



Published in final edited form as:

Obesity (Silver Spring). 2013 December ; 21(12): E775–E781. doi:10.1002/oby.20500.

ABUSE VICTIMIZATION IN CHILDHOOD OR ADOLESCENCE AND RISK OF FOOD ADDICTION IN ADULT WOMEN

Susan M. Mason, PhD¹, Alan J. Flint, MD^{2,3}, Alison E. Field, ScD^{3,4,5}, S. Bryn Austin, ScD^{4,5,6}, and Janet W. Rich-Edwards, ScD^{1,3}

¹Connors Center for Women's Health and Gender Biology, Brigham and Women's Hospital and Harvard Medical School, Boston, MA

²Department of Nutrition, Harvard School of Public Health, Boston, MA

³Department of Epidemiology, Harvard School of Public Health, Boston, MA

⁴Division of Adolescent and Young Adult Medicine, Boston Children's Hospital and Harvard Medical School, Boston, MA

⁵Channing Division of Network Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA

⁶Department of Society, Human Development, and Health, Harvard School of Public Health, Boston, MA

Abstract

Objective—Child abuse appears to increase obesity risk in adulthood, but the mechanisms are unclear. This study examined the association between child abuse victimization and food addiction, a measure of stress-related overeating, in 57,321 adult participants in the Nurses' Health Study II (NHSII).

Design and Methods—The NHSII ascertained physical and sexual child abuse histories in 2001 and current food addiction in 2009. Food addiction was defined as 3 clinically significant symptoms on a modified version of the Yale Food Addiction Scale. Confounder-adjusted risk ratios (RRs) and 95% confidence intervals (CIs) were estimated using modified Poisson regression.

Results—Over eight percent of the sample reported severe physical abuse in childhood, while 5.3% reported severe sexual abuse. Eight percent met the criteria for food addiction. Women with food addiction were 6 units of BMI heavier than women without food addiction. Severe physical and severe sexual abuse were associated with roughly 90% increases in food addiction risk (physical abuse RR=1.92; 95% CI: 1.76, 2.09; sexual abuse RR=1.87; 95% CI: 1.69, 2.05). The RR for combined severe physical abuse and sexual abuse was 2.40 (95% CI: 2.16, 2.67).

Users may view, print, copy, and download text and data-mine the content in such documents, for the purposes of academic research, subject always to the full Conditions of use:http://www.nature.com/authors/editorial_policies/license.html#terms

Correspondence to: Susan M. Mason.

Corresponding Author: Susan M. Mason, 1620 Tremont Street 3rd Floor, Boston, MA 02120. Phone: 617-525-9684. n2sma@channing.harvard.edu.

Competing Interests: The authors report no competing interests.

Conclusions—A history of child abuse is strongly associated with food addiction in this population.

Keywords

child abuse; exposure to violence; stress and coping; eating behaviors

Introduction

National survey results suggest that more than a third of girls in the United States experience some form of physical or sexual child abuse by the time they turn 18 years old (1–2). Published studies indicate that child abuse is related to adult obesity (3–7), with potentially serious consequences for long-term health. In our own work, we have found that child abuse is associated with substantial increases in obesity-related disease risk in adulthood, including hypertension (8), type 2 diabetes (9), and cardiovascular events (10). The mechanisms linking abuse to weight gain remain largely unexplored.

A compelling body of animal and clinical evidence suggests that stress can dysregulate eating, promoting a preference for highly palatable (high fat/high sugar) foods and disrupting homeostasis of body weight (11–12). Laboratory research has used social stressors to induce consumption of palatable foods in humans (13), and studies have demonstrated a dose-response relationship between exogenous administration of glucocorticoid stress hormones and food intake in both animals (14–15) and humans (16). Studies further suggest that stress-related overeating has important similarities to drug addiction. Palatable food ingestion and drug use both stimulate reward systems in the brain (17) that dampen the physiologic stress response (18). Rats exposed to stressors seek palatable food in much the same way that they seek cocaine (19–20). Over time, exposure to both palatable foods and addictive drugs appears to disrupt brain reward function, resulting in withdrawal symptoms when the foods or drugs are removed (21), and encouraging continued consumption even in the face of adverse stimuli (21); brain imaging studies have highlighted neurological overlaps between uncontrolled eating and drug use (22–24).

These and other findings have motivated recent calls to define certain eating behaviors as “food addiction” (25–26). The push to define food addiction as a psychiatric disorder is controversial, but regardless of its status as a diagnosis, the food addiction construct may be useful for identifying uncontrolled eating in response to distress. We hypothesized that addiction-like eating behaviors may be one potential pathway from child abuse to obesity. There are currently no published studies on child abuse as a risk factor for food addiction. In this study, we examined the association between history of child physical or sexual abuse and food addiction among women in the Nurses’ Health Study II.

