behavior, in particular, is a key predictor of future excessive weight gain (Gearhardt, 2018). Understanding whether family history of substance use is associated with child reward-driven eating behavior may therefore guide prevention efforts by identifying a risk factor, and a potentially modifiable behavior pathway from this risk factor to obesity. There is one prior study suggesting that family history of substance use may be associated with child reward-driven eating behavior. Specifically, Mennella and colleagues (2010) found that children [$M_{\text{age}} = 8.2 (\pm 0.1)$ years; Range = 5–12 years] with a family history of alcoholism demonstrated a greater affinity for sweet tastes in a drinking solution taste test. It is thus plausible that family history of substance use may confer risk for reward-driven eating as early as these ages in childhood.

Lastly, prior research has focused on associations between family history of *alcoholism* and sweet liking as well as adult obesity but, given the broader overlaps in rewarding characteristics of processed foods high in refined carbohydrates and fats and substances of abuse, it is important to also consider family history of other substance use. Tobacco cigarette smoking, in particular, is essential to consider because it remains a highly prevalent substance use behavior among adults with a recent estimate of 20 million tobacco cigarette smokers in the U.S. (Jamal et al., 2018). Tobacco cigarette smoking is also the second most frequent actual cause of death in the U.S. (U.S. Burden of Disease Collaborators, 2018). Some studies have found links between parental tobacco cigarette smoking and child obesity (Burke et al., 1998; Huerta, Bibi, Haviv, Scharf, & Gdalevich, 2006; Hui, Nelson, Yu, Li, & Fok, 2003), suggesting further investigation is warranted.

The present study therefore built upon the prior literature by being the first study to test whether family history of substance use would be associated with child obesogenic eating behavior. The primary aim was to investigate the associations of family history of substance use—specifically, severity of parental nicotine dependence and alcohol use (drinking frequency, drinking quantity, binge drinking, and number of annual drinks consumed)—with child homeostatic and reward-driven eating behaviors. These associations were tested in a sample of children around age 10 from lower income families who are at greater risk for obesogenic eating behavior (Pan, Blanck, Sherry, Dalenius, & Grummer-Strawn, 2012; Patrick & Nicklas, 2013). Our hypotheses were that more severe parental substance use would be associated with greater child reward-driven eating, but more severe parental substance use would not be associated with child homeostatic eating. A secondary, exploratory aim was to test the association of family history of substance use with child obesity.

Method

Participants

Fifty-seven children were recruited into the Appetite, Behavior, and Cortisol (ABC) Brains study from records of participation in the ABC Preschool study, which investigated biopsychosocial correlates of obesity in young children (Lumeng et al., 2014). Children were invited through four quarterly newsletters sent through postal mail and at least three times by phone, email, and/or text. The ABC Preschool study originally recruited

participants from Head Start, a federally-funded preschool program for families whose income falls below federal poverty guidelines (see Lumeng et al., 2014 for details regarding the original recruitment and exclusion criteria). The ABC Brains study included a functional magnetic resonance imaging (fMRI) component so children with contradictions to safely or fully participating in fMRI paradigms (e.g., metal in his/her body, history of brain injury, diagnosis of global developmental delay) were excluded.

The majority of the children ($n = 50$) had a mother report on her substance use behavior. However, given the prior literature and the present study aims, analyses included children who had a mother or a father report on their own substance use behavior ($n = 52$)¹. Demographics for this final study sample are presented in Table 1.

Procedure

The University of Michigan Institutional Review Board approved study procedures in accordance with the provisions of the World Medical Association Declaration of Helsinki. Parents provided informed consent while children provided age-appropriate assent.

Parents completed questionnaires reporting on their own behavior, which included the Fagerstrom Test for Nicotine Dependence (Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991) and questions about drinking frequency, drinking quantity, and binge drinking (National Institute on Alcohol Abuse and Alcoholism, 2003). Parents also completed questionnaires reporting on their child's behavior, which included the Child Eating Behavior Questionnaire (Wardle, Guthrie, Sanderson, & Rapoport, 2001). While parents were responding to questionnaires, children trained on the fMRI procedure in a mock scanner and also responded to questionnaires. Next, a research assistant measured children's height (Model # Seca 214 portable stadiometer) and weight (Detecto Portable Scale Model #DR550C), and then children completed the fMRI session. Details on the fMRI session are not reported here because those data were not included in the present study aims.

Families were compensated for their time on a prorated basis: \$60 for questionnaire completion and height/weight measurement, \$20 fMRI training completion, and \$20 for fMRI session completion. All families received a \$15 gift card after the visit to purchase dinner and a \$30 flat rate travel incentive or cab ride to/from the visit to help defray travel costs.

Measures

Parent

Nicotine Dependence: Parents completed the Fagerstrom Test for Nicotine Dependence (Heatherton et al., 1991), which is a 6-item questionnaire assessing current symptoms of substance use disorder specific to tobacco cigarette smoking (i.e., quantity of cigarettes smoked, compulsion to use, physical dependence). Sample items include, "How soon after waking do you smoke your first cigarette?" and, "How many cigarettes a day do you

¹Constraining the sample to just children who had a mother report on her substance use behavior did not change the pattern or significance of results with the exception of two findings (p s < .056).

smoke?” Yes/no items were scored from 0 to 1 and multiple-choice items were scored from 0 to 3. All items were summed to yield a total score of 0 to 10; higher total scores indicated more severe parental nicotine dependence. Parents who reported that they did not currently smoke tobacco cigarettes received a score of 0.

