

Original Investigation

Posttraumatic Stress Disorder Symptoms and Food Addiction in Women by Timing and Type of Trauma Exposure

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IMPORTANCE Posttraumatic stress disorder (PTSD) appears to increase obesity risk but the pathways by which PTSD leads to weight gain are not known. Identification of the links between PTSD and obesogenic eating behaviors is necessary to clarify this pathway and inform development of obesity prevention strategies in PTSD-affected populations.

OBJECTIVE To determine whether women with PTSD symptoms are more likely to report food addiction, a measure of perceived dependence on food, than women without PTSD symptoms. Also, to determine whether age at PTSD symptom onset and type of trauma influence the PTSD–food addiction association.


DESIGN, SETTING, AND PARTICIPANTS Cross-sectional analysis of 49 408 participants in the Nurses' Health Study II, a cohort comprising women nurses who were aged 25 to 42 years at the 1989 recruitment from 14 US states.

MAIN OUTCOMES AND MEASURES The Nurses' Health Study II ascertained lifetime trauma exposure and PTSD symptoms in 2008 and current food addiction in 2009. Food addiction was defined as 3 or more clinically significant symptoms on a modified version of the Yale Food Addiction Scale. Confounder-adjusted prevalence ratios and 95% CIs were estimated using modified Poisson regression.

RESULTS Approximately 80% of the study sample reported some type of trauma exposure, with 66% of the trauma-exposed participants reporting at least 1 lifetime PTSD symptom. Eight percent of the cohort met the criteria for food addiction. The prevalence of food addiction increased with the number of lifetime PTSD symptoms, and women with the greatest number of PTSD symptoms (6-7 symptoms) had more than twice the prevalence of food addiction as women with neither PTSD symptoms nor trauma histories (prevalence ratio, 2.68; 95% CI, 2.41-2.97). Symptoms of PTSD were more strongly related to food addiction when symptom onset occurred at an earlier age. The PTSD–food addiction association did not differ substantially by trauma type.

CONCLUSIONS AND RELEVANCE Symptoms of PTSD were associated with increased food addiction prevalence in this cohort of women. Strategies to reduce obesity associated with PTSD may require psychological and behavioral interventions that address dependence on food and/or use of food to cope with distress.

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Posttraumatic stress disorder (PTSD) is a potentially severe psychiatric condition affecting both military and civilian populations.¹⁻³ It is characterized by a set of 3 symptom clusters—reexperiencing, arousal, and avoidance/numbing⁴—that arise in response to a potentially traumatic event. In the US civilian population, lifetime PTSD occurrence is 6.8%, with a higher rate (approximately 12%) among women.^{2,5} In addition to causing acute psychological suffering, PTSD may have long-term effects on physical health and well-being. In particular, a growing body of evidence suggests that PTSD is an important risk factor for obesity and obesity-related diseases including type 2 diabetes mellitus and cardiovascular disease.⁶⁻¹² Posttraumatic stress disorder may be linked to cardiometabolic illness in part through direct biological mechanisms, such as inflammatory pathways,^{13,14} but it is likely that behavioral factors also play an important role, and these remain to be clarified.

A cross-sectional study by Hirth et al¹⁵ found PTSD symptoms to be associated with increased consumption of fast food and soda in young women. As noted by Brewerton¹⁶ in a commentary, these findings are suggestive of the use of food to self-medicate and are consistent with animal models indicating that stress may provoke increased consumption of highly palatable (high-fat/high-sugar) food, leading to weight gain.¹⁷⁻¹⁹ Neurological studies indicate that ingestion of high-fat and high-sugar foods may blunt feelings of distress by triggering dopaminergic reward responses,^{20,21} and animal studies suggest that stress-related consumption of palatable food can ultimately become addiction-like in nature.²²⁻²⁴ This growing body of animal and clinical literature has led to public health interest in the potential role of food addiction in the obesity epidemic.²⁵ In previous work, we found that childhood abuse is strongly associated with adult reports of food addiction and that women reporting food addiction were an average of 6 units of body mass index (calculated as weight in kilograms divided by height in meters squared) heavier than women without food addiction.²⁶ Food addiction is not currently established as a psychiatric diagnosis; however, the concept may nonetheless be helpful for identifying the reliance on food to cope with psychological distress, one plausible pathway from PTSD to obesity.

Understanding the links between PTSD and obesogenic eating behaviors is crucial for development of effective strategies to prevent and treat obesity in PTSD-affected populations. In this analysis, we examined the association between PTSD symptoms and food addiction among 49 408 participants in the Nurses' Health Study II.

