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Longitudinal Associations of Trauma with Disordered Eating: Lessons from the Great Smoky Mountains Study

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Abstract

Disordered eating is prevalent among trauma survivors, yet little is known about mechanisms underlying this relation. We explored cross-sectional and longitudinal associations of trauma exposure and posttraumatic stress disorder symptoms (PTSD) with disordered eating among 1,420 community-based youth participating in the Great Smoky Mountains Study. Participants were interviewed about trauma exposure, PTSD symptoms, and disordered eating at regular intervals throughout childhood, adolescence, and early adulthood. Our findings confirmed associations of all forms of trauma exposure (violent, sexual, and other) with disordered eating symptoms in childhood and adulthood, although the pattern of results varied by disordered eating symptom and trauma exposure type. Only non-sexual, non-violent trauma exposure in childhood had significant associations with any disordered eating symptoms in adulthood. Within childhood, trauma exposures but not PTSD symptoms showed significant longitudinal associations with bulimia nervosa symptoms and sustained appetite changes and preoccupation with eating. In adulthood, PTSD symptoms but not trauma exposures showed significant longitudinal associations only with bulimia nervosa symptoms. The association of specific PTSD clusters on bulimia nervosa symptoms was significant for reexperiencing, whereas hyperarousal symptoms trended toward significance. The impact of trauma exposures on disordered eating may vary by developmental period.

Keywords

Disordered eating; eating disorders; trauma; posttraumatic stress disorder; epidemiology

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Longitudinal Associations of Trauma with Disordered Eating: Lessons from the Great Smoky Mountains Study

Disordered eating (DE) is highly prevalent among those who have been exposed to trauma, but we know relatively little about the mechanisms underlying this relation (Brewerton, 2007). DE refers to aberrant eating and weight-control behaviors (e.g., food restriction, binge eating, purging and other inappropriate compensatory methods) that may or may not occur in the context of a clinically diagnosable eating disorder. Research on the association of trauma exposure and DE has largely relied on retrospective data and focused on traumatic experiences and diagnostic status of posttraumatic stress disorder (PTSD) rather than specific PTSD symptoms. Thus, longitudinal studies that explore how trauma exposure influences vulnerability for subsequent DE and the possible mechanisms by which this occurs would provide vital information for the development of targeted prevention and intervention programs.

Although clinically diagnosable eating disorders are relatively rare in the general population, (among adolescents, 0.3% and 0.9% lifetime prevalence for anorexia nervosa and bulimia nervosa; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011), DE is more common than such full-threshold disorders (Solmi et al., 2015). For example, past-year prevalence of self-induced vomiting among 16-year-old girls ranged from 3.5 to 9.7% across three adolescent cohort studies (N = 7,418; Solmi et al., 2015). Restrictive eating (e.g., fasting, skipping meals) was reported by 60.7% of high-school girls and 27.9% of high-school boys in the Project of Eating and Activity in Teens and Young Adults; Neumark-Sztainer, Wall, Larson, & Eisenberg, 2011). DE has been linked to impairments in physical and psychological functioning (Lipson & Sonneville, 2020; Solmi et al., 2015; Stice & Bearman, 2001). Given this, studying vulnerability factors for DE may be of particular importance. The current study examined the longitudinal associations of trauma exposure with DE in childhood and adulthood using data from a 16-year, population-based cohort study. We also examined the associations of specific PTSD symptom clusters with DE in adulthood.

Cross-sectional Associations of Trauma and DE

Research on trauma exposure and DE initially focused on childhood maltreatment, especially childhood sexual abuse (e.g., Smolak & Murnen, 2002). Recent meta-analyses provided evidence for associations between multiple types of childhood maltreatment with eating disorders (Caslini et al., 2016; Molendijk et al., 2017; Pignatelli et al., 2017). Molendijk and colleagues (2017) further found associations between childhood maltreatment with eating disorder severity and psychiatric comorbidity. Other forms of trauma exposure have also been linked to DE (e.g., physical and sexual assault, motor vehicle accidents, bullying; Copeland et al., 2015; Dansky, Brewerton, Kilpatrick, & O'Neil, 1997; Mitchell, Mazzeo, Schlesinger, Brewerton, & Smith, 2012). Although these studies provide important foundational knowledge about the connection between trauma exposures and DE, they have largely been limited by cross-sectional designs and retrospective recall of trauma histories (Trottier & MacDonald, 2017).