Methods and procedures

Data sources

The Nurses’ Health Study II (NHSII) follows 116,430 female registered nurses recruited at ages 25–42 in 1989. Biennial questionnaires gather sociodemographic, behavioral, and medical data. In 2001, a supplemental Violence Questionnaire asking about experiences of

physical and sexual abuse in childhood was sent to 91,297 NHSII participants who had responded to the previous biennial questionnaire within three mailings. Questionnaires were returned by 68,376 (75%) of the supplemental questionnaire recipients. Violence Questionnaire respondents were more likely to be white than the NHSII cohort as a whole, but had similar childhood socioeconomic status and childhood body size. The 2009 biennial questionnaire included a modified version of the Yale Food Addiction Scale (27), which ascertains the extent to which participants' eating behavior can be characterized as a dependency. The Institutional Review Board of Partners Health Care System (Boston, MA) approved this study.

Variables and variable definitions

Exposures—Our main exposures were physical and sexual abuse experienced in childhood or adolescence (up to and including age 17). Child and/or adolescent physical abuse was assessed using questions from the Revised Conflict Tactics Scale (28), which asked participants to report the frequency with which a parent, step-parent or adult guardian pushed, grabbed, or shoved; kicked, bit, or punched; hit with something that hurt; choked or burned; or physically attacked the participant when they were children (age 0–10) and when they were adolescents (age 11–17). As in previous analyses (9–10), we categorized physical abuse into the following four categories: none, mild (being pushed, grabbed, or shoved at any frequency or being kicked, bitten, or punched once or hit with something once), moderate (being hit with something more than once or physically attacked once), and severe (being kicked, bitten, or punched or physically attacked more than once or ever choked or burned).

Child and/or adolescent sexual abuse was ascertained by asking participants (1) whether and how often, as a child (age 0–10) or adolescent (age 11–17), they had ever been touched in a sexual way by an adult or an older child or forced to touch an adult or an older child in a sexual way when they did not want to, and (2) whether an adult or older child had ever forced or attempted to force them into any sexual activity by “threatening you, holding you down, or hurting you in some way when you did not want to?” (29). We categorized sexual abuse into the following four categories (9): none, sexual touching only, one experience of forced sexual activity, and more than one experience of forced sexual activity.

Outcome—Food addiction was assessed in 2009 using a modified version of the Yale Food Addiction Scale (YFAS) (27), which parallels measures of drug and alcohol addiction. The original YFAS uses 25 questionnaire items to assess 7 diagnostic criteria for food addiction, and has been shown to have adequate internal reliability, high convergent validity with other eating pathology constructs, and discriminant validity with related but distinct disorders such as alcohol abuse (27) in a non-clinical sample of undergraduate students. The modified Yale Food Addiction Scale (mYFAS) uses a core of 9 questionnaire items, with one question for each of the symptom groups included in the 7 diagnostic criteria, plus two items assessing clinically significant impairment and distress. In an application of the mYFAS to the validation data for the YFAS, 9% of participants met the criteria for food addiction using the mYFAS, while 11% met the criteria in the original YFAS validation

(30). The mYFAS shows good construct validity and reasonably high sensitivity (79%), providing a valid, though conservative, measure of food addiction.

The mYFAS defines food addiction as 3 or more of the following 7 symptoms plus clinically significant impairment or distress: (a) eating when no longer hungry four or more times per week, (b) worrying about cutting down on certain foods four or more times per week, (c) feeling sluggish or fatigued from overeating two or more times per week, (d) experiencing negative feelings from overeating that interfere with other activities two or more times per week, (e) having physical withdrawal symptoms when cutting down on certain foods two or more times per week, (f) continuing to consume the same amount of food despite significant emotional or physical problems due to overeating at any frequency, and (g) feeling the need to eat an increasing amount of food to reduce distress at any frequency. The mYFAS defines clinically significant impairment or distress as either (a) experiencing significant distress related to eating behavior two or more times per week or (b) experiencing a decrease in ability to function due to issues related to food two or more times per week.

Supplemental Table 1 presents the frequency with which each symptom was endorsed among women meeting the criteria for food addiction. We use the term ‘food addiction’ throughout the manuscript, as shorthand for this set of uncontrolled eating behaviors. But we caution the reader that the extent to which this reflects a physical dependency on certain foods has not been fully established.

Covariates—We included the following covariates, plausible predictors of both child abuse and food addiction, as potential confounders in adjusted models: age at baseline (continuous with a squared term), race (indicators for African American, Asian, Hispanic, and other, with non-Hispanic white as the referent), mother’s and father’s educational attainment when participant was an infant (indicators for <9, 9–11, 12, and 13–15 years, with 16+ as the referent), indicators for mother in professional occupation and father in professional occupation, indicator for parental home ownership when participant was an infant, recalled body size at age 5 (continuous; the participant could choose one of nine female figures ranging from very lean, a score of 1, to very obese, a score of 9, that best represented her body type at age five years(31)), and parental lifetime history of depression (indicator if either parent had depression history).