Methods

Data Sources

The Nurses' Health Study II follows up 116 430 women registered nurses recruited at ages 25 to 42 years in 1989. Biennial questionnaires are used to gather sociodemographic, behavioral, and medical data. Participants indicated their consent by returning completed questionnaires. In 2009, the biennial questionnaire included a modified version of the Yale Food Ad-

dition Scale (mYFAS),²⁷ which ascertains the extent to which participants' eating behavior can be characterized as a dependency. In 2008, a supplemental PTSD Questionnaire assessing trauma exposure and PTSD symptoms was sent to 60 894 Nurses' Health Study II participants who had responded to an earlier supplemental questionnaire on exposure to violence. The 2008 PTSD Questionnaire was returned by 54 700 women (a response rate of 90%). Of these, 53 616 responded to the 2009 biennial questionnaire, with 49 408 having complete food addiction and PTSD information. This study was approved by the institutional review board at Brigham and Women's Hospital, Boston, Massachusetts.

Variables and Variable Definitions

Exposures

The 2008 PTSD Questionnaire used a modified version of the Brief Trauma Questionnaire^{28,29} to ascertain women's experiences of 16 traumatic events including violent death of a loved one, childhood physical abuse, or "a serious traumatic event not already covered." Participants were further asked which event they considered to be the worst³⁰ and their age when it first occurred. Suggestive PTSD symptoms (hereafter referred to as *PTSD symptoms*) were queried with respect to the worst traumatic event using the 7-item Brief Screening Scale for *DSM-IV* PTSD.³¹ Participants were asked the age at which they first started experiencing the PTSD symptoms. More than half of individuals with PTSD do not seek treatment,³² and we therefore focus on PTSD symptoms rather than diagnosis to avoid missing most cases.

Breslau et al³¹ recommended using a cutoff of 4 or more symptoms on this screening scale for identifying PTSD cases based on trade-offs between sensitivity and specificity. Using a cutoff of 4 or more symptoms yields a positive predictive value of 71% compared with diagnostic interview, while a cutoff of 6 or more symptoms yields a positive predictive value of 87%. To avoid overemphasizing a single cut point, we classified participants into 5 exposure categories: (1) no trauma exposure, (2) exposed to a traumatic event but reported no PTSD symptoms, (3) trauma with 1 to 3 PTSD symptoms, (4) trauma with 4 to 5 PTSD symptoms, and (5) trauma with 6 to 7 PTSD symptoms. Symptoms of PTSD were queried with respect to a specific traumatic event; by definition, women without trauma were classified as having no PTSD symptoms.

Outcome

Food addiction was assessed in 2009 using the mYFAS,²⁷ which is modeled after measures of drug and alcohol addiction. The mYFAS defines food addiction as 3 or more of the following symptoms plus clinically significant impairment or distress: (1) eating when no longer hungry 4 or more times per week, (2) worrying about cutting down on certain foods 4 or more times per week, (3) feeling sluggish or fatigued from overeating 2 or more times per week, (4) experiencing negative feelings from overeating that interfere with other activities 2 or more times per week, (5) having physical withdrawal symptoms when cutting down on certain foods 2 or more times per week, (6) continuing to consume the same amount of food despite significant emotional or physical problems

due to overeating at any frequency, and (7) 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 (1) experiencing significant distress related to eating behavior 2 or more times per week or (2) experiencing a decrease in the ability to function owing to issues related to food 2 or more times per week. For further details on the adaptation of the original YFAS, see the eAppendix in the Supplement.

Covariates

We included the following potential confounders in adjusted models because they are plausible predictors of both PTSD and food addiction³³: age at Nurses' Health Study II baseline (continuous with a squared term), race/ethnicity (indicators for African American, Asian, Hispanic, and other, with non-Hispanic white as the referent), mother 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 years (continuous; the participant could choose 1 of 9 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 5 years³⁴), and parental lifetime history of depression (indicator if either parent had depression history) assessed in 2005.

Data Analysis

We used modified Poisson regression (Poisson regression with robust standard errors³⁵) to estimate prevalence ratios (PRs) for food addiction as a function of trauma exposure and number of PTSD symptoms. Analyses included women who responded to both the 2008 Stress Questionnaire, on which abuse was ascertained, and the 2009 biennial questionnaire, on which food addiction was assessed ($n = 53\ 616$). We excluded women who left 3 or more food addiction symptom questions blank ($n = 2270$), those who were missing information on clinical significance of symptoms ($n = 291$), and those whose trauma-PTSD status could not be ascertained owing to large amounts of missing data ($n = 1607$) or inconsistency in their reports ($n = 40$), leaving 49 408 women (92% of all respondents to the PTSD and 2009 biennial questionnaires).

We ran 3 main analyses. First, we examined the cross-sectional association between lifetime trauma and PTSD symptoms, reported in 2008, and current food addiction, assessed in 2009. We ran an age-adjusted model and a model adjusted for additional potential confounders.

Because the 2009 questionnaire did not ask about the timing of food addiction symptom onset, the temporal order of PTSD and food addiction is unknown; therefore, the results of these analyses are presented as prevalence rather than risk ratios.