Alcohol use: Parents answered questions about drinking frequency, drinking quantity, and binge drinking. These questions were developed by the Task Force on Recommended Alcohol Questions from the National Institute on Alcohol Abuse and Alcoholism (National Institute on Alcohol Abuse and Alcoholism, 2003). Drinking frequency was assessed with the question, “During the last 12 months, how often did you usually have any kind of drink containing alcohol? By a drink we mean half an ounce of absolute alcohol (e.g., a 12 ounce can or glass of beer or cooler, a 5 ounce glass of wine, or a drink containing 1 shot of liquor),” and responses ranged from 1 (every day) to 10 (never). Responses were reverse scored so that higher scores indicated greater parental drinking frequency, and then were converted into number of days of drinking in the last 12 months based on the midpoint of the selected response category (Dawson, 2003). Drinking quantity was assessed with the question, “During the last 12 months, how many alcoholic drinks did you have on a typical day when you drank alcohol?” and responses ranged from 1 (25 or more drinks) to 10 (1 drink). Responses were reverse scored so that higher scores indicated greater parental drinking quantity, and then were converted into number of alcoholic drinks based on the midpoint of the selected response category (Dawson, 2003). Binge drinking frequency was assessed with the question, “During the last 12 months, how often did you have 5 or more (males) or 4 or more (females) drinks containing any kind of alcohol within a two-hour period? That would be the equivalent of at least 5 or 4 12-ounce cans or bottles of beer, 5 or 4 five ounce glasses of wine, or 5 or 4 drinks each containing one shot of liquor or spirits. Choose only one,” and responses ranged from 1 (every day) to 10 (never). Responses were reverse scored so that higher scores indicated greater parental binge drinking frequency. In addition to these indices, total number of annual drinks consumed was calculated (number of days of drinking in the last 12 months x number of drinks on typical drinking day) to capture both parental drinking frequency and drinking quantity in one index (e.g., heavy, frequent vs. heavy, infrequent drinking; Dawson, 2003).

Child

Eating behavior: Parents completed the Child Eating Behavior Questionnaire (Wardle et al., 2001), which is a 35-item questionnaire assessing multiple dimensions of eating behavior observed in children. For the full questionnaire, including the text for all 35 items, see Wardle et al. (2001). Responses to items ranged from 1 (Never) to 5 (Always). For the present study, the Food Responsiveness, Desire to Drink, and Enjoyment of Food subscales were used as separate indices of child *reward-driven eating* (Gearhardt, 2018). There was high internal consistency among items for each of these subscales as indicated by Cronbach α s of .81, .92, and .87, respectively. Food Responsiveness assesses appetitive drive and responsiveness to food cues; sample items from this subscale include, “My child is always asking for food,” and, “Even if my child is full up s/he finds room to eat his/her favourite food.” Enjoyment of Food assesses interest in food; sample items from this subscale include, “My child loves food,” and, “My child looks forward to mealtimes.” Desire to Drink

assesses drive and responsiveness to non-alcoholic beverage cues; sample items from this subscale include, “My child is always asking for a drink,” and, “If given the chance, my child would drink continuously throughout the day.” The Satiety Responsiveness subscale was used as an index of *homeostatic eating*. There was adequate internal consistency among items for this subscale as indicated by a Cronbach α of .73. Satiety Responsiveness assesses sensitivity to internal satiety signals; sample items from this subscale include, “My child cannot eat a meal if s/he has had a snack just before,” and, “My child gets full before his/her meal is finished.”

Obesity: Children were categorized as having obesity (BMI 95th percentile) or not based on U.S. Centers for Disease Control and Prevention reference growth curves for child age and biological sex (see Kuczmarski et al., 2002 for more details).

Data Analytic Plan

Prior to analysis, all variables were reviewed for normality, outliers, and missing data. Due to little missing data (< 3.5% of total data were missing), a pairwise deletion approach was used for all analyses. Model estimation was conducted in SPSS Statistics Version 24 (IBM Corp.). Significance was set at $p < .05$. Linear regression was used to test associations between parental substance use and child reward-driven eating, and parental substance use and child homeostatic eating. Logistic regression was used to test whether parental substance use was associated with greater odds of child obesity.

During inspection of whether models with all parental substance use variables entered simultaneously met the assumptions for linear regression, issues of multicollinearity among the parental substance use variables were identified (see Table S1 in Supplementary Material for correlations among the parental substance use variables). All other assumptions were met. Thus, each parental substance use index was entered in a separate model with each index of child reward-driven and homeostatic eating (e.g., Model 1 = parental nicotine dependence with child Food Responsiveness, Model 2 = parental drinking frequency with child Food Responsiveness) and child obesity (e.g., Model 1 = parental nicotine dependence with child obesity, Model 2 = parental drinking frequency with child obesity). Multiple testing was corrected for using the false discovery rate (Benjamini & Hochberg, 1995). For this false discovery rate analysis, tests for each dependent variable are listed in rank-order of their p -values. Each rank is then multiplied by .05 and divided by the number of tests for that dependent variable to produce a new corrected threshold for determining significance (Benjamini & Hochberg, 1995). Child age, child biological sex, and family income were included in all models as covariates given prior associations with the variables of interest (Pan et al., 2012; Patrick & Nicklas, 2013; Wardle et al., 2001; Webber, Hill, Saxton, Van Jaarsveld, & Wardle, 2009). Family income was assessed with the question, “Thinking about your income and the income of everyone else who lives with you, what was your total household income before taxes in the past 12 months?” Responses ranged from 1 (Under \$5,000) to 9 (Greater than \$75,000).