Data analysis

We used modified Poisson regression (Poisson regression with robust standard error estimation (32)) to estimate risk ratios for food addiction comparing women with histories of abuse to women without histories of abuse. Analyses included those women who responded to both the 2001 Violence Questionnaire, on which abuse was ascertained, and the 2009 biennial questionnaire, on which food addiction was assessed (n=63,002). Women who left 3 or more food addiction symptom questions blank were excluded (n=5,344), as were 337 additional women missing information on clinical significance of symptoms, leaving 57,321 participants for our analyses. To assess the sensitivity of our results these exclusions, we re-ran our models in datasets recreated under various assumptions about the value of the

missing outcome data; results indicated that our findings are robust to a variety of missingness patterns (see Supplemental Tables 2a and 2b).

For each of our abuse exposures, we ran an age-adjusted model and a model adjusted for additional potential confounders. Physical abuse and sexual abuse were first modeled separately. We then examined physical abuse–food addiction associations with and without any sexual abuse exposure, and sexual abuse–food addiction associations with and without any physical abuse exposure. Women missing physical abuse data were excluded from physical abuse analyses (n=176), and women missing sexual abuse data were excluded from sexual abuse analyses (n=374). Analyses of combined physical and sexual abuse excluded women missing either physical or sexual abuse (n=428).

We examined the possibility of a multiplicative interaction between physical abuse and sexual abuse by running a modified Poisson model with a physical abuse indicator, a sexual abuse indicator, and a physical-by-sexual abuse interaction term. We used a Wald test to assess the impact of the interaction term on model fit. Likewise, we assessed the potential additive interaction between physical and sexual abuse with a Wald test of the interaction term in a Poisson model with an identity link.

To determine whether food addiction risk varies with abuse timing and duration, we estimated the effects of abuse in childhood only, adolescence only, and in childhood and adolescence relative to no abuse in either period.

Finally, to estimate absolute age-adjusted food addiction risks at each level of physical and sexual abuse severity, we ran a Poisson model with an identity link, including indicator variables for each of the 16 cross-classifications of the four physical and four sexual abuse severity levels, plus age, centered at the baseline mean (35 years), coded as a continuous variable with a squared term. Food addiction risks for each level of physical and sexual abuse severity were calculated by adding the intercept of this model to the parameter estimates for each physical and sexual abuse category.

Results

There were 57,321 Violence Questionnaire respondents with complete food addiction data. Of 57,145 women with complete physical abuse data, 18.5% reported mild physical abuse in childhood or adolescence, 26.3% reported moderate physical abuse, and 8.5% reported severe physical abuse. Of 56,947 women with complete sexual abuse data, 22.4% reported sexual touching only, 5.8% reported one experience of forced sexual activity, and 5.3% reported repeated experiences of forced sexual activity.

Table 1 presents the distribution of covariates by physical and sexual abuse severity categories. Of the confounders examined, parental depression was most strongly related to child abuse. Physical and sexual child abuse were highly correlated; for example, repeated forced sex was reported by 21.3% of women with a history of severe physical abuse, compared to just 2.4% of women with no physical abuse history. Overall, 8.2% of our sample met the criteria for food addiction. Women meeting the criteria for food addiction were 6 units of BMI heavier in 2009 than women not meeting the food addiction criteria;

almost two thirds of women with food addiction were obese (body mass index >30 kg/m²) in 2009, compared to a quarter of women without food addiction.

Table 2 shows risk ratios (RRs) and 95% confidence intervals (CIs) for food addiction as a function of physical and sexual child abuse severity. We saw a dose-response relationship between physical abuse severity and food addiction risk, with confounder-adjusted RRs of 1.24 (95% CI: 1.14, 1.34), 1.39 (95% CI: 1.30, 1.49), and 1.92 (95% CI: 1.76, 2.09) for mild, moderate, and severe physical abuse, compared to no physical abuse. The relationship of sexual abuse severity to food addiction was similar (Table 3), with an RR for the most severe category of sexual abuse, repeated forced sex, of 1.87 (95% CI: 1.69, 2.05).

Combined exposure to both types of abuse imparted greater risk for food addiction than either type alone. For example, compared to women with neither physical nor sexual abuse histories, the RR associated with severe physical abuse when sexual abuse was also present was 2.40 (95% CI: 2.16, 2.67; Table 2). Likewise, women with repeated experiences of forced sex in addition to a history of physical abuse had an RR for food addiction of 2.32 (95% CI: 2.07, 2.59; Table 3). There was no evidence of a multiplicative interaction (Wald test p -value=0.68). A test of additive interaction between any physical abuse and any sexual abuse approached statistical significance ($p=0.06$).

We estimated similar effects of abuse occurring in childhood only and abuse occurring in adolescence only (Table 4), with RRs of 1.21 (95% CI: 1.12, 1.30) and 1.29 (95% CI: 1.13, 1.47) for physical abuse and comparable RRs for sexual abuse. Longer duration of abuse conferred a greater food addiction risk: when experienced in both childhood and adolescence, physical abuse was associated with an RR of 1.61 (95% CI: 1.51, 1.71) and sexual abuse was associated with an RR of 1.79 (95% CI: 1.65, 1.94).

The age-adjusted risks of food addiction by cross-classifications of child physical and sexual abuse severity ranged from 6.1% among women with no history of physical or sexual abuse to 16.1% among women with a history of both severe physical and severe sexual abuse (Figure 1).

