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POSTTRAUMATIC STRESS DISORDER SYMPTOMS AND PROBLEMATIC OVEREATING BEHAVIORS IN YOUNG MEN AND WOMEN

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Abstract

Background—PTSD is a risk factor for obesity, but the range of behaviors that contribute to this association are not known.

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Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent: Informed consent was obtained from all individual participants included in the study.

Purpose—To examine associations between self-reported PTSD symptoms in 2007, with and without comorbid depression symptoms, and three problematic overeating behaviors in 2010; to estimate the associations of PTSD-related overeating behaviors with obesity.

Methods—Cross-sectional and longitudinal analyses included 7438 male (n=2478) and female (n=4960) participants from the Growing Up Today Study (mean age 22–29 years in 2010). Three eating behavior outcomes were assessed: Binge eating (eating a large amount of food in a short period of time with loss of control), top quartile of coping-motivated eating (from the Motivations to Eat scale), and top quartile of disinhibited eating (from the Three-Factor Eating Questionnaire).

Results—PTSD symptoms were associated with 2- to 3-fold increases in binge eating and top-quartile coping-motivated eating; having ≥ 4 PTSD symptoms, relative to no PTSD symptoms, was associated with covariate-adjusted RRs of 2.7 (95% CI: 2.1, 3.4) for binge eating, 2.1 (95% CI: 1.9, 2.4) for the top quartile of coping-motivated eating, and 1.5 (95% CI: 1.3, 1.7) for the top quartile of disinhibited eating. There was a trend toward PTSD symptoms in 2007 predicting new onset binge eating in 2010. Having depression symptoms comorbid with PTSD symptoms further increased risk of binge eating and coping-motivated eating. All eating behaviors were associated with obesity.

Conclusion—Clinicians treating patients with PTSD should know of potential comorbid problematic eating behaviors that may contribute to obesity.

Keywords

posttraumatic stress disorder; depression; obesity; eating behavior; binge eating

INTRODUCTION

Posttraumatic stress disorder (PTSD) affects 6.8% of US civilians at some point in their lives (1), with many more affected by subthreshold symptoms (2). PTSD is increasingly recognized as a risk factor for obesity (3–8) and obesity-related diseases (2; 9–11). The mechanisms driving the PTSD–obesity association are not known, but eating as a way to cope with distress may be a contributing factor. The eating-to-cope hypothesis is supported by neurological studies indicating that ingestion of high-fat and high-sugar foods blunts feelings of distress by triggering dopaminergic reward responses (12; 13). Further, a relatively large literature indicates that PTSD is frequently comorbid with eating disorders relevant to weight gain (e.g. binge eating disorder)(14–17). This literature has generally focused on eating disorder diagnoses, the most severe end of a spectrum of potentially problematic eating. Far less is known about how PTSD symptoms are associated with common, problematic overeating behaviors that do not meet diagnostic thresholds but may nonetheless contribute to obesity risk.

PTSD is frequently comorbid with depression (18), and it is plausible that the combination of PTSD symptoms and depression symptoms is associated with additional weight-related risks (19). Depression has been found to predict obesity in some studies (19; 20), potentially due to its association with disordered eating behaviors (21). There is also evidence that depressive symptoms reduce individuals' capacity to cope with life stressors (22; 23). This

reduced coping capacity may put those with a combination of PTSD and depressive symptoms at uniquely high risk for maladaptive coping such as problematic eating.

A greater understanding of the links of PTSD symptoms, with and without comorbid depression, with a range of prevalent obesogenic eating behaviors is needed to understand PTSD-related comorbidities and highlight potential targets for obesity prevention in PTSD-affected populations. To our knowledge the current literature on this topic is limited to a handful of studies. These include two studies on PTSD and emotional eating (24; 25) (one showing a positive finding, the other null), two studies showing PTSD symptoms to be associated with food addiction (26; 27), and four studies finding greater intake of unhealthy foods and/or lower intake of healthy foods among those with PTSD (28–30). One study of earthquake survivors found that meeting PTSD diagnostic criteria was associated with increased craving for sweets or carbohydrates, as well as both increases and decreases in appetite (31). Other common overeating behavior phenotypes have not been examined. Further, few studies have explicitly examined the combination of PTSD and depression symptoms in relation to eating behavior. Finally, none of the existing studies have been able to examine the longitudinal links between PTSD symptoms and new onset of eating behaviors.