Results

Descriptives

Descriptives of variables of interest, including means and standard deviations of reward-driven eating, homeostatic eating, and obesity, are presented in Table 1.

Child Eating Behavior

Linear regression estimates from the separate models testing associations between child reward-driven eating and parental substance use, and between child homeostatic eating and parental substance use, are presented in Table 2 (see Table S2 in Supplementary Materials for the full models including F -change estimates for R^2). Over and above the influence of child age, child biological sex, and family income, greater child reward-driven eating (including child Food Responsiveness and Enjoyment of Food) was associated with more severe parental nicotine dependence, greater parental drinking frequency, and greater number of annual drinks consumed; however, child Desire to Drink was not associated with these variables. Also, greater reward-driven eating (including child Food Responsiveness, Enjoyment of Food, and Desire to Drink) was not associated with parental drinking quantity and binge drinking.

Child homeostatic eating (child Satiety Responsiveness) was not associated with parental substance use.

Child Obesity

Logistic regression estimates from the separate models testing associations between child obesity and parental substance use are presented in Table 3 (see Table S3 in Supplementary Materials for the full models). Child obesity was not associated with parental substance use.

Correction for Multiple Comparisons

The false discovery rate analysis is presented in Table 4. Significant results remained for all associations except the following: child Enjoyment of Food and parental drinking frequency ($p = .038$; corrected threshold $p = .01$); child Enjoyment of Food and parental nicotine dependence symptoms ($p = .045$; corrected threshold $p = 0.02$); and child Enjoyment of Food and parental annual drinks consumed ($p = .048$; corrected threshold $p = .03$).

Discussion

Family history of substance use is a robust risk factor for greater substance use in adolescence and adulthood but little research has examined it as a risk factor for other mental and/or physical health outcomes across the lifespan. The biological vulnerability hypothesis suggests that family history of substance use might predict obesogenic eating behavior because there are similar rewarding characteristics between substances and certain foods (e.g., processed foods high in refined carbohydrates and fat). The present study's primary aim was to test whether family history of substance use might confer risk for child obesogenic eating behavior. Indeed, family history of substance use—specifically, more severe parental nicotine dependence and frequent and/or heavy, frequent parental alcohol use

—was associated with greater child reward-driven eating, or eating motivated by pleasure. Parental substance use was not associated with child homeostatic eating, or eating regulated by satiety, and with child obesity.

These findings should be interpreted in light of study strengths and weaknesses. Foremost, family history of substance use was operationalized by parental severity of current nicotine dependence and alcoholic drinking in the past 12 months but did not include lifetime histories (e.g., a parent who smoked tobacco cigarettes in the past but has since quit; prenatal versus postnatal use). Research shows, however, that after young adulthood tobacco cigarette smoking and alcoholic drinking patterns are relatively stable in adults (Chen & Kandel, 1995), and that adults who smoke tobacco cigarettes have the most stable substance use patterns (Chassin, Presson, Pitts, & Sherman, 2000; Chen & Kandel, 1995). Also, family history of substance use was not operationalized based on diagnosis of substance use disorder. The operational definition excluded other substances of abuse (e.g., marijuana, cocaine) and substance use of other relatives (e.g., aunts, uncles, grandparents). Prior work in this area has sometimes included and sometimes excluded substance use of other relatives, and has not included substances of abuse besides alcohol (Gruza et al., 2010; Kampov-Polevoy et al., 2003; Kampov-Polevoy et al., 2001; Mennella et al., 2010). Future research that broadens the scope of the operational definition of family history of substance use may yield new insights about associations of family history of substance use with obesogenic eating behavior and obesity.

On one hand, child reward-driven eating was associated with greater parental drinking frequency and number of annual drinks consumed. On the other hand, child reward-driven eating was not associated with parental likelihood of binge drinking or drinking a greater number of drinks at one occasion. Parents reported on average drinking 1–2 alcoholic drinks/drinking day and binge drinking between 1 or 2 days and 3–11 days in the past year, which is consistent with U.S. national estimates (Chan, Neighbors, Gilson, Larimer, & Marlatt, 2007; Kanny, Liu, Brewer, & Lu, 2013). Thus, parents that reported heavy but infrequent drinking and/or binge drinking may represent more normative substance users (and indeed, the majority of U.S. adults who binge drink do not meet criteria for alcohol dependence; Esser et al., 2014). In contrast, parents that reported frequent drinking and/or heavy, frequent drinking may exhibit characteristics of individuals with a substance use disorder. It will be critical for future research to investigate associations between family history of substance use and obesogenic eating behavior/obesity in more clinically severe samples.

Child eating behavior was assessed with a parent-report questionnaire, which can be affected by subjective bias. However, this approach has advantages because children's eating behavior is difficult to observe in the laboratory (Wardle et al., 2001). Parents also evaluate many other aspects of children's behavior (e.g., temperament) and already inform on child eating behavior to clinicians (Wardle et al., 2001). Moreover, child eating behavior was assessed relatively early in the lifespan [$M_{\text{age}} = 10.18 (0.83)$ years] and, given that effects were observed at this age, future research could investigate if family history of substance use is a risk factor for child obesogenic eating behavior at even younger ages. Also, child obesity was measured objectively by measuring height and weight in the laboratory.