Discussion

In this study, we found dose-response associations between physical and sexual child abuse severity and the likelihood of adult food addiction. Combined experiences of physical and sexual abuse conferred the greatest food addiction risks, with RRs approaching 2.5. Likewise, abuse that occurred in both childhood and adolescence was associated with greater food addiction risk than abuse in a single time period.

No previous study has examined child abuse and food addiction. A relatively large number of cross-sectional studies suggest a relationship between child abuse and both anorexia nervosa and bulimia nervosa (33–35). A small number of studies provide suggestive evidence for an association between child abuse and binge eating disorder (36–37), which captures a distinct but related (38) uncontrolled eating phenotype that affects an estimated 2–3% of the US population (39). In general, studies of child abuse and eating behaviors have relied on small sample sizes, and have been unable to distinguish different types of eating

disorder outcomes; few have examined eating behaviors prevalent enough to contribute importantly to obesity rates. One exception is an examination of childhood abuse and “using food in response to stress” among 1650 adult respondents in the National Survey of Midlife in the US. The authors reported that childhood exposure to frequent physical and psychological abuse was associated with using food in response to stress, which partially explained a 40% increase in obesity incidence in adults reporting child abuse (7). The use of food in response to stress was ascertained with two questionnaire items and was associated with a two-fold increase in obesity prevalence.

Our large cohort allowed us to conduct in-depth examinations of the associations between the type and timing of child abuse and food addiction, a measure of uncontrolled eating reported by 8% of our sample. Nearly two thirds of women meeting the food addiction criteria were obese in 2009, compared to a quarter of women without food addiction, suggesting that food addiction may contribute significantly to obesity rates. The NHSII has rich data on possible confounders, including childhood socioeconomic status and family history of depression, allowing us to adjust for common causes of abuse and eating behaviors that are frequently overlooked.

Despite these strengths, our study has several important limitations. First, we were unable to date the onset of food addiction symptoms, and were therefore unable to establish with certainty that child abuse occurred prior to food addiction. While we believe that a mechanism from child abuse to food addiction is more plausible than one from food addiction to child abuse, this study would have been strengthened by data on timing of food addiction symptoms, which would have allowed us to identify periods of vulnerability to food addiction. Second, the degree to which food addiction represents a valid eating disorder phenotype—and in particular the extent to which the food addiction construct reflects a physical dependency—remains in question; the potential application of these study results will depend in part on whether food addiction is ultimately seen as physical or behavioral in nature and whether effective treatments are identified. Third, the NHSII is comprised primarily of white women, and thus our findings may not be generalizable to the US population as a whole. Future studies in more diverse population would enrich our understanding of eating behavior sequelae of child abuse. Finally, as with many studies of child abuse, we relied on women’s self-report of child abuse, which could not be validated. Because child abuse goes underreported (40), it is unclear what gold standard should be used to validate self-reports of child abuse. Using reported and substantiated cases of child abuse would identify only the most severe cases of abuse and would miss the majority of the exposed population. While we could not validate self-reports of child abuse, the prevalence in our study is very similar to the child abuse prevalence self-reported on national surveys (1–2).

Our finding that child abuse victimization is associated with food addiction adds to accumulating evidence of the importance of stress in the etiology of some obesity phenotypes (12), and may help to inform the development of weight-loss regimens for women with abuse histories. Our study also contributes to a growing body of literature documenting widespread and long-lasting mental and physical health repercussions of child

abuse, which help to clarify the true societal costs of child maltreatment and lend urgency to abuse prevention efforts.

The epidemic prevalence of obesity and its toll on health call for focused efforts to understand widespread obesity risk factors that may be modified to improve public health. A better understanding of the mechanisms by which child abuse, experienced by over a third of girls (1–2), influences weight gain is likely to be important in addressing obesity risk in women. Our study suggests that uncontrolled eating in response to distress may be one important element of this pathway. Future work should further articulate the pathways from abuse to weight gain, to identify critical periods of vulnerability and targets for intervention that can inform prevention and treatment efforts.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgement

Funding: This project was supported by grants RO1HL081557 and RO1HL064108 from the National Institutes of Health.