We aimed to address these gaps in the literature using data from the Growing Up Today Study, a community-based longitudinal cohort of over 7000 young men and women. Our study aims were to estimate: (1) the cross-sectional associations between PTSD symptoms and three common types of problematic overeating: sub-threshold binge eating (binge eating at any frequency), coping-motivated eating (32), and disinhibited eating (33); (2) the longitudinal association between PTSD symptoms and subsequent new onset of binge eating among women and men without a prior binge eating history; and (3) the independent and joint impacts of PTSD and depression symptoms on eating behaviors. Finally, to establish the relevance of these findings to the PTSD-obesity association, we aimed to (4) examine the association of each type of problematic eating with obesity. Because of prior literature suggesting that the PTSD-obesity association is stronger for women than men (4–6), we examined modifications of associations of PTSD, eating behaviors, and obesity by gender, and present stratified results where appropriate.

METHODS

Study Population

The Growing Up Today Study (GUTS) comprises children of participants in the Nurses' Health Study II (NHSII). In 1996, NHSII participants with children aged 9–14 years were asked to provide consent for their children to participate in GUTS; 18,526 mothers (54%) consented, and their children (n=26,765) were invited to participate. Of these, 16,882 (9039 girls and 7843 boys) returned the baseline questionnaire and have been followed through a series of 11 online and mailed questionnaires collecting self-reported demographic, behavioral, and medical data.

We excluded participants from our analytic sample if they did not complete the 2007 GUTS questionnaire on which PTSD symptoms were assessed (n=2870 women and 4150 men), if

they had missing or incomplete PTSD data (n=285 women and 304 men), or if they did not complete the 2010 questionnaire on which eating behavior outcomes were ascertained (n=924 women and 911 men). This left 7438 participants for our analysis. Participants were aged 22 to 29 years in 2010 when eating behaviors were assessed.

Variables

Exposures

PTSD symptoms in 2007: The 2007 questionnaire assessed PTSD symptoms using Breslau's Short Screening Scale (34). Participants were asked to report up to 7 lifetime PTSD symptoms, including intrusive thoughts, emotional numbness, and hypervigilance, in reference to the "most distressing" event that they had experienced. Participants were not asked to specify the event they found most distressing. For each PTSD symptom endorsed, participants were asked to report how frequently they had experienced that symptom in the past 4 weeks (5-point scale from "none of the time" to "most or all of the time"). We defined participants as having a current PTSD symptom if they reported experiencing it with any frequency in the past 4 weeks. These symptoms do not designate a clinical PTSD diagnosis; however, self-reported symptoms assessed with this screener have been found to predict important health outcomes such as increases in weight status and type 2 diabetes (3; 35). Breslau et al. recommend ≥4 symptoms as the cut-off for significant PTSD symptoms (34), reporting 80% sensitivity and 97% specificity for this cut-off (34). Preliminary analyses indicated that additional symptoms beyond this cut-off did not confer additional risk for the eating behavior outcomes, and we therefore categorized PTSD symptoms in three levels: 0, 1–3, and ≥4 symptoms.

Joint PTSD and depressive symptoms in 2007: PTSD and depression are frequently comorbid, and depression has been associated with obesity in some, though not all, studies (20; 36). We were therefore interested in examining whether the combination of PTSD and depression symptoms conferred additional risk of problematic eating behaviors. The 2007 questionnaire included the short form of the Centers for Epidemiologic Studies-Depression (CESD-10) scale (37), which assesses the frequency of depressive symptoms over the past week. A score of 10 or more is considered indicative of significant depressive symptoms (37), with research showing 100% sensitivity and 93% specificity for major depression in a community-based sample (38). To examine joint PTSD and depressive symptoms, we dichotomized PTSD symptoms at ≥4 and the CESD-10 score at ≥10 and created a 4-category variable with the following levels: No significant PTSD or depression symptoms (below the cut-offs on both the PTSD screener and the CESD-10 scale), significant depression symptoms only, significant PTSD symptoms only, and both significant PTSD and significant depression symptoms.

Outcomes—We examined the association of 2007 PTSD and depression symptoms with three problematic eating behavior phenotypes, measured in 2010: subthreshold binge eating, coping-motivated eating, and disinhibited eating. In addition, we examined the association of each eating behavior, and PTSD symptoms, with obesity in 2013.

Binge eating in 2010: Binge eating self-report has been collected on each survey over GUTs follow-up. For our main analyses, we used binge eating reported on the 2010 questionnaire (the first questionnaire after the 2007 assessment of PTSD) as our outcome. Binge eating questions asked participants whether, over the past year, they had eaten an extremely large quantity of food over a short period of time ("overeating"), and the frequency with which this had occurred (5-point scale from "Never" to "More than once a week"). Participants who indicated that they had overeaten at any frequency were asked whether they had felt unable to stop eating at that time ("loss of control"). We defined binge eating as overeating with loss of control at any frequency in 2010. This measure captures a relatively common form of problematic eating, as distinct from binge eating disorder (which has specific, severe clinical manifestations). A comparison of this measure with results of clinical interviews assessing binge eating disorder among girls found that the survey measure had a sensitivity of 0.53, specificity of 0.79, and negative predictive value of 0.98.[39] These values suggest that whereas the measure is not optimized to capture the small number of individuals with binge eating disorder, it correctly separates most respondents who do not exhibit binge eating symptoms from those who do (i.e., high negative predictive value).