Second, to gain insight into the temporal order of PTSD and food addiction, we examined both the number of PTSD symptoms and the timing of their onset. We were particularly interested in whether onset of PTSD in childhood would be associated with food addiction because eating disorders typically

manifest in adolescence or later,³⁶ and thus an association between preadolescent PTSD and food addiction would support a temporal sequence from PTSD to food addiction. To conduct this analysis, we categorized women into 19 cross-classifications based on symptom level (none, 1-3 symptoms, 4-5 symptoms, and 6-7 symptoms) and age at onset in 6 intervals (<10, 10-19, 20-29, 30-39, 40-49, and ≥ 50 years). We report food addiction PRs for each category of symptom level and age at onset, with no symptoms at any time as the referent. Among women with at least 1 PTSD symptom, we conducted a test for trend of age at symptom onset. We also tested the interaction between categorical PTSD symptoms and age at onset.

We ran 2 sensitivity analyses related to timing of PTSD onset. Because women were asked to report symptoms in reference to their worst trauma, timing of PTSD symptoms could plausibly be defined using age at worst trauma; therefore, we re-ran our analyses using reported age at worst trauma to time of the onset of PTSD symptoms. In addition, because we were concerned about the plausibility of recall from a very young age, we ran a sensitivity analysis excluding the 136 women reporting symptom onset prior to age 4 years.

For our third analysis, we explored PTSD-food addiction associations by type of trauma. We categorized women based on cross-classifications of the trauma they identified as their worst and the number of PTSD symptoms experienced in response, with no trauma as the reference category. We previously reported strong relationships of childhood and adolescent physical and sexual abuse victimization with food addiction,²⁶ and thus we were particularly interested in examining the influence of these traumas. While childhood physical abuse (physical abuse before the age of 18 years) was included in the list of traumas on the PTSD Questionnaire, childhood sexual abuse was not—rather, women were asked about unwanted sexual contact at any age. For this analysis, because we were interested in childhood sexual abuse, we defined a participant as having PTSD symptoms in response to childhood sexual abuse if she indicated unwanted sexual contact as her worst trauma and reported that her worst trauma occurred prior to the age of 18 years. We modeled food addiction as a function of the cross-classification of 16 trauma types and 3 PTSD symptom levels using modified Poisson regression and adjusting for potential confounders. Approximately 6.5% of trauma-exposed women did not specify which trauma was their worst and were excluded from this analysis.

Results

Of 49 408 women meeting inclusion criteria for our main analyses, 81% reported experiencing at least 1 traumatic event (Table 1). Of these, 34% reported no PTSD symptoms, 39% reported 1 to 3 symptoms, 17% reported 4 to 5 symptoms, and 10% reported 6 to 7 symptoms. On average, women with PTSD symptoms reported that their first symptom occurred at age 30.5 years (SD, 14.7 years). The food addiction prevalence was 8%, varying from 6% among women with no lifetime PTSD symptoms to almost 18% among women with 6 to 7 PTSD

Table 1. Age-Adjusted Characteristics of the Study Sample by Reported Trauma Exposure and Number of Lifetime PTSD Symptoms

Variable	Trauma and No. of Lifetime PTSD Symptoms ^a				
	No Trauma (n = 9445)	Trauma But No Symptoms (n = 13 445)	Trauma and Symptoms		
			1-3 (n = 15 757)	4-5 (n = 6859)	6-7 (n = 3902)
Age at first PTSD symptom, mean (SD), y ^b	NA	NA	30.9 (14.3)	30.5 (14.9)	28.4 (15.4)
Food addiction cases, (prevalence in exposure category), No. (%)	575 (6.1)	785 (5.9)	1210 (7.7)	774 (11.3)	698 (17.8)
Covariates					
Continuous, mean (SD)					
Age at NHSII baseline, y ^b	35.1 (4.7)	35.3 (4.7)	35.3 (4.6)	35.3 (4.5)	35.3 (4.4)
Educational attainment, y					
Mother's	12.4 (1.9)	12.3 (2.0)	12.4 (2.0)	12.4 (2.0)	12.4 (2.0)
Father's	12.5 (2.4)	12.4 (2.4)	12.5 (2.5)	12.5 (2.5)	12.5 (2.5)
Somatogram at age 5 y ^c	2.5 (1.2)	2.5 (1.2)	2.5 (1.2)	2.6 (1.3)	2.6 (1.3)
Categorical, %					
Race/ethnicity					
Black	0.8	1.1	1.1	0.7	0.6
Latina	0.9	1.2	1.4	1.2	1.1
Asian	1.7	1.3	1.1	0.9	0.9
White	93.4	93.4	94.0	94.7	94.5
Other	1.8	1.6	1.3	1.4	1.7
In professional occupation					
Mother	11.5	11.7	11.3	11.1	11.5
Father	28.0	27.3	28.4	29.0	27.7
Parents owned home	53.0	52.6	50.6	50.5	49.9
Parental history of depression	10.1	11.2	15.4	20.4	25.6