References

1. Tjaden, P.; Thoennes, N. Prevalence, incidence, and consequences of violence against women: findings from the National Violence Against Women Survey. Washington, DC: US Department of Justice, Office of Justice Programs; 2000.
2. Black, MC.; Basile, KC.; Breiding, MJ., et al. The National Intimate Partner and Sexual Violence Survey (NISVS): 2010 Summary Report. Atlanta, GA: National Center for Injury Prevention and Control, Centers for Disease Control and Prevention; 2011.
3. Lissau I, Sorensen TI. Parental neglect during childhood and increased risk of obesity in young adulthood. *Lancet*. 1994; 343(8893):324–327. [PubMed: 7905145]
4. Noll JG, Zeller MH, Trickett PK, et al. Obesity risk for female victims of childhood sexual abuse: a prospective study. *Pediatrics*. 2007; 120(1):e61–e67. [PubMed: 17606550]
5. Mamun AA, Lawlor DA, O'Callaghan MJ, et al. Does childhood sexual abuse predict young adult's BMI? A birth cohort study. *Obesity (Silver Spring)*. 2007; 15(8):2103–2110. [PubMed: 17712129]
6. Thomas C, Hypponen E, Power C. Obesity and type 2 diabetes risk in midadult life: the role of childhood adversity. *Pediatrics*. 2008; 121(5):e1240–e1249. [PubMed: 18450866]
7. Greenfield EA, Marks NF. Violence from parents in childhood and obesity in adulthood: using food in response to stress as a mediator of risk. *Soc Sci Med*. 2009; 68(5):791–798. [PubMed: 19185965]
8. Riley EH, Wright RJ, Jun HJ, et al. Hypertension in adult survivors of child abuse: observations from the Nurses' Health Study II. *J Epidemiol Community Health*. 2010; 64(5):413–418. [PubMed: 20445210]
9. Rich-Edwards JW, Spiegelman D, Lividoti Hibert EN, et al. Abuse in childhood and adolescence as a predictor of type 2 diabetes in adult women. *Am J Prev Med*. 2010; 39(6):529–536. [PubMed: 21084073]
10. Rich-Edwards JW, Mason S, Rexrode K, et al. Physical and Sexual Abuse in Childhood as Predictors of Early Onset Cardiovascular Events in Women. *Circulation*. 2012; 126(8):920–927. [PubMed: 22787111]
11. Rowland NE, Antelman SM. Stress-induced hyperphagia and obesity in rats: a possible model for understanding human obesity. *Science*. 1976; 191(4224):310–312. [PubMed: 1246617]
12. Dallman MF, Pecoraro N, Akana SF, et al. Chronic stress and obesity: a new view of "comfort food". *Proc Natl Acad Sci U S A*. 2003; 100(20):11696–11701. [PubMed: 12975524]

13. Epel E, Lapidus R, McEwen B, et al. Stress may add bite to appetite in women: a laboratory study of stress-induced cortisol and eating behavior. *Psychoneuroendocrinology*. 2001; 26(1):37–49. [PubMed: 11070333]
14. Dallman MF, la Fleur SE, Pecoraro NC, et al. Minireview: glucocorticoids--food intake, abdominal obesity, and wealthy nations in 2004. *Endocrinology*. 2004; 145(6):2633–2638. [PubMed: 15044359]
15. Freedman MR, Horwitz BA, Stern JS. Effect of adrenalectomy and glucocorticoid replacement on development of obesity. *Am J Physiol*. 1986; 250(4 Pt 2):R595–R607. [PubMed: 3515971]
16. Tataranni PA, Larson DE, Snitker S, et al. Effects of glucocorticoids on energy metabolism and food intake in humans. *Am J Physiol*. 1996; 271(2 Pt 1):E317–E325. [PubMed: 8770026]
17. Cota D, Tschoep MH, Horvath TL, et al. Cannabinoids, opioids and eating behavior: the molecular face of hedonism? *Brain Res Rev*. 2006; 51(1):85–107. [PubMed: 16364446]
18. Ulrich-Lai YM, Christiansen AM, Ostrander MM, et al. Pleasurable behaviors reduce stress via brain reward pathways. *Proc Natl Acad Sci U S A*. 2010; 107(47):20529–20534. [PubMed: 21059919]
19. Adam TC, Epel ES. Stress, eating and the reward system. *Physiol Behav*. 2007; 91(4):449–458. [PubMed: 17543357]
20. Goeders NE. Stress and cocaine addiction. *J Pharmacol Exp Ther*. 2002; 301(3):785–789. [PubMed: 12023504]
21. Johnson PM, Kenny PJ. Dopamine D2 receptors in addiction-like reward dysfunction and compulsive eating in obese rats. *Nat Neurosci*. 2010; 13(5):635–641. [PubMed: 20348917]
22. Volkow ND, Wang GJ, Tomasi D, et al. Obesity and addiction: neurobiological overlaps. *Obes Rev*. 2013; 14(1):2–18. [PubMed: 23016694]
23. Gearhardt AN, Yokum S, Orr PT, et al. Neural correlates of food addiction. *Arch Gen Psychiatry*. 2011; 68(8):808–816. [PubMed: 21464344]
24. Stice E, Spoor S, Bohon C, et al. Relation of reward from food intake and anticipated food intake to obesity: a functional magnetic resonance imaging study. *J Abnorm Psychol*. 2008; 117(4):924–935. [PubMed: 19025237]
25. Gearhardt AN, Grilo CM, DiLeone RJ, et al. Can food be addictive? Public health and policy implications. *Addiction*. 2010; 106:1208–1212. [PubMed: 21635588]
26. Davis C, Curtis C, Levitan RD, et al. Evidence that 'food addiction' is a valid phenotype of obesity. *Appetite*. 2011; 57(3):711–717. [PubMed: 21907742]
27. Gearhardt AN, Corbin WR, Brownell KD. Preliminary validation of the Yale Food Addiction Scale. *Appetite*. 2009; 52(2):430–436. [PubMed: 19121351]
28. Straus, MA.; Gelles, RJ. *Physical Violence in American Families: Risk Factors and Adaptations to Violence in 8,145 Families*. New Brunswick: Transaction Publishers; 1990.
29. Moore DW, Gallup DH, Schussel R. *Disciplining Children in America: A Gallup Poll Report*. The Gallup Organization. 1995
30. Flint AJ, Gearhardt AN, Corbin WR, et al. A Food Addiction Scale Measurement in Two Cohorts of Middle-Aged and Older Women. Under review.
31. Stunkard, AJ.; Sorensen, T.; Schulsinger, F. *The Genetics of Neurological and Psychiatric Disorders*. New York: Raven Press; 1983.
32. Zou G. A modified poisson regression approach to prospective studies with binary data. *Am J Epidemiol*. 2004; 159(7):702–706. [PubMed: 15033648]
33. Fischer S, Stojek M, Hartzell E. Effects of multiple forms of childhood abuse and adult sexual assault on current eating disorder symptoms. *Eat Behav*. 2010; 11(3):190–192. [PubMed: 20434068]
34. Sanci L, Coffey C, Olsson C, et al. Childhood sexual abuse and eating disorders in females: findings from the Victorian Adolescent Health Cohort Study. *Arch Pediatr Adolesc Med*. 2008; 162(3):261–267. [PubMed: 18316664]
35. Gustafson TB, Sarwer DB. Childhood sexual abuse and obesity. *Obes Rev*. 2004; 5(3):129–135. [PubMed: 15245381]