Binge eating was also assessed in 2007. In our longitudinal analysis of PTSD and incident binge eating (described in the analysis section), we excluded participants with prevalent binge eating behavior reported in 2007 (the time of PTSD assessment) so that we could establish the temporal order from PTSD symptoms to binge onset.

Coping-motivated eating in 2010: The coping-motivated eating subscale of the Motivations to Eat Scale (32) was included on the 2010 questionnaire. Jackson et al. report an internal consistency reliability of α=0.88 and have found the subscale scores to be positively correlated with binge eating (32). Items in this 5-item subscale ascertain frequency (never or almost never, rarely, sometimes, often, almost always or always) with which participants eat because they feel depressed, sad, or worthless; to cope; to comfort themselves; or to distract themselves. We examined coping-motivated eating both as a continuous outcome and dichotomized at the gender-specific 75th percentile. In preliminary analyses, we found that those with scores below the 75th percentile were normal weight on average, whereas those above were overweight, suggesting that this is a substantively relevant cut-point.

Disinhibited eating in 2010: The 2010 questionnaire included the disinhibited eating subscale of the Three Factor Eating Questionnaire (33). This 10-item subscale measures the tendency to overeat in response to certain stimuli, such as during social occasions, when with someone who is overeating, in the presence of palatable foods, and as a reaction to dieting. In a recent study, reliability of this subscale ranged from 0.78 in normal-weight individuals to 0.80 in obese individuals (40). As with the coping-motivated eating scale, we examined disinhibited eating both as a continuous measure and dichotomized it at the gender-specific 75th percentile. Similar to coping-motivated eating, we found that the 75th percentile cut-point roughly aligned with the point at which the average body weight changed from normal-weight to overweight.

Obesity in 2013. Obesity was defined as a body mass index (BMI) of >30 kg/m^2 based on self-reported height and weight in 2013. Prior work has shown that self-reported BMI is closely correlated with measured BMI (41; 42).

Covariates—For our main adjusted analyses, we included potential confounders of the association between PTSD and disordered eating, including age at baseline (continuous), nonwhite race (dichotomous), perceived social status (participants were asked to indicate their family's status in American society, from 1, best off, to 10, worst off; continuous), and participant BMI percentile at 1996 study baseline standardized to CDC growth charts (43) (continuous with squared term). We ran subsequent models additionally adjusting for other health behavior correlates of eating behaviors that may be downstream from PTSD, including participant smoking history in 2007 (ever/never), history of dieting (ever/never) in 2005 (the most recent data available prior to 2010 outcomes), and BMI in 2007. In sensitivity analyses, we adjusted for participants' mothers' BMI at age 18. Maternal BMI is a strong predictor of child BMI, and potentially a predictor of child eating behaviors (via maternal eating behaviors); it may also be a marker of risk factors (e.g., low socioeconomic status, intergenerational maltreatment) for child exposure to trauma and/or propensity toward PTSD. (Mothers are Nurses' Health Study II participants; weight at age 18 was asked on the NHSII baseline survey and validated against nursing school entry physical exams (44)).

Data analysis

Our analytic sample included 7438 participants (4960 women and 2478 men) with non-missing PTSD symptom data who had completed the 2010 questionnaire on which the eating behavior outcomes of interest were ascertained. For analyses of each outcome, we excluded the following number of participants because they were missing the outcome variable: 124 women and 122 men missing binge eating, 307 women and 322 men missing coping-motivated eating, and 304 women and 321 men missing disinhibited eating.

Association of PTSD symptoms with eating behaviors—We used modified Poisson regression (45) to estimate risk ratios for each eating behavior outcome in 2010 as a function of the number of reported PTSD symptoms experienced in 2007. We then assessed the longitudinal nature of the relationship between PTSD and binge eating by running models of incident binge eating in 2010 as a function of the number of current PTSD symptoms reported in 2007, among participants with no history of binge eating prior to or in 2007 (when PTSD was assessed; n=1645 women and 303 men). Finally, we examined eating behaviors as a function of joint exposure to significant PTSD symptoms and/or significant depression symptoms.

For each analysis above, we ran a series of models in the following sequence: crude models, addition of main sociodemographic covariates and baseline BMI percentile, and addition of adult health behaviors and 2007 body mass index. We tested for interactions between PTSD symptoms and gender in predicting each outcome. No interactions were statistically significant (Wald test p-values for gender interactions with binge eating, continuous coping-

motivated eating, and continuous disinhibited eating were 0.67, 0.21, and 0.30, respectively). We therefore examined these associations in the combined sample of men and women.