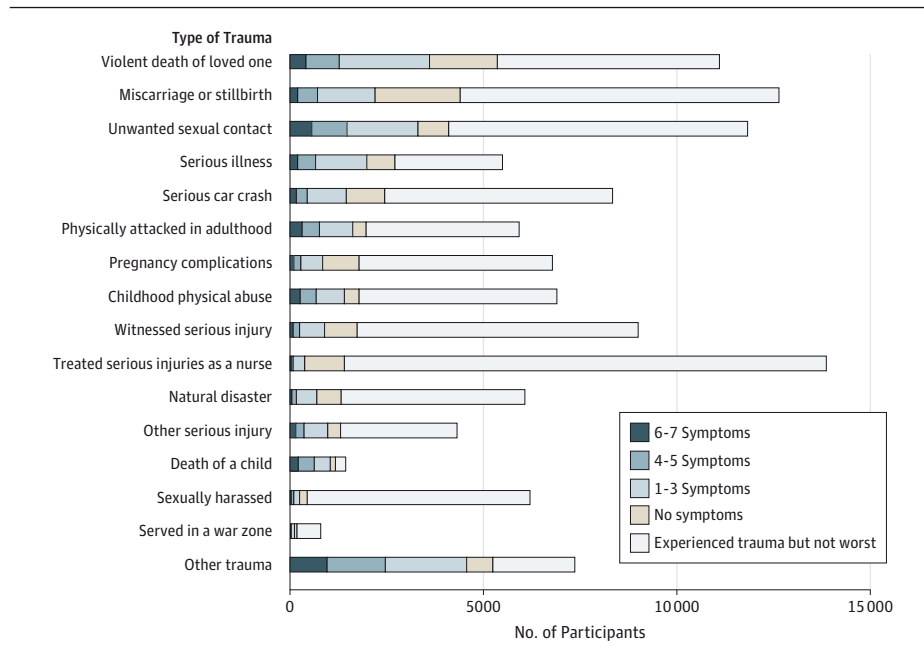
Abbreviations: NA, not applicable; NHSII, Nurses' Health Study II; PTSD, posttraumatic stress disorder.

^a Traumatic symptoms are queried with regard to trauma exposure and thus, by definition, women without trauma could not have traumatic symptoms.

^b Value is not age adjusted.

^c Participants chose the image of a female figure that best approximated their body type at age 5 years, ranging from 1 (very lean) to 9 (obese).

Figure 1. Number of Participants Reporting Each Trauma Type as Their Worst by the Level of Posttraumatic Stress Disorder Symptoms They Reported Experiencing in Response



Traumas are ordered by descending frequency with which they were reported as worst. Participants could choose only 1 worst trauma; thus, participants in 1 worst-trauma category will not be in any other worst-trauma category. In contrast, participants may be represented in the category of experienced trauma but not worst for multiple trauma types.

symptoms. Family depression history, but not socioeconomic and demographic variables, was associated with PTSD symptoms (Table 1).

The violent death of a family member or friend was most frequently cited as the respondent's worst trauma, followed by miscarriage or stillbirth (Figure 1). Child death was most strongly

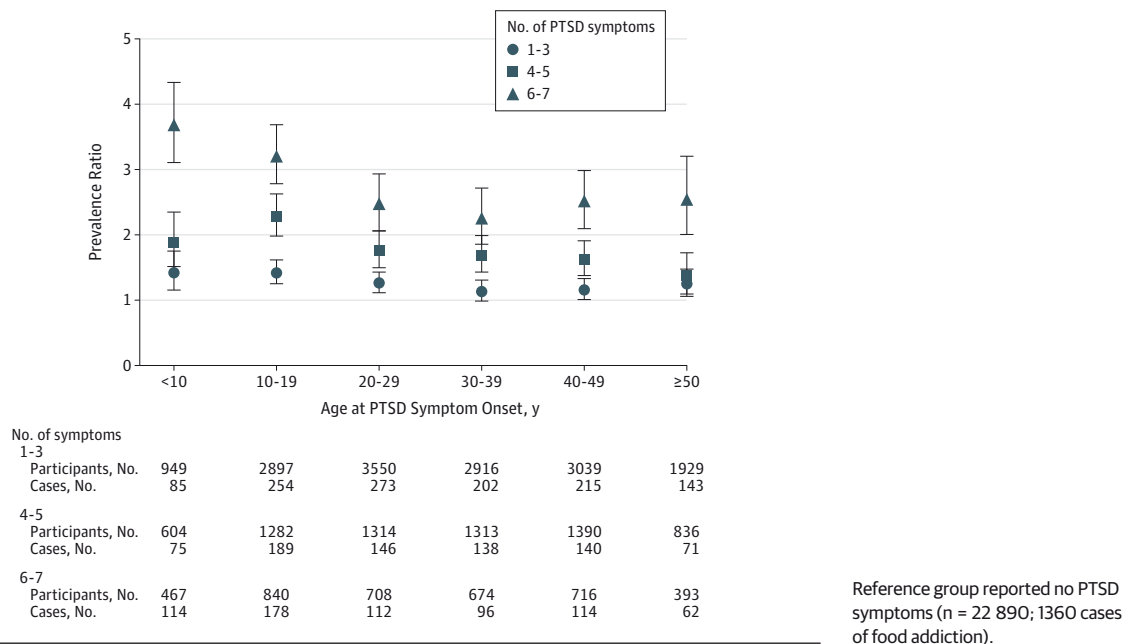
Table 2. Prevalence Ratios for 2009 Food Addiction by Reported Lifetime Number of PTSD Symptoms