The present study sample consisted of children from lower income families, and there is substantial research documenting that children from lower income families are at elevated risk for obesogenic eating behavior (Pan et al., 2012; Patrick & Nicklas, 2013). Nevertheless, the sample was moderately sized and, at best, powered to detect medium effect sizes. The associations that remained significant after correcting for multiple testing showed medium effect sizes (13.7–17.9% of variance explained). The three associations that did not remain significant after correcting for multiple testing, however, showed smaller effect sizes (8.7–9.4% of variance explained). Thus, future research should test the association of family history of substance use with child eating behavior and with child obesity in a larger sample. Moreover, our lack of observed associations between family history of substance use and child obesity may in part be explained by the small sample size. Indeed, prior research establishing associations between family history of alcoholism and adult obesity were in large, nationally representative samples (Gruza et al., 2010).

Nonetheless, the present results extend prior findings that family history of alcoholism was associated with child and adult sweet liking (Kampov-Polevoy et al., 2003; Kampov-Polevoy et al., 2001; Mennella et al., 2010). They also support the shared biological vulnerability hypothesis, which purports that the biological vulnerabilities that predispose individuals towards substance use could also predispose them towards obesogenic eating behavior (Gearhardt & Corbin, 2009). Specifically, family history of substance use confers risk for substance use in part through inheritance of biological vulnerabilities in neural reward circuitries including the mesolimbic dopamine and endogenous opioid pathways (Oberlin et al., 2013; Wand et al., 1998), and these circuitries are implicated in reward-driven eating (Berridge et al., 2010). The present study found parental substance use particularly associated with child reward-driven eating and not with child homeostatic eating. It is therefore plausible that inherited vulnerabilities in the mesolimbic dopamine and endogenous opioid pathways explain why parental substance use conferred risk for child reward-driven eating.

The present study did not directly test the biological vulnerability hypothesis, however. Thus, additional factors might explain why parental substance use was associated with child reward-driven eating. For instance, family history of substance use might confer risk for greater child reward-driven eating through family environmental factors such as less parental monitoring or greater family stress, which are additional pathways through which parental substance use confers risk for adolescent substance use (Chassin, Pillow, Curran, Molina, & Barrera, 1993). Indeed, family stress contributes to child reward-driven eating (Miller et al., 2018). It is also possible that family history of substance use may confer risk for greater child-reward driven eating through prenatal exposure to substances. In non-human animal models, prenatal exposure to substances increases offspring motivational drive for and acquisition of reward (Malanga & Kosofsky, 2003), which may increase reward-driven eating. Our measures of parental substance use did not assess parental substance use during the prenatal period, so future research should tease apart effects arising from purely postnatal versus prenatal parental substance use. For children with a family history of substance use, inherited biological vulnerabilities in neural reward circuitries, environmental factors like family stress, and prenatal exposure to substances might also work in concert to elevate risk for reward-driven eating.

Although significant associations between parental substance use and child obesity were not found in the present study, prior work consistently finds that child reward-driven eating indexed by child Food Responsiveness and Enjoyment of Food is associated with child body mass index and waist circumference (Domoff, Miller, Kaciroti, & Lumeng, 2015; Webber et al., 2009). Moreover, obesity develops over the lifespan, and a recent meta-analysis indicated that—while there was a very strong link between child and adult obesity—70% of adults with obesity did not have obesity in childhood (Simmonds, Llewellyn, Owen, & Woolacott, 2016). Thus, family history of substance use might predict adult obesity (Grucza et al., 2010) but not child obesity. Future longitudinal research could identify precisely when in the lifespan family history of substance use emerges as a risk factor for obesity.

If family history of substance use confers risk for both obesogenic eating behavior and substance use, future research might also investigate why children with a family history of substance use may become more susceptible to one, both, or neither of these health outcomes as they develop. For instance, adolescence is a developmental period when most people first have access to substances such as alcohol, tobacco, and illicit drugs. How will that access change susceptibilities to reward-driven eating? Some research shows that longitudinal increases in body mass index and consumption of processed foods high in sugar and fat during adolescence predicts less use of substances including alcohol and illicit drugs (Cummings, Ray, & Tomiyama, 2017; Gearhardt, Waller, Jester, Hyde, & Zucker, 2018). However, associations between adolescent body mass index trajectories and nicotine dependence are more complex including positive associations between adolescent obesity and young adulthood nicotine dependence (Gearhardt et al., 2018). Investigating biological and familial environmental variables relevant to those with family history of substance use as they interact over time may shed light on simultaneous, bidirectional developmental trajectories of obesogenic eating behavior and substance use.

Conclusion

Family history of substance use may be an important transdiagnostic risk factor that identifies children at risk for certain obesogenic, reward-driven eating behaviors. Recent clinical approaches have highlighted the benefits of identifying individuals best suited for interventions (Collins & Varmus, 2015) as well as the benefits of intervening on multiple behaviors at once (Spring, King, Pagoto, Van Horn, & Fisher, 2015). Screening for family history of substance use could be an efficient way of identifying individuals who may benefit from multiple behavior interventions related to obesogenic eating behavior and substance use. Future research on family history of substance use and associated mental and physical health outcomes across the lifespan will inform precisely how and when this transdiagnostic risk factor should be assessed.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

Jenna R. Cummings was supported by Award Number T32HD079350 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development. The present research was supported by Award Number R01HD061356 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, and by Award Numbers R21DK090718, R01DK098983, and RC1DK086376 from the National Institute of Diabetes and Digestive and Kidney Diseases. The present research was also supported by a MCubed grant from the University of Michigan to Julie C. Lumeng, Luke W. Hyde, and Ashley N. Gearhardt. The content of this manuscript is solely the responsibility of the authors and does not necessarily represent the official views of the Eunice Kennedy Shriver National Institute of Child Health and Human Development, the National Institute of Diabetes and Digestive and Kidney Diseases, or the National Institutes of Health.