We ran three sensitivity analyses of PTSD symptoms and eating behaviors to assess the sensitivity of our results to various modeling decisions. First, we reran PTSD–eating behavior models with additional adjustment for maternal BMI at age 18 years, as a potential shared risk factor for PTSD symptoms and eating behaviors. Second, to assess the impact of attrition out of the sample and related selection bias, we reran PTSD–eating behavior models using stabilized inverse probability weights to adjust for differential non-response to the 2007 and 2010 questionnaires, on which our exposure and outcome were measured. Third, in an attempt to assess whether results were likely to be driven by PTSD-related medication use we reran PTSD–eating behavior models after excluding those who reported past-year use of psychotropic medications (antidepressants or antianxiety medications) on the 2013 questionnaire, the only questionnaire to assess use of these medications.

Associations of eating behaviors and PTSD symptoms with obesity—To examine the extent to which our findings on PTSD and eating behavior would have implications for weight-related health, we examined the association of each eating behavior outcome with obesity. We also estimated risk ratios for obesity in 2013 as a function of 2007 PTSD symptoms. We used modified Poisson regression (45) to estimate risk ratios for obesity in 2013 as a function of (in separate models) binge eating, top quartile of coping-motivated eating, top quartile of disinhibited eating, and categorical PTSD symptoms. Continuous coping-motivated and disinhibited eating were not linearly associated with obesity, and methods to relax the linearity assumption such as inclusion of squared terms or splines make interpretation difficult; we therefore include results for these eating behaviors dichotomized at the top quartile. For each exposure (eating behavior or PTSD symptoms), we ran covariate-adjusted models and an additional model adjusted for baseline BMI. We tested for interactions between gender and each exposure. We found that interactions were significant for gender with PTSD symptoms (Wald p-value for interaction=0.03), but not for gender with eating behaviors. However, for consistency, we present all obesity results stratified by gender.

Results

Of 7438 participants in the analytic sample, 13% reported ≥ 4 PTSD symptoms in 2007. Participants with and without PTSD symptoms were similar in age, race, and perceived social status. Participants with PTSD symptoms were more likely than those without to be female, have a history of smoking or dieting, and to have engaged in binge eating, coping-motivated eating, and disinhibited eating in 2010 (Table 1).

Association of PTSD symptoms with eating behaviors—In main analyses of the association between PTSD symptoms and eating behaviors, we found that the prevalence of all three eating behavior phenotypes increased with the number of PTSD symptoms (Table 2). Associations were generally stronger for binge eating and coping-motivated eating than for disinhibited eating. For example, having ≥ 4 PTSD symptoms, relative to no PTSD symptoms, was associated with main covariate-adjusted RRs of 2.7 (95% CI: 2.1, 3.4) for

binge eating, 2.1 (95% CI: 1.9, 2.4) for the top quartile of coping-motivated eating, and 1.5 (95% CI: 1.3, 1.7) for the top quartile of disinhibited eating (Table 2). In sensitivity analyses, adjusting for maternal BMI made little difference in the associations of PTSD symptoms with eating behaviors. Likewise, results were comparable when adjusted for non-response to key questionnaires using inverse probability weights, and when excluding participants taking psychotropic medications.

Longitudinal association of PTSD symptoms with incident binge eating—We ran a longitudinal model restricting our sample to participants with no history of binge eating prior to PTSD symptom ascertainment. We found suggestive evidence that PTSD symptoms in 2007 predicted new onset of binge eating in 2010 (Table 3). Those with ≥ 4 PTSD symptoms in 2007 had twice the risk of reporting binge eating for the first time in 2010, (main covariate-adjusted RR=2.2; 95% CI: 1.1, 4.3). Additional adjustment for maternal BMI attenuated this RR to 1.9 (95% CI: 0.9, 3.9).

Comorbid PTSD and depression symptoms and eating behaviors—PTSD and depression are frequently comorbid, and their combination may confer additional risk of problematic eating behaviors. Analyses of combinations of significant depressive symptoms (CESD-10 ≥ 10) and PTSD symptoms (≥ 4) indicated that each mental health problem was independently associated with each of the eating behavior phenotypes of interest (Table 4). However, the strength of the associations across the joint PTSD-depression categories differed qualitatively by eating behavior. Main covariate-adjusted binge eating analyses indicated that PTSD with depression (RR= 2.8; 95% CI: 2.3, 3.6) and PTSD without depression (RR=2.2; 95% CI: 1.5, 3.3) were more strongly associated with binge eating than depression alone (RR=1.8; 95% CI: 1.4, 2.2). In analyses of coping-motivated eating, exposure to the combination of PTSD and depression was associated with a larger elevation in risk (RR=2.3; 95% CI: 2.0, 2.6) than either depression alone (RR=1.9; 95% CI: 1.7, 2.1) or PTSD alone (RR=1.8; 95% CI: 1.4, 2.3). RRs were overall smaller for disinhibited eating and were similar across categories of PTSD, depression, and their combination.