36. Rohde P, Ichikawa L, Simon GE, et al. Associations of child sexual and physical abuse with obesity and depression in middle-aged women. *Child Abuse Negl.* 2008; 32:878–887. [PubMed: 18945487]
37. Striegel-Moore RH, Dohm FA, Pike KM, et al. Abuse, bullying, and discrimination as risk factors for binge eating disorder. *Am J Psychiatry.* 2002; 159(11):1902–1907. [PubMed: 12411226]
38. Gearhardt AN, White MA, Masheb RM, et al. An examination of the food addiction construct in obese patients with binge eating disorder. *Int J Eat Disord.* 2012; 45(5):657–663. [PubMed: 22684991]
39. Grilo, CM. Binge Eating Disorder. In: Fairburn, CG.; Brownell, KD., editors. *Eating Disorders and Obesity.* New York: The Guilford Press; 2002. p. 180
40. Hampton RL, Newberger EH. Child abuse incidence and reporting by hospitals: significance of severity, class, and race. *Am J Public Health.* 1985; 75(1):56–60. [PubMed: 3966600]

What is already known about this subject

- Women with a history of child abuse victimization appear to be at increased risk of obesity in adulthood, but the mechanisms are not known.
- Child abuse is associated with the development of rare eating disorders such as anorexia nervosa and bulimia nervosa, but less is known about more prevalent obesity-related eating behaviors.
- Animal studies suggest that chronic stress may provoke increased consumption of high-calorie food, possibly leading to “food addiction.”

What this study adds

- Food addiction is relatively common in this population, with 8% of the sample meeting food addiction criteria.
- Women who meet the criteria for food addiction are on average 6 units of BMI heavier than women without food addiction.
- Severe physical and sexual abuse victimization in childhood are both associated with 90% increases in adult food addiction risks in this population.