References

- Berridge KC, Ho CY, Richard JM, & DiFeliceantonio AG (2010). The tempted brain eats: Pleasure and desire circuits in obesity and eating disorders. *Brain Research*, 1350, 43–64. doi:10.1016/j.brainres.2010.04.003 [PubMed: 20388498]
- Biederman J, Faraone SC, Monuteaux MC, & Feighner JA (2000). Patterns of alcohol and drug use in adolescents can be predicted by parental substance use disorders. *Pediatrics*, 106(792–797). [PubMed: 11015524]
- Brownell KD (2002). The environment and obesity In Fairburn CG & Brownell KD (Eds.), *Eating disorders and obesity: A comprehensive handbook* (Second Edition ed., pp. 433–438). New York, NY: Guilford Publications.
- Burke V, Gracey MP, Milligan RAK, Thompson C, Taggart AC, & Beilin LJ (1998). Parental smoking and risk factors for cardiovascular disease in 10- to 12-year old children. *The Journal of Pediatrics*, 133(2), 206–213. [PubMed: 9709707]
- Carnell S, & Wardle J (2007). Measuring behavioural susceptibility to obesity: validation of the child eating behaviour questionnaire. *Appetite*, 48(1), 104–113. [PubMed: 16962207]
- Chan KK, Neighbors C, Gilson M, Larimer ME, & Marlatt GA (2007). Epidemiological trends in drinking by age and gender: providing normative feedback to adults. *Addictive Behaviors*, 32, 967–976. doi:10.1016/j.addbeh.2006.07.003 [PubMed: 16938410]
- Chassin L, Pillow DR, Curran PJ, Molina BSG, & Barrera M (1993). Relation of parental alcoholism to early adolescent substance use: A test of three mediating mechanisms. *Journal of Abnormal Psychology*, 102(1), 3–19. [PubMed: 8436697]
- Chassin L, Presson CC, Pitts SC, & Sherman SJ (2000). The natural history of cigarette smoking from adolescence to adulthood in a midwestern community sample: Multiple trajectories and their psychosocial correlates. *Health Psychology*, 19(3), 223–231. doi:10.1037/TO7S-6133.I9.3.223 [PubMed: 10868766]
- Chen K, & Kandel DB (1995). The natural history of drug use from adolescence to the mid-thirties in a general population sample. *American Journal of Public Health*, 85(1), 41–47. [PubMed: 7832260]
- Collaborators, U. S. B. o. D., Mokdad AH, Ballestros K, Echko M, Glenn S, Olsen HE, ... Murray CJL (2018). The state of US health, 1990–2016: Burden of diseases, injuries, and risk factors among US states. *JAMA*, 319(14), 1444–1472. doi:10.1001/jama.2018.0158 [PubMed: 29634829]
- Collins FS, & Varmus H (2015). A new initiative on precision medicine. *The New England Journal of Medicine*, 372(9), 794–795.
- Cummings JR, Ray LA, & Tomiyama AJ (2017). Food-alcohol competition: As young females eat more food, do they drink less alcohol? *Journal of Health Psychology*, 22(5), 674–683. doi: 10.1177/1359105315611955 [PubMed: 26542418]
- Dawson DA (2003). Methodological issues in measuring alcohol use. *Alcohol Research & Health*, 27(1), 18–29. [PubMed: 15301397]
- Domoff SE, Miller AL, Kaciroti N, & Lumeng JC (2015). Validation of the Children's Eating Behaviour Questionnaire in a low-income preschool-aged sample in the United States. *Appetite*, 95, 415–420. doi:10.1016/j.appet.2015.08.002 [PubMed: 26247701]