Longitudinal Associations Between Trauma and DE

Prospective analyses of trauma exposure and DE are limited. In the first longitudinal cohort study of trauma and DE, Johnson, Cohen, Casen, and Brook (2002) analyzed the association of childhood trauma and subsequent DE among a sample of youth from upstate New York. Childhood sexual abuse and physical neglect predicted an eating disorder diagnosis or endorsing purging orstrict dieting in adolescence or young adulthood, even after controlling for childhood DE. Other studies have combined prospective assessments of DE with retrospective reports of childhood trauma. For example, Sanci and colleagues (2008) prospectively assessed DE symptoms throughout adolescence in a cohort of Australian youth and later asked participants if they had experienced sexual abuse during childhood (i.e., prior to designated assessments of DE). Results showed that exposure to two or more episodes of childhood sexual abuse was associated with later onset of bulimia nervosa (BN) symptoms among girls. In a longitudinal assessment of the effects of combat exposure, Jacobson and colleagues (2008) found that this trauma exposure predicted onset of DE among women military personnel. Taken together, these studies suggest multiple forms of trauma exposure may precede DE, but further research in diverse samples is needed to confirm these relations.

Associations of PTSD Symptoms with DE

Parsing the effects of trauma exposure from PTSD symptoms on DE represents an important step for the field (e.g., Brewerton, 2007; Trottier & MacDonald, 2017; Trottier, Wonderlich, Monson, Crosby, & Olmsted, 2016). PTSD symptoms have been found to predict subsequent binge eating among various adult populations (e.g., community samples, military personnel, women who identify as sexual minorities; Cronce, Bedard-Gilligan, Zimmerman, Hodge, & Kaysen, 2017; Mason et al., 2017; Mitchell, Porter, Boyko, and Field, 2016). There is growing evidence for PTSD symptoms as a mediator of trauma exposure and DE, although much of this is based on cross-sectional data (Dubosc et al., 2012; Holzer et al., 2008; Huston et al., 2019).

It is unclear how or whether specific symptoms of PTSD relate differentially to DE. Trottier, Wonderlich, Monson, Crosby, and Olmsted (2016) proposed that DE may function to facilitate avoidance of trauma-related affect and cognitions or downregulate negative arousal among people with PTSD. They note that the behaviors may also stem from negative self-focused cognitions and affect associated with PTSD; others have hypothesized that disordered eating may reflect body-focused shame or responses to negative cognitions about control or safety among trauma survivors (Trim, Galovski, Wagner, & Brewerton, 2017). A network analysis of PTSD symptoms and DE identified heightened connectivity across several constructs: irritability and binge eating, nightmares and body dissatisfaction, and concentration difficulties associated with PTSD symptoms and concentration difficulties linked with distress over shape and weight (Vanzhula, Calebs, Fewell, & Levinson, 2019). The authors suggested that binge eating may function to regulate irritability (or provoke irritability), whereas nightmares related to trauma experiences may increase body dissatisfaction if individuals ruminate on the potential role of body shape and size in their victimization. Substantial work remains to confirm these interpretations and clarify associations both cross-sectionally and over time, however.

Current Study

Although researchers have noted associations of trauma exposures (especially childhood maltreatment) with DE, there are limited examples of longitudinal studies that prospectively examine a range of trauma exposures, PTSD, and DE symptoms. Further, much of the extant literature has relied on retrospective recall of trauma exposures and DE symptoms. Prospective assessment of trauma exposure, PTSD symptoms, and DE is thus critical to clarifying the role of trauma exposure and PTSD symptoms in subsequent DE.

Accordingly, we had had three main aims in the current study. First, we sought to confirm previous research on the association of trauma exposure and DE symptoms by evaluating their associations within childhood and adulthood in a single cohort. We focused on DE symptoms rather than clinical eating disorders due to the greater prevalence of the former in both our sample and the general population. We hypothesized that DE symptoms would be heightened among those exposed to sexual or violent trauma in comparison to no trauma exposure, given extensive evidence for cross-sectional associations of such trauma exposures and DE in the literature. Second, we sought to assess the longitudinal association of trauma exposure with DE. We did this both across age groups (i.e., testing the association of childhood trauma exposure with adulthood DE after controlling for childhood DE) and within age group (i.e., using trauma exposure at one observation to predict DE symptoms at the next observation within childhood and adulthood periods). We hypothesized that sexual or violent trauma exposures in childhood would each significantly relate to DE in adulthood after controlling for baseline DE. We also hypothesized that exposure to sexual or violent trauma at one wave would significantly relate to increased DE symptoms at the following wave during both developmental periods. Finally, we evaluated the association of PTSD symptoms (examined in total and by symptom cluster) with subsequent DE. We anticipated significant associations of PTSD with DE but had no a priori hypotheses about the associations of each PTSD symptom cluster on DE symptoms.