Associations of eating behavior and PTSD symptoms with obesity—In analyses assessing the implications of our eating behaviors for obesity risk, we found that binge, coping-motivated, and disinhibited eating in 2010 were each associated with elevated obesity prevalence in both women and men (Table 5). Among women, top quartile of coping-motivated eating and top quartile of disinhibited eating were associated with main covariate- and baseline BMI-adjusted RRs for obesity of 1.5 (95% CI: 1.3, 1.7), and 1.5 (95% CI: 1.3, 1.8), respectively, relative to those without the eating behavior. For binge eating, the sociodemographic-adjusted RR for obesity was 1.8 (95% CI: 1.5, 2.2), but this was attenuated upon adjustment for baseline BMI (RR=1.1; 95% CI: 0.9 1.3). It is possible that baseline BMI was influenced by long-standing binge eating behavior and that adjustment for baseline BMI therefore adjusted away some of the association of interest. For men, the obesity RRs associated with binge, coping-motivated, and disinhibited eating adjusting for sociodemographics and baseline BMI were 1.5 (95% CI: 1.0, 2.2), 1.5 (95% CI: 1.2, 1.9), and 2.0 (95% CI: 1.6, 2.6). PTSD symptoms in 2007 were also associated with obesity in 2013 in women (main covariate-adjusted RR=1.5, 95% CI: 1.2, 1.8; baseline

BMI-adjusted RR=1.3, 95% CI: 1.1, 1.6); however, they were unrelated to obesity in 2013 in men.

Discussion

In this large cohort of young men and women, we found strong associations between reported PTSD symptoms and three eating behavior phenotypes that are associated with obesity. We also found suggestive evidence of longitudinal associations between PTSD symptoms and new onset of binge eating. In general, PTSD symptoms were less strongly associated with disinhibited eating than with binge eating and coping-motivated eating, perhaps because binge and coping-motivated eating appear to be driven by emotional states (32; 46), whereas disinhibited eating may be more strongly influenced by external cues and/or problems with impulse control (33). Most eating behaviors examined were associated with increases in obesity risk. (Among women, the RR for binge eating was close to 1 after adjustment for baseline BMI; it is possible that this null value is due to overadjustment, as baseline BMI have been influenced by pre-baseline binge eating.)

Overall, these findings support hypotheses suggesting that overconsumption of food may be a common coping strategy for those experiencing significant psychological distress (47), which may in turn drive elevated obesity risks associated with PTSD symptoms. However, we did not find that PTSD symptoms were universally associated with obesity; only women in our cohort showed an association between PTSD symptoms in 2007 and obesity in 2013. It is unclear why no such association emerged for men; we explored the possibility that men with PTSD and problematic overeating behaviors disproportionately compensated for their overeating (e.g., by fasting or purging). However, we found that few men reported compensatory behaviors. It seems likely that PTSD-related obesity may emerge over time in men if those with PTSD symptoms continue to engage in obesogenic eating behaviors at a high rate (3). Consistent with our findings, prior studies have found stronger PTSD–obesity links among women (4–6), and there is recent evidence for an emergence of PTSD-related obesity among men at older ages (48).

Our findings also suggest that the combination of PTSD and depressive symptoms may put men and women at particularly high risk for problematic eating. The combination of PTSD and depressive symptoms may be more strongly associated with problematic eating simply because it indicates especially severe psychiatric distress. It is also possible that depressive symptoms undermine individuals' capacity to cope with PTSD symptoms in constructive ways (22), exacerbating reliance on unhealthy coping strategies such as overeating.

Our study expands on existing literature linking PTSD to eating disorder diagnoses (14; 16; 17; 49–51), by highlighting common and less severe eating behaviors that may nonetheless be of concern for long-term health. In particular, revealing the associations between PTSD symptoms and sub-clinical behaviors may help better pinpoint targets for earlier intervention. The limitations of our study include lack of information about time of onset of coping-motivated eating and disinhibited eating, which prevented us from establishing the temporal relationship between PTSD symptoms and these eating behaviors. Further, our measures were survey-based and are therefore subject to errors in reporting. In particular, our measure of obesity is based on self-report and may be subject to error, despite several

studies suggesting this error is likely to be minimal (41; 42). PTSD symptoms were assessed at a single time-point, limiting our ability to discriminate those with longer-term versus shorter-term experiences of PTSD. Nevertheless, our study has several notable strengths including a large community-based sample of both men and women and rich covariate data including baseline BMI. In addition, we were able to address prior gaps in the literature by examining the joint contributions of PTSD and depressive symptoms, looking at several eating behavior phenotypes, and exploring longitudinal relationships between PTSD symptoms and subsequent incidence of binge eating.