Trauma and No. of PTSD Symptoms	Participants (Cases), No.	Prevalence Ratio (95% CI)	
		Age Adjusted	Covariate Adjusted ^a
No trauma	9445 (575)	1 [Reference]	1 [Reference]
Trauma, no symptoms	13 445 (785)	0.96 (0.87-1.07)	0.95 (0.86-1.06)
Trauma and symptoms			
1-3	15 757 (1210)	1.26 (1.14-1.39)	1.22 (1.11-1.34)
4-5	6859 (774)	1.85 (1.67-2.05)	1.73 (1.56-1.92)
6-7	3902 (698)	2.93 (2.64-3.25)	2.68 (2.41-2.97)

Abbreviation: PTSD, posttraumatic stress disorder.

^a Adjusted for age in 1989, race/ethnicity, mother and father's educational attainment, mother and father in professional occupation, parental home ownership, and parental history of depression. Additional adjustment for parental history of diabetes mellitus, early myocardial infarction, and stroke did not have an important impact on the results.

Figure 2. Prevalence Ratio for Food Addiction as a Function of the Number of Reported Posttraumatic Stress Disorder (PTSD) Symptoms by Age at Onset of First Symptom

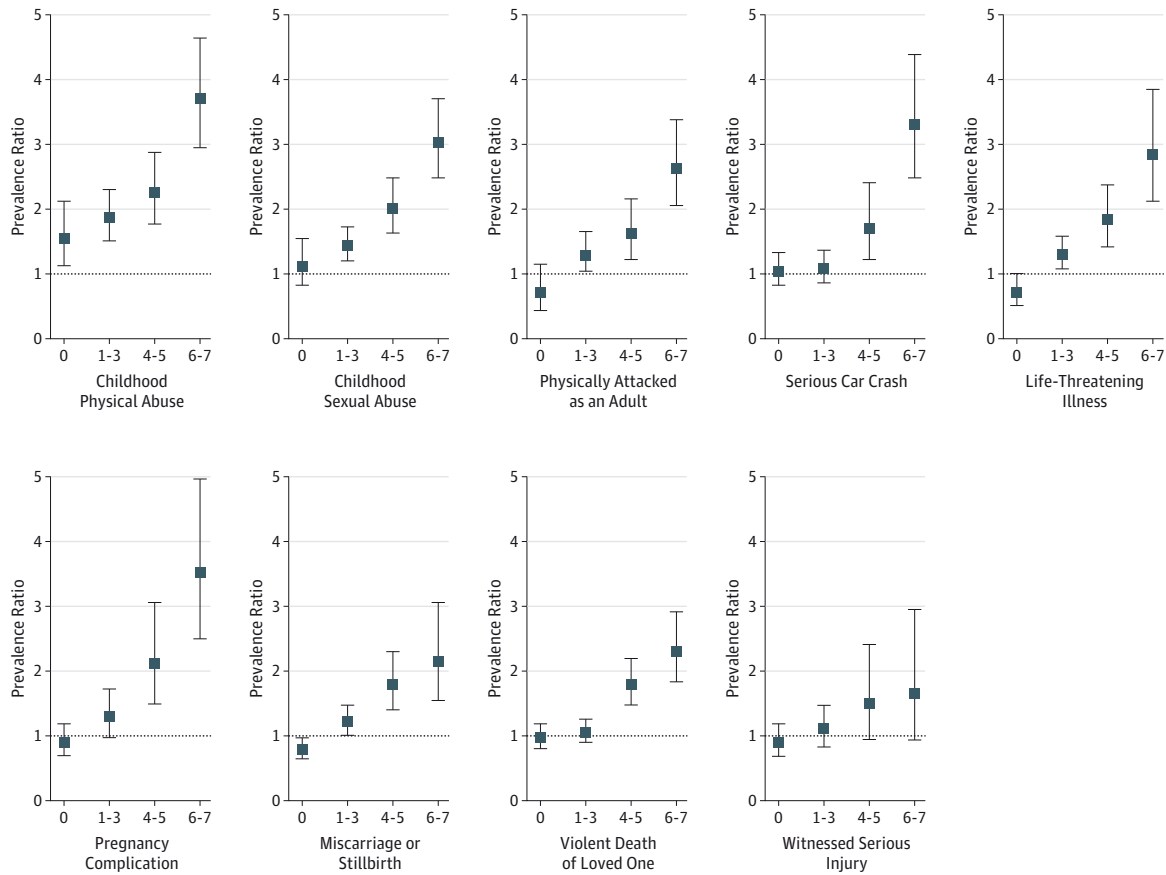


associated with a high level of PTSD symptoms. The most commonly reported trauma experience in this nurse population was treating civilians with traumatic injuries (Figure 1), although this was rarely cited as the respondent's worst trauma.