Methods

Participants

Participants included individuals (n = 1420, 51.1% female) recruited as part of the Great Smoky Mountains Study, a population-based longitudinal cohort study examining trajectories of emotional and behavioral concerns from youth to adulthood (Costello et al., 1996). The study began in 1992 and continues today. The screening and recruitment process are described in detail in Costello et al. (1996) and Costello, Copeland, and Angold (2016). Briefly, a sample of 4,500 youth enrolled in public schools across 11 counties in western North Carolina was randomly selected to participate in an initial eligibility screening. Children whose parents or caregivers reported heightened levels of behavioral concerns were invited to participate in the full study. Ten percent of children who scored below this threshold were also randomly selected to participate in the study. Additionally, all American Indian youth attending reservation schools in the Qualla Boundary in North Carolina (n = 450) were invited to participate. At baseline, the sample was 64.51% Caucasian, 5.56% African American, 0.21% Asian American, 0.42% Hispanic, and 22.75% American Indian. Less than one percent of the sample identified as more than one race (Costello et al.,

1996). Over the course of childhood, approximately one-third of participants lived below the federal poverty line during at least one assessment (Copeland, Angold, Shanahan, & Costello, 2015).

Procedures

Participants were recruited in 1993 at ages 9 (n = 508), 11 (n = 497), or 13 (n = 415). Trained study personnel interviewed both the youth and their parents at baseline and annually for four years (i.e., until the oldest participants were 16). The participants were then interviewed alone at ages 19, 21, 25, and 30. They completed 11,084 interviews over the course of the study, with at least 76 percent of participants completing interviews at any given assessment (Costello, Copeland, and Angold, 2016). Childhood trauma exposure was unrelated to dropout during adulthood (Copeland et al., 2018). All interviews were audiotaped, and interviewers participated in regular reviews of randomly selected tapes to ensure fidelity. The study protocol and consent forms for each assessment were approved by the Duke University Medical Center Institutional Review Board, and participants received payment for their time.

Measures

Disordered Eating—Participants were interviewed using the Child and Adolescent Psychiatric Assessment (CAPA; Angold et al., 1995) until age 16, followed by a version adapted for young adults (the Young Adult Psychiatric Assessment, YAPA; Angold & Costello, 2000). Parents were interviewed about their child's symptoms until the child was 16. Evaluation of the CAPA by multiple indices (e.g., prevalence of detected diagnoses, association with expected outcomes, etc.) support the test-retest reliability and construct validity of the instrument (Angold & Costello, 2000; Copeland, Wolke, Shanahan, & Costello, 2015). DE items remained the same across both measures, although the YAPA was not separately validated. Participants reported on eating behaviors and body image concerns over the three months prior to each observation. The instruments specifically assessed for the presence of each symptom of anorexia nervosa (AN) and bulimia nervosa (BN) in the Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV; American Psychiatric Association, 1994). We used clusters described in Copeland et al., 2015: AN symptoms, BN symptoms, and associated disordered eating (i.e., preoccupation with eating, sustained increased/decreased appetite). See Table 1 for items pertaining to each cluster. Items were coded as present if endorsed by either the youth or their parent. We examined each of these clusters separately throughout the analyses. AN items can be recoded to correspond to DSM-5 symptoms. In childhood, 0.1 percent of participants met DSM-5 criteria for AN (0.6% through adulthood). Substantially greater proportions of participants endorsed DE symptoms.

Trauma Exposure and Posttraumatic Stress Disorder—The CAPA/YAPA and parent interviews assessed for a range of traumas (e.g., exposure to actual or threatened death, severe injury, or sexual violence). Youth and parents were interviewed about the youth's lifetime and recent (i.e., past three months) trauma history at each assessment through age 16; thereafter, participants were asked about past three-month trauma exposure as well as any exposure since last assessment. We characterized trauma exposure using

categories defined by Copeland and colleagues (2018): violent (e.g., death of a loved one through violent means, physical abuse), sexual (e.g., rape, sexual abuse; violent sexual assaults were also categorized as sexual trauma), and non-sexual, non-violent (e.g., natural disaster).

Until age 16, the full PTSD module was only completed if the parent or child reported the presence of each PTSD symptom cluster (reexperiencing, hyperarousal, and avoidance) as assessed with three screening questions. This was done to minimize response burden. Due to concern that the screening questions might have been overly restrictive, at ages 19 and 21, the PTSD module was completed if the participants reported exposure to events that involved significant threat to themselves or others. The original screening structure (three screening questions assessing reexperiencing, hyperarousal, and avoidance) was re-adopted at ages 25 and 30.