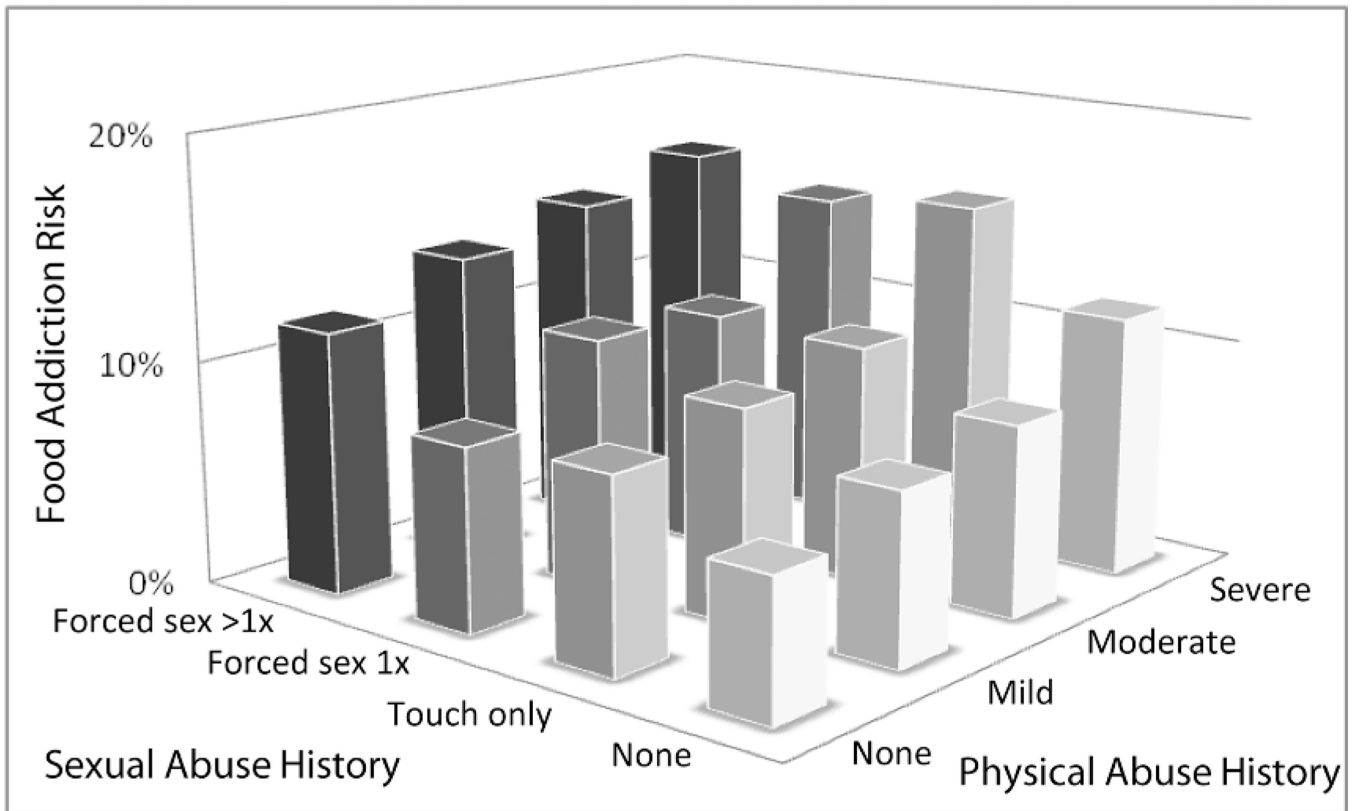


Figure 1. Age-adjusted food addiction risk by severity of physical and sexual child abuse history: Nurses' Health Study II, 2009.

Table 1
Age-standardized covariate distributions across physical and sexual child abuse exposure categories: Nurses' Health Study 2.

Variable	Child and/or adolescent physical abuse				Child and/or adolescent sexual abuse			
	None (n=26,676)	Mild (n=10,593)	Moderate (n=15,024)	Severe (n=4,852)	None (n=37,936)	Touch only (n=12,731)	Forced sex once (n=3,290)	Forced sex >once (n=2,990)
	Mean(SD)	Mean(SD)	Mean(SD)	Mean(SD)	Mean(SD)	Mean(SD)	Mean(SD)	Mean(SD)
<i>Continuous:</i>								
Age in 1989*	35.1(4.7)	35.1(4.6)	35.2(4.5)	35.6(4.5)	35.1(4.7)	35.5(4.6)	35.2(4.5)	35.4(4.5)
Mother's education	12.5(2.0)	12.4(2.0)	12.2(2.0)	12.1(2.0)	12.4(1.9)	12.2(2.0)	12.1(2.0)	12.1(2.1)
Father's education	12.6(2.5)	12.5(2.4)	12.3(2.5)	12.2(2.5)	12.6(2.4)	12.3(2.5)	12.3(2.5)	12.1(2.5)
Somatogram at age 5	2.5(1.2)	2.5(1.2)	2.5(1.2)	2.5(1.3)	2.5(1.2)	2.5(1.2)	2.5(1.3)	2.6(1.3)
<i>Categorical:</i>	%	%	%	%	%	%	%	%
Sexual abuse								
None	74.2	66.2	60.9	43.5	--	--	--	--
Touch only	19.5	23.4	26.0	24.6	--	--	--	--
Forced sex once	3.9	6.4	7.2	10.7	--	--	--	--
Forced sex >once	2.4	4.0	6.0	21.3	--	--	--	--
Physical abuse								
None	--	--	--	--	52.1	40.7	31.2	21.7
Mild	--	--	--	--	18.4	19.4	20.5	14.0
Moderate	--	--	--	--	24.0	30.6	32.7	29.9
Severe	--	--	--	--	5.5	9.3	15.6	34.3
Race								
African-American	0.7	0.7	1.9	0.9	0.8	1.4	1.2	1.7
Latina	0.9	1.2	1.7	1.7	0.9	1.8	1.6	2.0
Asian	1.2	1.0	1.7	1.7	1.3	1.6	1.2	1.2
Caucasian	94.3	94.4	91.9	92.4	94.0	92.6	93.1	92.0
Other	1.5	1.5	1.5	2.0	1.6	1.4	1.7	1.7
Mother in professional occupation	11.8	11.6	10.3	10.1	11.4	10.9	10.2	11.5

Variable	Child and/or adolescent physical abuse				Child and/or adolescent sexual abuse			
	None (n=26,676)	Mild (n=10,593)	Moderate (n=15,024)	Severe (n=4,852)	None (n=37,936)	Touch only (n=12,731)	Forced sex once (n=3,290)	Forced sex >once (n=2,990)
Father in professional occupation	29.9	27.9	24.1	22.0	28.8	25.1	23.9	21.6
Parents owned home	52.9	50.3	48.4	44.9	51.5	49.4	48.1	47.7
Mother or father with history of depression	11.5	14.9	16.2	24.5	12.9	16.0	19.6	21.4

* Value is not age-standardized

Risk ratios for food addiction by physical child abuse severity in all women and by exposure to sexual child abuse: Nurses' Health Study 2.