Our findings add to a growing body of evidence suggesting that problematic eating behaviors are common among those suffering from PTSD symptoms, and provide suggestive support for a temporal sequence from PTSD to initiation of problematic eating. Our results further suggest that these eating behaviors may be important contributors to obesity, highlighting a potentially important mechanism by which PTSD symptoms increase obesity risk. This potential mechanism warrants additional research, especially because it will clarify eating behaviors as possible targets for intervention in the prevention of obesity among those with PTSD. Clinicians treating those with traumatic stress symptoms should be aware that many patients, particularly those with both PTSD and depressive symptoms, have comorbid eating behaviors that may put them at risk for weight gain and obesity. Screening for eating problems in PTSD care, and development of integrated treatments that address trauma and problematic eating simultaneously, may be warranted. Further, clinicians treating people with obesity might consider screening for traumatic stress symptoms and associated eating phenotypes. Such screening could lead to more sensitive and effective care than lifestyle counseling alone. Finally, our results emphasize the need for health promotion and obesity prevention strategies that address the potential influence of psychological factors on individuals' health behaviors.

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Table 1

Distribution of outcome and covariates by number of PTSD symptoms among women and men in the Growing Up Today study.

Outcomes and covariates	0 symptoms (n=3925) % or Mean(SD)	1–3 symptoms (n=2544) % or Mean(SD)	4–7 symptoms (n=969) % or Mean(SD)
Binge eating in 2010[a]	4.6	7.0	12.6
Top quartile of coping-motivated eating[b]	3.0(3.7)	4.0(4.1)	5.5(4.8)
Top quartile of disinhibited eating[c]	2.8(2.3)	3.1(2.4)	3.5(2.6)
Age at 1996 baseline (years)	12.1(1.6)	12.0(1.6)	12.0(1.6)
Gender (female)	62.1	70.9	73.9
Nonwhite race	3.0	3.2	3.6
BMI percentile at 1996 baseline[d]	55.2(29.1)	54.8(30.2)	55.9(30.9)
Perceived social status relative to US[e]	3.8(1.2)	3.9(1.2)	3.9(1.2)
Participant history of smoking	50.9	57.1	62.1
Participant history of dieting	59.3	65.4	71.2

[a]Participants were defined as having binge eating in 2010 if they responded that they had eaten an extremely large quantity of food over a short period of time with any frequency over the past year, and that during that episode they had felt unable to stop eating.

[b]Coping-motivated eating was measured with 5 items ascertaining the frequency (never or almost never, rarely, sometimes, often, almost always or always) with which participants eat because they feel depressed, sad, or worthless; to cope; to comfort themselves; or to distract themselves. The score was dichotomized at the gender-specific 75th percentile.

[c]Disinhibited eating was assessed with 10 items measuring the tendency to overeat in response to certain stimuli, such as during social occasions, when with someone who is overeating, in the presence of palatable foods, and as a reaction to dieting. The score was dichotomized at the gender-specific 75th percentile.

[d]BMI percentile at baseline was computed from self-reported height and weight, standardized to CDC growth charts.

[e]Perceived social status relative to the US was assessed by asking participants where on a scale of 1 (best off) and 10 (worst off) their family fell in American society.

Table 2

Associations between PTSD symptoms experienced in 2007 and three eating behavior outcomes in 2010 in the Growing Up Today Study

Eating behavior outcome	Number of PTSD symptoms		Model 1: Crude		Model 2: Adjusted for main covariates[a]		Model 3: Adjusted for health behaviors [b]	
Dichotomous outcomes		***N (cases)***	***RR***	***(95% CI)***	***RR***	***(95% CI)***	***RR***	***(95% CI)***
Binge eating								
	0	3810 (174)	1	–	1	–	1	–
	1–3	2451 (172)	1.5	(1.3, 1.9)	1.5	(1.2, 1.8)	1.4	(1.2, 1.8)
	4–7	931 (120)	2.8	(2.3, 3.5)	2.7	(2.1, 3.4)	2.5	(2.0, 3.2)
Top quartile of coping-motivated eating								
	0	3604 (589)	1	–	1	–	1	–
	1–3	2315 (566)	1.5	(1.3, 1.7)	1.5	(1.4, 1.7)	1.5	(1.4, 1.7)
	4–7	890 (320)	2.2	(2.0, 2.5)	2.1	(1.9, 2.4)	2.0	(1.8, 2.3)
Top quartile of disinhibited eating								
	0	3605 (710)	1	–	1	–	1	–
	1–3	2318 (522)	1.1	(1.0, 1.3)	1.2	(1.1, 1.3)	1.2	(1.0, 1.3)
	4–7	890 (253)	1.4	(1.3, 1.6)	1.5	(1.3, 1.7)	1.4	(1.2, 1.6)
Continuous outcomes		*N (means)*	*β*	*(95% CI)*	*β*	*(95% CI)*	*β*	*(95% CI)*
Coping-motivated eating (range 0–20)								
	0	3604 (3.0)	0	–	0	–	0	–
	1–3	2315 (4.0)	1.0	(0.8, 1.2)	0.8	(0.6, 1.0)	0.7	(0.5, 1.0)
	4–7	890 (5.5)	2.4	(2.1, 2.8)	2.1	(1.8, 2.5)	2.0	(1.7, 2.4)
Disinhibited eating (range 0–10)								
	0	3605 (2.8)	0	–	0	–	0	–
	1–3	2318 (3.1)	0.4	(0.2, 0.5)	0.3	(0.2, 0.4)	0.3	(0.2, 0.4)
	4–7	890 (3.6)	0.8	(0.6, 1.0)	0.7	(0.5, 0.9)	0.6	(0.4, 0.8)