In age-adjusted analyses, PTSD symptoms were associated with the prevalence of food addiction in a dose-response manner (Table 2), with PRs, relative to no trauma, of 0.96 (95% CI, 0.87-1.07), 1.26 (95% CI, 1.14-1.39), 1.85 (95% CI, 1.67-2.05), and 2.93 (95% CI, 2.64-3.25) for trauma but no PTSD symptoms and 1 to 3, 4 to 5, and 6 to 7 PTSD symptoms, respectively. Covariate adjustment attenuated these modestly, yielding PRs of 0.95 (95% CI, 0.86-1.05), 1.22 (95% CI, 1.11-1.34), 1.73 (95% CI, 1.56-1.92), and 2.68 (95% CI, 2.41-2.97) for trauma but no symptoms and 1 to 3, 4 to 5, and 6 to 7 symptoms, respectively. Inclusion of family history of depression resulted in the greatest attenuation in the estimates, although its impact was still small (resulting in an approximately 3% change in the estimate).

When PTSD symptom onset occurred at younger than 10 years of age, the adjusted PR for food addiction associated with 6 to 7 PTSD symptoms, relative to no PTSD symptoms, was 3.67 (95% CI, 3.10-4.33) (Figure 2); this declined to 2.24 (95% CI, 1.85-2.71) when symptom onset occurred between 30 and 39 years of age. This pattern of diminishing effect sizes with increasing age was less apparent for lower levels of PTSD symptoms. Findings were robust to using reported age at worst trauma to define the age increments and excluding women reporting trauma symptoms prior to age 4 years. Among women with at least 1 PTSD symptom, the age at symptom onset was statistically significantly associated with food addiction (*P* for trend < .001) but the interaction between age at symptom onset and number of PTSD symptoms was not (*P* = .33). This suggested that, among women with at least 1 PTSD symptom, earlier onset predicts a higher baseline prevalence of food addiction but does not influence the dose-response association between additional PTSD symptoms and the prevalence of food addiction.

Figure 3. Prevalence Ratios for Food Addiction as a Function of the Type of Worst Trauma and Number of Posttraumatic Stress Disorder Symptoms Experienced in Response



The figure presents the 9 queried traumas most commonly reported as being the worst. The numbers on the x-axis represent the number of posttraumatic stress disorder symptoms.

The type of trauma reported as the worst did not have a clear influence on the magnitude of the PTSD-food addiction association (Figure 3 and the eTable in the Supplement). Traumatic symptoms in response to physical abuse in childhood showed the strongest associations with food addiction overall (PRs ranged from 1.6; 95% CI, 1.1-2.1 for physical abuse with no PTSD symptoms to 3.7; 95% CI, 3.0-4.7 for physical abuse with 6-7 symptoms). However, confidence intervals were overlapping across various trauma types, providing little statistical evidence for differences in PTSD-food addiction associations, and the overall pattern of associations were fairly similar.

Discussion

In this analysis, we found a dose-response association between the number of lifetime PTSD symptoms and prevalence of food addiction in middle adulthood. Food addiction prevalence appeared to be further elevated when PTSD symptoms occurred earlier in life. To our knowledge, this is the first study to look at the association between PTSD symptoms and

food addiction. Our findings are relevant to ongoing questions regarding the mechanisms behind observed associations between PTSD and obesity,¹² and they provide support for hypotheses suggesting that associations between PTSD and obesity might partly originate in maladaptive coping and use of food to blunt trauma-associated distress. If replicated longitudinally, these results may have implications for both the etiology of obesity and for treatment of individuals with PTSD.

Our findings are consistent with a small body of literature on the comorbidity of PTSD and disordered eating³⁷ including 2 cross-sectional studies documenting an association between PTSD and binge eating disorder,^{10,38} a related but distinct overeating phenotype.³⁹ The value of the food addiction measure is that it explicitly captures feelings of dependence on food and distress as a trigger for eating and is thus more directly in line with hypothesized mechanisms involving eating as a coping strategy.

While we were limited to cross-sectional analyses by lack of data on timing of food addiction onset, we were able to examine the relationship between age at PTSD onset and food addiction. We found PTSD symptoms to be associated with food addiction even when they are experienced prior to adoles-

cence, the typical age at eating disorder onset, consistent with a pathway from PTSD to food addiction. Early-onset PTSD-food addiction associations are also consistent with the idea that childhood is a critical window of exposure during which health insults may have especially profound effects.⁴⁰⁻⁴² However, it is also possible that these results arise from recall bias: women may have difficulty recalling early-life trauma unless it was severe, and self-reports of childhood PTSD may therefore overrepresent the most severe PTSD exposure.

While there was some variation in the PTSD-food addiction association across trauma types, evidence for an influence of trauma type on the association was limited. We found that PTSD symptoms in response to childhood physical abuse had the strongest overall associations with food addiction but these findings may be due to chance, and overall patterns of association between PTSD symptoms and food addiction were otherwise similar.