- Esser MB, Hedden SL, Kanny D, Brewer RD, Gfroerer JC, & Naimi TS (2014). Prevalence of alcohol dependence among U.S. adult drinkers, 2009–2011. *Prevalence of Chronic Disease*, 11. doi: 10.5888/pcd11.140329.
- Falbe J, Thompson HR, Patel A, & Madsen KA (2018). Potentially addictive properties of sugar-sweetened beverages among adolescents. *Appetite*, 133, 130–137. doi:10.1016/j.appet.2018.10.032 [PubMed: 30385262]
- French SA, & Jeffery RW (1995). Weight concerns and smoking: A literature review. *Annals of Behavioral Medicine*, 17(3), 234–244. [PubMed: 24203535]
- Gale SM, Castracane D, & Mantzoros CS (2004). Energy homeostasis, obesity and eating disorders: Recent advances in endocrinology. *The Journal of Nutrition*, 134(2), 295–298. [PubMed: 14747663]
- Gearhardt A (2018). Role of reward pathways in appetitive drive and regulation In Lumeng JC & Fisher JO (Eds.), *Pediatric Food Preferences and Eating Behaviors* (pp. 111–126): Academic Press.
- Gearhardt AN, & Corbin WR (2009). Body mass index and alcohol consumption: Family history of alcoholism as a moderator. *Psychology of Addictive Behaviors*, 23(2), 216–225. doi:10.1037/a0015011 [PubMed: 19586138]
- Gearhardt AN, Waller R, Jester JM, Hyde LW, & Zucker RA (2018). Body mass index across adolescence and substance use problems in early adulthood. *Psychology of Addictive Behaviors*, 32(3), 309–319. doi:10.1037/adb0000365 [PubMed: 29771559]
- Gruzca RA, Krueger RF, Racette SB, Norberg KE, Hipp PR, & Bierut LJ (2010). The emerging link between alcoholism risk and obesity in the United States. *Archives of General Psychiatry*, 67(12), 1301–1308. [PubMed: 21135330]
- Harris HR, Willett WC, & Michels KB (2013). Parental smoking during pregnancy and risk of overweight and obesity in the daughter. *International Journal of Obesity*, 37(10), 1356–1363. doi: 10.1038/ijo.2013.101 [PubMed: 23736356]
- Heatherton TF, Kozlowski LT, Frecker RC, & Fagerstrom K-O (1991). The Fagerstrom Test for Nicotine Dependence: A revision of the Fagerstrom Tolerance Questionnaire. *British Journal of Addiction*, 86, 1119–1127. [PubMed: 1932883]
- Heinz A, Daedelow LS, Wackerhagen C, & Di Chiara G (2019). Addiction theory matters - Why there is no dependence on caffeine or antidepressant medication. *Addiction Biology*. doi:10.1111/adb.12735
- Huerta M, Bibi H, Haviv J, Scharf S, & Gdalevich M (2006). Parental smoking and education as determinants of overweight in Israeli children. *Preventing Chronic Disease: Public Health Research, Practice, and Policy*, 3(2), 1–9.
- Hui LL, Nelson EA, Yu LM, Li AM, & Fok TF (2003). Risk factors for childhood overweight in 6- to 7-y-old Hong Kong children. *International Journal of Obesity*, 27(11), 1411–1418. doi:10.1038/sj.ijo.0802423 [PubMed: 14574354]
- Jamal A, Phillips E, Gentzke AS, Homa DM, Babb S, King BA, & Neff LJ (2018). Current cigarette smoking among adults - United States, 2016. *Morbidity and Mortality Weekly Report*, 67(2), 53–59. [PubMed: 29346338]
- Kampov-Polevoy AB, Garbutt JC, & Khalitov E (2003). Family history of alcoholism and response to sweets. *Alcoholism: Clinical & Experimental Research*, 27(11), 1743–1749. doi:10.1097/01.ALC.0000093739.05809.DD
- Kampov-Polevoy AB, Tsoi MV, Zvartau EE, Neznanov NG, & Khalitov E (2001). Sweet liking and family history of alcoholism in hospitalized alcoholic and non-alcoholic patients. *Alcohol & Alcoholism*, 36(2), 165–170. [PubMed: 11259214]
- Kanny D, Liu Y, Brewer RD, & Lu H (2013). Binge drinking—United States, 2011. *MMWR Surveillance Summary*, 62(3), 77–80.
- Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, Mei Z, ... Johnson CL (2002). 2000 CDC growth charts for the United States: methods and development. *Vital Health Stat* 11, 5(246), 1–190.
- Lowe MR, & Butryn ML (2007). Hedonic hunger: A new dimension of appetite? *Physiology & Behavior*, 91(4), 432–439. doi:10.1016/j.physbeh.2007.04.006 [PubMed: 17531274]