[a]Model 2 is adjusted for participant age at baseline (continuous), gender, participant body mass index at baseline (standardized to CDC standard growth curves; continuous with a squared term), nonwhite race, and perceived social status (continuous).

[b]Model 3 is adjusted for Model 2 covariates plus participant body mass index in 2007 when PTSD was ascertained (in kg/m^2, continuous with a squared term), participant history of dieting (yes or no, through 2005) and participant history of smoking (yes or no, through 2007).

Table 3

Association between PTSD symptoms in 2007 and first onset of binge eating in 2010 in the Growing Up Today Study with no history of binge eating prior to 2010 (n=5244)

Number of PTSD symptoms	N (cases)	Model 1: Crude		Model 2: Adjusted for main covariates [a]		Model 3: Adjusted for health behaviors[b]		Model 4: Adjusted for maternal BMI[c]	
		RR	(95% CI)	RR	(95% CI)	RR	(95% CI)	RR	(95% CI)
0	2997 (32)	1	–	1	–	1	–	1	–
1–3	1704 (31)	1.7	(1.0, 2.8)	1.6	(1.0, 2.8)	1.7	(1.0, 2.8)	1.7	(1.0, 2.8)
4–7	543 (14)	2.4	(1.3, 4.5)	2.2	(1.1, 4.3)	2.1	(1.1, 4.2)	1.9	(0.9, 3.9)

[a]Model 2 is adjusted for participant age at baseline (continuous), gender, participant body mass index at baseline (standardized to CDC standard growth curves; continuous with a squared term), nonwhite race, and perceived social status (continuous).

[b]Model 3 is adjusted for Model 2 covariates plus participant body mass index in 2007 when PTSD was ascertained (in kg/m^2, continuous with a squared term), participant history of dieting (yes or no, through 2005) and participant history of smoking (yes or no, through 2007).

[c]Model 4 is adjusted for Model 3 covariates plus participant's mother's body mass index at age 18 (mothers are Nurses' Health Study II participants; weight at age 18 was asked on the NHSII baseline survey and validated against nursing school entry physical exams).

Table 4

Associations of significant PTSD and depression symptoms in 2007 with three eating behavior phenotypes in 2010 in the Growing Up Today Study

Eating behavior outcome	Exposure to PTSD and/or Depression			Model 1: Crude		Model 2: Adjusted for main covariates[a]		Model 3: Adjusted for health behaviors[b]	
Dichotomous outcomes	***PTSD***	***Depression***	***N (cases)***	***RR***	***(95% CI)***	***RR***	***(95% CI)***	***RR***	***(95% CI)***
Binge eating									
	No	No	4567 (209)	1	–	1	–	1	–
	No	Yes	1572 (136)	1.9	(1.5, 2.3)	1.8	(1.4, 2.2)	1.6	(1.3, 2.0)
	Yes	No	245 (25)	2.2	(1.5, 3.3)	2.2	(1.5, 3.3)	2.1	(1.4, 3.2)
	Yes	Yes	685 (95)	3.0	(2.4, 3.8)	2.8	(2.2, 3.6)	2.6	(2.1, 3.3)
Top quartile of coping-motivated eating									
	No	No	4325 (679)	1	–	1	–	1	–
	No	Yes	1478 (467)	2.0	(1.8, 2.2)	1.9	(1.7, 2.1)	1.8	(1.6, 2.0)
	Yes	No	232 (64)	1.8	(1.4, 2.2)	1.8	(1.4, 2.3)	1.7	(1.4, 2.1)
	Yes	Yes	657 (255)	2.5	(2.2, 2.8)	2.3	(2.0, 2.6)	2.2	(1.9, 2.5)
Top quartile of disinhibited eating									
	No	No	4326 (772)	1	–	1	–	1	–
	No	Yes	1480 (443)	1.7	(1.5, 1.9)	1.6	(1.5, 1.8)	1.5	(1.4, 1.7)
	Yes	No	232 (51)	1.2	(1.0, 1.6)	1.5	(1.2, 1.9)	1.4	(1.1, 1.8)
	Yes	Yes	657 (201)	1.7	(1.5, 2.0)	1.7	(1.4, 1.9)	1.5	(1.3, 1.8)
Continuous outcomes	*PTSD*	*Depression*	*N (means)*	*β*	*(95% CI)*	*β*	*(95% CI)*	*β*	*(95% CI)*
Coping-motivated eating (range 0–20)									
	No	No	4325 (3.0)	0	–	0	–	0	–
	No	Yes	1478 (4.8)	1.8	(1.6, 2.1)	1.6	(1.4, 1.9)	1.5	(1.3, 1.8)
	Yes	No	232 (4.6)	1.7	(1.1, 2.2)	1.4	(0.9, 2.0)	1.4	(0.8, 1.9)
	Yes	Yes	657 (5.8)	2.8	(2.4, 3.2)	2.5	(2.1, 2.9)	2.4	(2.0, 2.8)
Disinhibited eating (range 0–10)									
	No	No	4326 (2.7)	0	–	0	–	0	–
	No	Yes	1480 (3.5)	0.8	(0.7, 1.0)	0.7	(0.6, 0.9)	0.6	(0.5, 0.7)
	Yes	No	232 (3.4)	0.7	(0.4, 1.0)	0.7	(0.4, 1.0)	0.7	(0.3, 1.0)