Several limitations of our study warrant comment. First, we assessed PTSD retrospectively with a screening questionnaire (rather than a diagnostic interview), which likely reduced the accuracy of PTSD classification but enabled the collection of PTSD data from a large cohort. Second, women who reported more than 1 PTSD symptom may not have experienced all of their symptoms in the same age range that they reported symptom onset; therefore, the classification of women by age at PTSD onset is a crude measure of the timing of PTSD and does not completely capture the nuances of PTSD symptoms over the life course. Finally, food addiction is con-

troversial, and the clinical applicability of our findings will depend in part on whether mental health professionals determine that food addiction is a legitimate psychiatric diagnosis and identify effective prevention and treatment strategies for it. However, we believe that the value of the food addiction construct goes beyond its identification of psychiatric illness by capturing a potentially important maladaptive coping behavior that may provide insight into mechanisms linking trauma and PTSD to obesity.

Conclusions

To our knowledge, this study provides the first evidence of an association between PTSD symptoms and food addiction, 2 disorders of emerging concern for obesity risk. Our findings are consistent with the hypothesis that observed links between PTSD and obesity¹² might be partly explained by a tendency to use food to self-medicate traumatic stress symptoms.¹⁶ If replicated longitudinally, these findings suggest that interrupting the pathway from PTSD to obesity may require psychological and behavioral interventions that address dependence on eating to cope with distress. Our study provides additional support for the recommendations by Kubzansky et al¹² for integration of health behavior modification into PTSD treatment and highlights one eating behavior phenotype that might be modified to reduce long-term obesity risk among populations with PTSD.

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Acquisition, analysis, or interpretation of data: All authors.

Drafting of the manuscript: Mason, Roberts, Rich-Edwards.

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REFERENCES

1. Thomas JL, Wilk JE, Riviere LA, McGurk D, Castro CA, Hoge CW. Prevalence of mental health problems and functional impairment among active component and National Guard soldiers 3 and 12 months following combat in Iraq. *Arch Gen Psychiatry*. 2010;67(6):614-623.
2. Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication [published correction appears in *Arch Gen Psychiatry*. 2005;62(7):768]. *Arch Gen Psychiatry*. 2005;62(6):593-602.
3. Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB. Posttraumatic stress disorder in the National Comorbidity Survey. *Arch Gen Psychiatry*. 1995;52(12):1048-1060.
4. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 4th ed, text revision. Washington, DC: American Psychiatric Association; 2000:943.
5. Resnick HS, Kilpatrick DG, Dansky BS, Saunders BE, Best CL. Prevalence of civilian trauma and posttraumatic stress disorder in a representative national sample of women. *J Consult Clin Psychol*. 1993;61(6):984-991.
6. Ahmadi N, Hajsadeghi F, Mirshkarlo HB, Budoff M, Yehuda R, Ebrahimi R. Post-traumatic stress disorder, coronary atherosclerosis, and mortality. *Am J Cardiol*. 2011;108(1):29-33.
7. Boyko EJ, Jacobson IG, Smith B, et al; Millennium Cohort Study Team. Risk of diabetes in US military service members in relation to combat deployment and mental health. *Diabetes Care*. 2010;33(8):1771-1777.
8. Dedert EA, Becker ME, Fuemmeler BF, Braxton LE, Calhoun PS, Beckham JC. Childhood traumatic stress and obesity in women: the intervening effects of PTSD and MDD. *J Trauma Stress*. 2010;23(6):785-63.
9. Dedert EA, Calhoun PS, Watkins LL, Sherwood A, Beckham JC. Posttraumatic stress disorder, cardiovascular, and metabolic disease: a review of the evidence. *Ann Behav Med*. 2010;39(1):61-78.
10. Pagoto SL, Schneider KL, Bodenlos JS, et al. Association of post-traumatic stress disorder and obesity in a nationally representative sample. *Obesity (Silver Spring)*. 2012;20(1):200-205.