- Lumeng JC, Miller A, Peterson KE, Kaciroti N, Sturza J, Rosenblum K, & Vazquez DM (2014). Diurnal cortisol pattern, eating behaviors and overweight in low-income preschool-aged children. *Appetite*, 73, 65–72. doi:10.1016/j.appet.2013.10.016 [PubMed: 24177439]
- Malanga CJ, & Kosofsky BE (2003). Does drug abuse beget drug abuse? Behavioral analysis of addiction liability in animal models of prenatal drug exposure. *Developmental Brain Research*, 147, 47–57. doi:10.1016/j.devbrainres.2003.09.019 [PubMed: 14741750]
- Mennella JA, Pepino MY, Lehmann-Castor SM, & Yourshaw LM (2010). Sweet preferences and analgesia during childhood: Effects of family history of alcoholism and depression. *Addiction*, 105(4), 666–675. doi:10.1111/j.1360-0443.2009.02865.x [PubMed: 20148789]
- Merikangas KR, Stolar M, Stevens DE, Goulet J, Preisig MA, Fenton B, ... Rounsaville BJ (1998). Familial transmission of substance use disorders. *Archives of General Psychiatry*, 55, 973–979. [PubMed: 9819065]
- Miller AL, Gearhardt AN, Retzliff L, Sturza J, Kaciroti N, & Lumeng JC (2018). Early childhood stress and child age predict longitudinal increases in obesogenic eating among low-income children. *Academic Pediatrics*, 18(6), 685–691. doi:10.1016/j.acap.2018.01.007 [PubMed: 29357310]
- National Institute on Alcohol Abuse and Alcoholism (2003, 10). Recommended alcohol questions [Online guidelines and resources]. Retrieved from: <https://www.niaaa.nih.gov/research/guidelines-and-resources/recommended-alcohol-questions>
- Oberlin BG, Dziedzic M, Tran SM, Soeurt CM, Albrecht DS, Yoder KK, & Kareken DA (2013). Beer flavor provokes striatal dopamine release in male drinkers: Mediation by family history of alcoholism. *Neuropsychopharmacology*, 38(9), 1617–1624. doi:10.1038/npp.2013.91 [PubMed: 23588036]
- Pan L, Blanck HM, Sherry B, Dalenius K, & Grummer-Strawn LM (2012). Trends in the prevalence of extreme obesity among US preschool-aged children living in low-income families, 1998–2010. *Journal of the American Medical Association*, 308(24), 2563–2565. [PubMed: 23268509]
- Patrick H, & Nicklas TA (2013). A review of family and social determinants of children's eating patterns and diet quality. *Journal of the American College of Nutrition*, 24(2), 83–92. doi: 10.1080/07315724.2005.10719448
- Schulte EM, Avena NM, & Gearhardt AN (2015). Which foods may be addictive? The roles of processing, fat content, and glycemic load. *PLoS One*, 10(2), e0117959. doi:10.1371/journal.pone.0117959 [PubMed: 25692302]
- Schulte EM, Smeal JK, & Gearhardt AN (2017). Foods are differentially associated with subjective effect report questions of abuse liability. *PLoS One*, 12(8), e0184220. doi:10.1371/journal.pone.0184220 [PubMed: 28859162]
- Schulte EM, Smeal JK, Lewis J, & Gearhardt AN (2018). Development of the Highly Processed Food Withdrawal Scale. *Appetite*, 131, 148–154. doi:10.1016/j.appet.2018.09.013 [PubMed: 30227182]
- Simmonds M, Llewellyn A, Owen CG, & Woolcott N (2016). Predicting adult obesity from childhood obesity: A systematic review and meta-analysis. *Obesity Reviews*, 17(2), 95–107. doi: 10.1111/obr.12334 [PubMed: 26696565]
- Spring B, King AC, Pagoto SL, Van Horn L, & Fisher JD (2015). Fostering multiple healthy lifestyle behaviors for primary prevention of cancer. *American Psychologist*, 70(2), 75–90. doi:10.1037/a0038806 [PubMed: 25730716]
- Volkow ND, Wang GJ, Fowler JS, & Telang F (2008). Overlapping neuronal circuits in addiction and obesity: Evidence of systems pathology. *Philosophical Transactions of the Royal Society London B: Biological Sciences*, 363(1507), 3191–3200. doi:10.1098/rstb.2008.0107
- Volkow ND, Wang GJ, Tomasi D, & Baler RD (2013). Obesity and addiction: neurobiological overlaps. *Obesity Reviews*, 14(1), 2–18. doi:10.1111/j.1467-789X.2012.01031.x [PubMed: 23016694]
- Wand GS, Mangold D, & El Deiry S (1998). Family history of alcoholism and hypothalamic opioidergic activity. *JAMA Psychiatry*, 55(12), 1114–1119. doi:10.1001/archpsyc.55.12.1114
- Wardle J, Guthrie CA, Sanderson S, & Rapoport L (2001). Development of the Children's Eating Behaviour Questionnaire. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 42(7), 963–970. doi:10.1017/S0021963001007727

- Webber L, Hill C, Saxton J, Van Jaarsveld CH, & Wardle J (2009). Eating behaviour and weight in children. *International Journal of Obesity*, 33(1), 21–28. doi:10.1038/ijo.2008.219 [PubMed: 19002146]
- Wing RR, & Phelan S (2005). Long-term weight loss maintenance. *American Journal of Clinical Nutrition*, 82, 222S–225S. [PubMed: 16002825]

Table 1

Demographics of Study Sample & Descriptives of Variables of Interest (n = 52)

	Mean	SD
	%	
Child Age	10.18	0.83
Child Biological Sex (% Female)	55.8%	
Child Race/Ethnicity (% Non-Hispanic White)	53.8%	
Family Income		
Under \$5,000	8.5%	
\$5,000 to \$9,999	12.8%	
\$10,000 to \$14,999	12.8%	
\$15,000 to \$19,999	14.9%	
\$20,000 to \$24,999	4.3%	
\$25,000 to \$34,999	12.8%	
\$35,000 to \$49,999	23.4%	
\$50,000 to \$74,999	2.1%	
Greater than \$75,000	8.5%	
Parental Body Mass Index	35.83	10.94
Underweight	9.6%	
Normal	23.1%	
Overweight	17.3%	
Obese	50.0%	
Parental Fagerstrom Test for Nicotine Dependence Summed Score	1.53	2.11
Parental Drinking Frequency (Number of drinking days in past 12 months)	31.0	69.1
Parental Drinking Quantity (Number of drinks on drinking day)	1.6	1.4
Parental Binge Drinking (Number of binge drinking events in past 12 months)	1.4	0.9
Parental Annual Drinks Consumed (Number of drinks in past 12 months)	67.8	147.9
Child Food Responsiveness	2.91	0.88
Child Enjoyment of Food	4.00	0.74
Child Desire to Drink	3.07	1.19
Child Satiety Responsiveness	2.46	0.58
Child Standardized Body Mass Index (zBMI)	1.18	0.95
Normal	42.3%	
Overweight	25.0%	
Obese	32.7%	

Notes: Standardized Body Mass Index (zBMI) quantifies the distance of children's Body Mass Index from the average Body Mass Index for their age and biological sex. zBMI was calculated using the U.S. Centers for Disease Control and Prevention recommended formula, and based on U.S. Centers for Disease Control and Prevention reference growth curves for child age and biological sex (see Kuczmarski et al., 2002 for more details).