Table 2

Physical abuse Severity [‡]	All women				By sexual abuse exposure			
	Age-adjusted		Confounder-adjusted*		No sexual abuse exposure		With sexual abuse exposure [†]	
	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	N (cases)	RR* (95% CI)	N (cases)	RR* (95% CI)
None	1.00 --	1.00 --	1.00 --	1.00 --	19,732 (1,156)	1.00 --	6,849 (581)	1.41 (1.28, 1.55)
Mild	1.26 (1.16, 1.36)	1.24 (1.14, 1.34)	1.23 (1.11, 1.35)	1.66 (1.48, 1.86)	6,982 (511)	1.23 (1.11, 1.35)	3,562 (359)	1.66 (1.48, 1.86)
Moderate	1.42 (1.33, 1.52)	1.39 (1.30, 1.49)	1.39 (1.27, 1.52)	1.79 (1.63, 1.96)	9,095 (751)	1.39 (1.27, 1.52)	5,858 (642)	1.79 (1.63, 1.96)
Severe	2.06 (1.90, 2.25)	1.92 (1.76, 2.09)	1.82 (1.59, 2.08)	2.40 (2.16, 2.67)	2,088 (236)	1.82 (1.59, 2.08)	2,725 (418)	2.40 (2.16, 2.67)

* Adjusted for age in 1989, race, mother's educational attainment, father's educational attainment, mother in professional occupation, father in professional occupation, parental history of depression.

[†] Any sexual abuse, including sexual touching only, forced sexual activity once, and forced sexual activity more than once.

[‡] Mild physical abuse was defined as being pushed, grabbed, or shoved at any frequency or being kicked, bitten, or punched once or hit with something once; moderate physical abuse was defined as being hit with something more than once or physically attacked once, and severe physical abuse was defined as being kicked, bitten, punched, or physically attacked more than once or ever choked or burned.

Table 3

Risk ratios for food addiction by sexual child abuse severity in all women and stratified by exposure to physical child abuse: Nurses' Health Study 2.

Sexual abuse severity	All women						By physical abuse exposure					
	Age-adjusted			Confounder-adjusted*			No physical abuse exposure			With physical abuse exposure [†]		
	N (cases)	RR (95% CI)	RR (95% CI)	N (cases)	RR (95% CI)	RR (95% CI)	N (cases)	RR* (95% CI)	RR* (95% CI)	N (cases)	RR* (95% CI)	
	37,936			19,732			18,165					
None	(2,658)	1.00	--	(1,156)	1.00	--	(1,498)	1.38	(1.28, 1.48)			
Touch	12,731			5,179			7,538					
only	(1,237)	1.39	(1.30, 1.48)	(427)	1.38	(1.24, 1.44)	(809)	1.76	(1.61, 1.92)			
Forced	3,290			1,025			2,263					
sex once	(341)	1.48	(1.33, 1.64)	(80)	1.28	(1.03, 1.59)	(261)	1.86	(1.64, 2.11)			
Forced	2,990			645			2,344					
sex >once	(423)	2.02	(1.83, 2.22)	(74)	1.85	(1.69, 2.05)	(349)	2.32	(2.07, 2.59)			

* Adjusted for age in 1989, race, mother's educational attainment, father's educational attainment, mother in professional occupation, father in professional occupation, parental history of depression.

[†] Any physical abuse, including mild, moderate, or severe.

Table 4 Risk ratios for food addiction by timing of child and adolescent physical and sexual abuse: Nurses' Health Study 2.

Abuse timing	Physical abuse				Sexual abuse			
	N (cases)	Age-adjusted		N (cases)	Age-adjusted		Confounder- adjusted*	
		RR	(95% CI)		RR	(95% CI)		RR
None	26,676 (1,749)	1.00	--	37,936 (2,658)	1.00	--	1.00	--
Childhood (age 0–10) only	12,051 (971)	1.23	(1.14, 1.32)	6,994 (662)	1.21	(1.12, 1.30)	1.35	(1.25, 1.47)
Adolescence (age 11–7) only	2,598 (227)	1.33	(1.16, 1.52)	7,123 (678)	1.29	(1.13, 1.47)	1.36	(1.25, 1.47)
Childhood and adolescence	15,820 (1,734)	1.67	(1.57, 1.78)	4,894 (661)	1.61	(1.51, 1.71)	1.93	(1.78, 2.09)

* Adjusted for age in 1989, race, mother's educational attainment, father's educational attainment, mother in professional occupation, father in professional occupation, parental home ownership, parental history of depression