11. Roberts AL, Agnew-Blais J, Spiegelman D, et al. Posttraumatic stress disorder and type 2 diabetes incidence in women: an 18-year longitudinal study. *Compr Psychiatry*. 2013;54(8):e34-e35. doi:10.1016/j.comppsych.2013.07.059.
12. Kubzansky LD, Bordoelois P, Jun HJ, et al. The weight of traumatic stress: a prospective study of posttraumatic stress disorder symptoms and weight status in women. *JAMA Psychiatry*. 2014;71(1):44-51.
13. Wieck A, Grassi-Oliveira R, Hartmann do Prado C, Teixeira AL, Bauer ME. Neuroimmunoendocrine interactions in post-traumatic stress disorder: focus on long-term implications of childhood maltreatment. *Neuroimmunomodulation*. 2014;21(2-3):145-151.
14. Rasmusson AM, Schnurr PP, Zukowska Z, Scioli E, Forman DE. Adaptation to extreme stress: post-traumatic stress disorder, neuropeptide Y and metabolic syndrome. *Exp Biol Med (Maywood)*. 2010;235(10):1150-1162.
15. Hirth JM, Rahman M, Berenson AB. The association of posttraumatic stress disorder with fast food and soda consumption and unhealthy weight loss behaviors among young women. *J Womens Health (Larchmt)*. 2011;20(8):1141-1149.
16. Brewerton TD. Posttraumatic stress disorder and disordered eating: food addiction as self-medication. *J Womens Health (Larchmt)*. 2011;20(8):1133-1134.
17. Corwin RL, Avena NM, Boggiano MM. Feeding and reward: perspectives from three rat models of binge eating. *Physiol Behav*. 2011;104(1):87-97.
18. Rowland NE, Antelman SM. Stress-induced hyperphagia and obesity in rats: a possible model for understanding human obesity. *Science*. 1976;191(4224):310-312.
19. 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.
20. Cota D, Tschöp MH, Horvath TL, Levine AS. Cannabinoids, opioids and eating behavior: the molecular face of hedonism? *Brain Res Rev*. 2006; 51(1):85-107.
21. 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.
22. Adam TC, Epel ES. Stress, eating and the reward system. *Physiol Behav*. 2007;91(4):449-458.
23. Goeders NE. Stress and cocaine addiction. *J Pharmacol Exp Ther*. 2002;301(3):785-789.
24. 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.
25. Gearhardt AN, Grilo CM, DiLeone RJ, Brownell KD, Potenza MN. Can food be addictive? public health and policy implications. *Addiction*. 2011;106(7):1208-1212.
26. Mason SM, Flint AJ, Field AE, Austin SB, Rich-Edwards JW. Abuse victimization in childhood or adolescence and risk of food addiction in adult women. *Obesity (Silver Spring)*. 2013;21(12):e775-e781.
27. Gearhardt AN, Corbin WR, Brownell KD. Preliminary validation of the Yale Food Addiction Scale. *Appetite*. 2009;52(2):430-436.
28. Schnurr PP, Lunney CA, Sengupta A, Spiro A III. A longitudinal study of retirement in older male veterans. *J Consult Clin Psychol*. 2005;73(3):561-566.
29. Schnurr P, Spiro A, Velhauer M, Findler M, Hamblen J. Trauma in the lives of older men: findings from the Normative Aging Study. *J Clin Geropsychol*. 2002;8(3):175-187.
30. Breslau N, Peterson EL, Poisson LM, Schultz LR, Lucia VC. Estimating post-traumatic stress disorder in the community: lifetime perspective and the impact of typical traumatic events. *Psychol Med*. 2004;34(5):889-898.
31. Breslau N, Peterson EL, Kessler RC, Schultz LR. Short screening scale for DSM-IV posttraumatic stress disorder. *Am J Psychiatry*. 1999;156(6):908-911.
32. Roberts AL, Gilman SE, Breslau J, Breslau N, Koenen KC. Race/ethnic differences in exposure to traumatic events, development of post-traumatic stress disorder, and treatment-seeking for post-traumatic stress disorder in the United States. *Psychol Med*. 2011;41(1):71-83.
33. Koenen KC, Moffitt TE, Poulton R, Martin J, Caspi A. Early childhood factors associated with the development of post-traumatic stress disorder: results from a longitudinal birth cohort. *Psychol Med*. 2007;37(2):181-192.
34. Stunkard A, Sorenson T, Schulsinger F. *The Genetics of Neurological and Psychiatric Disorders*. New York, NY: Raven Press; 1983.
35. Zou G. A modified Poisson regression approach to prospective studies with binary data. *Am J Epidemiol*. 2004;159(7):702-706.
36. Kessler RC, Berglund PA, Chiu WT, et al. The prevalence and correlates of binge eating disorder in the World Health Organization World Mental Health Surveys. *Biol Psychiatry*. 2013;73(9):904-914.
37. Mitchell KS, Mazzeo SE, Schlesinger MR, Brewerton TD, Smith BN. Comorbidity of partial and subthreshold PTSD among men and women with eating disorders in the national comorbidity survey-replication study. *Int J Eat Disord*. 2012;45(3):307-315.
38. Dansky BS, Brewerton TD, Kilpatrick DG, O'Neil PM. The National Women's Study: relationship of victimization and posttraumatic stress disorder to bulimia nervosa. *Int J Eat Disord*. 1997;21(3):213-228.
39. Gearhardt AN, White MA, Masheb RM, Morgan PT, Crosby RD, Grilo CM. An examination of the food addiction construct in obese patients with binge eating disorder. *Int J Eat Disord*. 2012;45(5):657-663.
40. Teicher MH, Tomoda A, Andersen SL. Neurobiological consequences of early stress and childhood maltreatment: are results from human and animal studies comparable? *Ann N Y Acad Sci*. 2006;1071:313-323.
41. Heim C, Nemeroff CB. The role of childhood trauma in the neurobiology of mood and anxiety disorders: preclinical and clinical studies. *Biol Psychiatry*. 2001;49(12):1023-1039.
42. Heim C, Shugart M, Craighead WE, Nemeroff CB. Neurobiological and psychiatric consequences of child abuse and neglect. *Dev Psychobiol*. 2010;52(7):671-690.