Table 2

Multiple Linear Regression Estimates of Associations with Child Eating Behavior (n = 52)

	<i>B</i>	<i>SE_B</i>	β	<i>p</i>	95% CI		<i>R</i> ²
					Lower	Upper	
Child Food Responsiveness							
Parental FTND Sum	.19	.06	.45	.004	0.07	0.32	.179
Parental Drinking Frequency	.01	.002	.42	.013	0.001	0.01	.137
Parental Drinking Quantity	-.002	.09	-.003	.986	-0.19	0.18	.000
Parental Binge Drinking	.02	.15	.03	.882	-0.27	0.32	.001
Parental Annual Drinks Consumed	.002	.001	.44	.006	0.001	0.004	.163
Child Enjoyment of Food							
Parental FTND Sum	.12	.06	.32	.045	0.003	0.24	.088
Parental Drinking Frequency	.004	.002	.35	.038	0.0002	0.01	.094
Parental Drinking Quantity	.03	.08	.06	.705	-0.14	0.20	.003
Parental Binge Drinking	-.07	.14	-.08	.650	-0.36	0.23	.006
Parental Annual Drinks Consumed	.002	.001	.32	.048	0.0002	0.003	.087
Child Desire to Drink							
Parental FTND Sum	.01	.09	.02	.909	-0.18	0.20	.000
Parental Drinking Frequency	.005	.003	.31	.064	-0.0003	0.01	.077
Parental Drinking Quantity	-.07	.13	-.08	.599	-0.32	0.19	.007
Parental Binge Drinking	-.10	.19	-.09	.600	-0.48	0.28	.007
Parental Annual Drinks Consumed	.002	.001	.30	.067	-0.0002	0.01	.075
Child Satiety Responsiveness							
Parental FTND Sum	-.07	.04	-.24	.136	-0.16	0.02	.051
Parental Drinking Frequency	-.002	.001	-.20	.249	-0.004	0.001	.031
Parental Drinking Quantity	-.09	.06	-.21	.175	-0.21	0.04	.044
Parental Binge Drinking	-.05	.10	-.08	.645	-0.26	0.16	.006
Parental Annual Drinks Consumed	-.001	.001	-.23	.165	-0.002	0.0004	.045

Notes: FTND = Fagerstrom Test for Nicotine Dependence. Each parental substance use index was entered in a separate model with each index of child reward-driven and homeostatic eating. Child age, child biological sex, and family income were included as covariates in all models.

Table 3

Logistic Regression Estimates of Associations with Child Obesity (n = 52)

	OR	<i>p</i>	95% CI	
			Lower	Upper
Parental FTND Sum	1.37	.079	0.97	1.93
Parental Drinking Frequency	1.00	.465	0.99	1.01
Parental Drinking Quantity	1.35	.257	0.81	2.25
Parental Binge Drinking	1.26	.562	0.58	2.75
Parental Annual Drinks Consumed	1.00	.217	1.00	1.01

Notes: FTND = Fagerstrom Test for Nicotine Dependence. Each parental substance use index was entered in a separate model with child obesity. Child age, child biological sex, and family income were included as covariates in all models.

Table 4

False Discovery Rate Multiple Testing Correction

Rank of <i>p</i> -value		<i>p</i> -value from Analysis	Corrected Threshold	Accept as Significant?
Child Food Responsiveness				
1	Parental FTND Sum	.004	.01	Yes
2	Parental Annual Drinks Consumed	.006	.02	Yes
3	Parental Drinking Frequency	.013	.03	Yes
4	Parental Binge Drinking	.882	.04	No
5	Parental Drinking Quantity	.986	.05	No
Child Enjoyment of Food				
1	Parental Drinking Frequency	.038	.01	No
2	Parental FTND Sum	.045	.02	No
3	Parental Annual Drinks Consumed	.048	.03	No
4	Parental Binge Drinking	.650	.04	No
5	Parental Drinking Quantity	.705	.05	No
Child Desire to Drink				
1	Parental Drinking Frequency	.064	.01	No
2	Parental Annual Drinks Consumed	.067	.02	No
3	Parental Drinking Quantity	.599	.03	No
4	Parental Binge Drinking	.600	.04	No
5	Parental FTND Sum	.909	.05	No
Child Satiety Responsiveness				
1	Parental FTND Sum	.136	.01	No
2	Parental Annual Drinks Consumed	.165	.02	No
3	Parental Drinking Quantity	.175	.03	No
4	Parental Drinking Frequency	.249	.04	No
5	Parental Binge Drinking	.645	.05	No
Child Obesity				
1	Parental FTND Sum	.079	.01	No
2	Parental Annual Drinks Consumed	.217	.02	No
3	Parental Drinking Quantity	.257	.03	No
4	Parental Drinking Frequency	.465	.04	No
5	Parental Binge Drinking	.562	.05	No

Notes: FTND = Fagerstrom Test for Nicotine Dependence. For this false discovery rate analysis, tests for each dependent variable are listed in rank-order of their *p*-values. Each rank is then multiplied by .05 and divided by the number of tests for that dependent variable to produce a new corrected threshold for determining significance (Benjamini & Hochberg, 1995).