Eating behavior outcome	Exposure to PTSD and/or Depression			Model 1: Crude		Model 2: Adjusted for main covariates[a]		Model 3: Adjusted for health behaviors[b]	
Dichotomous outcomes	***PTSD***	***Depression***	***N (cases)***	***RR***	***(95% CI)***	***RR***	***(95% CI)***	***RR***	***(95% CI)***
	Yes	Yes	657 (3.6)	0.9	(0.7, 1.1)	0.7	(0.5, 1.0)	0.6	(0.4, 0.8)

[a]Model 2 is adjusted for participant age at baseline (continuous), gender, participant body mass index at baseline (standardized to CDC standard growth curves; continuous with a squared term), nonwhite race, and perceived social status (continuous).

[b]Model 3 is adjusted for Model 2 covariates plus participant body mass index in 2007 when PTSD was ascertained (in kg/m^2, continuous with a squared term), participant history of dieting (yes or no, through 2005) and participant history of smoking (yes or no, through 2007)

Table 5

Associations of PTSD symptoms in 2007 and eating behaviors in 2010 with obesity risk in 2013 in the Growing Up Today Study

Eating behavior/PTSD symptoms	N(cases)	Model 1: Crude		Model 2: Adjusted for sociodemographics[a]		Model 3: Adjusted for baseline BMI[b]	
		RR	(95% CI)	RR	(95% CI)	RR	(95% CI)
Women Binge eating							
No	3672 (496)	1	–	1	–	1	–
Yes	334 (91)	2.0	(1.7, 2.4)	1.8	(1.5, 2.2)	1.1	(0.9, 1.3)
Top quartile of coping-motivated eating							
No	3036 (333)	1	–	1	–	1	–
Yes	857 (237)	2.5	(2.2, 2.9)	2.4	(2.0, 2.8)	1.5	(1.3, 1.7)
Top quartile of disinhibited eating							
No	3164 (352)	1	–	1	–	1	–
Yes	732 (219)	2.7	(2.3, 3.1)	2.7	(2.3, 3.2)	1.5	(1.3, 1.8)
PTSD symptoms							
0	2047 (258)	1	–	1	–	1	–
1–3	1465 (233)	1.3	(1.1, 1.5)	1.2	(1.0, 1.4)	1.2	(1.0, 1.4)
4–7	566 (104)	1.5	(1.2, 1.8)	1.5	(1.2, 1.8)	1.3	(1.1, 1.6)
Men Binge eating							
No	1753 (245)	1	–	1	–	1	–
Yes	48 (18)	2.7	(1.8, 3.9)	2.7	(1.8, 4.0)	1.5	(1.0, 2.2)
Top quartile of coping-motivated eating							
No	1351 (158)	1	–	1	–	1	–
Yes	340 (94)	2.4	(1.9, 3.0)	2.5	(1.9, 3.1)	1.5	(1.2, 1.9)
Top quartile of disinhibited eating							
No	1226 (109)	1	–	1	–	1	–
Yes	466 (143)	3.5	(2.8, 4.3)	3.4	(2.6, 4.3)	2.0	(1.6, 2.6)
PTSD symptoms							
0	1136(176)	1	–	1	–	1	–
1–3	551(73)	0.9	(0.7, 1.1)	0.8	(0.6, 1.1)	0.7	(0.6, 0.9)
4–7	185(30)	1.0	(0.7, 1.5)	1.1	(0.7, 1.5)	1.0	(0.7, 1.3)