Data Analytic Plan

We accounted for the stratified sampling design by applying sampling weights throughout the analyses; weights were inversely proportional to a participant's chances of selection. To assess the association of trauma exposure and DE symptoms in childhood and adulthood (Aim 1), we first summed each type of trauma exposure across the childhood observations (i.e., through age 16) and adulthood observations (i.e., ages 19, 21, 25 and 30). This generated a cumulative record of each form of trauma exposure during these periods. See Table 1 for full definitions of each trauma exposure and childhood and cumulative lifetime prevalence.

We used a two-step process to calculate the DE scores used in Aim 1. First, we dichotomized whether the participant endorsed each of the items assessed in a DE symptom cluster at each observation. For example, the AN cluster encompassed three items (see Table 1); AN scores at the observation level ranged from 0 to 3. We then formed an aggregate score across childhood observations by summing the number of items within that DE symptom cluster endorsed at any observation through age 16. Items endorsed at more than one observation were only counted once, consistent with other analyses based on GSMS data (e.g., Copeland, Wolke, Shanahan, & Costello, 2015). Aggregate AN scores across childhood observations thus also ranged from 0 to 3. We repeated the process for aggregating across adult observations (i.e., ages 19, 21, 25, and 30). We used the same procedure to calculate scores for BN symptoms in childhood and adulthood (both observation-level and aggregate scores ranged from 0 to 3) and associated DE symptoms (observation-level and aggregated scores again ranged from 0 to 3). We computed mean ratios for each DE symptom cluster in childhood and adulthood, comparing scores for those exposed to each type of trauma versus those with no trauma exposure.

Aim 2 was to assess the longitudinal associations of trauma exposure with DE across and within developmental periods. To assess associations across developmental period, we regressed the aggregated scores for each DE cluster in adulthood on childhood trauma exposure. We examined each form of childhood trauma exposure in a different model. All models were adjusted for sex, race, childhood SES, and the aggregated score for the same DE symptom cluster in childhood.

We then used generalized estimating equations (GEE) with robust variance (sandwich) estimators to assess the associations of trauma exposure at one observation in childhood with the total score for each DE symptom cluster at the next observation (through age 16). As AN, BN, and associated DE symptoms were highly right-skewed, we used the Poisson distribution to account for their non-normality. We repeated this process for the adulthood assessments, using trauma exposure recorded at age 19 to predict DE symptoms at age 21, etc. Models controlled for DE symptoms at the preceding observation as well as sex, race, and childhood socioeconomic status (SES).

Aim 3 was to explore the incremental utility of specific PTSD symptom clusters on subsequent DE. We first used GEE with robust variance estimates to examine the association of endorsing any PTSD symptoms (versus no symptoms) with later DE, as we could conduct this analysis in both developmental periods by using childhood responses to the symptom cluster screening questions. We then focused on adult observations to test the association of each PTSD symptom cluster (i.e., painful recall, avoidance, hyperarousal) with DE at the next observation, controlling for DE at the reference wave, sex, race/ethnicity, and SES.

All analyses were conducted in SAS; regression models for Aims 2 and 3 were implemented using PROC GENMOD.

Results

Aim 1: Assess Associations between Trauma and DE in Childhood and Adulthood

The top section of Table 2 shows mean childhood DE symptoms by trauma type, along with the association of trauma exposure with DE, adjusted for sex, race/ethnicity, and SES. All associations are shown as mean ratios, with no trauma exposure as the reference group. Unexpectedly, each form of trauma exposure in childhood was associated with increased DE symptoms during childhood. The bottom of Table 2 shows the associations between lifetime trauma exposures and DE symptoms in adulthood, adjusted for sex, race/ethnicity, and SES. As compared to those with no trauma exposure, participants reporting any lifetime violent trauma exposure reported significantly higher levels of each DE outcome in adulthood. Both exposures to lifetime sexual trauma and non-violent, non-sexual trauma were associated with higher levels of BN symptoms, appetite changes and preoccupation with food in adulthood.

Aim 2: Assess Longitudinal Associations of Trauma with DE

Table 3 shows the longitudinal association of childhood trauma exposure with DE symptoms in adulthood, controlling for sex, race, SES, and childhood DE. Any childhood trauma exposure was linked to increased appetite changes and preoccupation with food in adulthood. Of the specific categories of trauma exposure, however, only non-sexual, non-violent childhood trauma had significant longitudinal associations with adulthood DE symptoms (although violent trauma exposure trended toward significance).

Table 4 shows the associations of exposure to each form of trauma with DE symptoms at the following observation within childhood and adulthood. Analyses were adjusted for DE symptoms at the previous observation, along with sex, race, and SES. The top section shows

relations across observations within childhood; the bottom section shows relations across adulthood observations. Violent trauma exposure had significant longitudinal associations with BN symptoms and associated DE at the next wave in childhood (versus no trauma). Non-sexual, non-violent trauma exposure also showed a significant longitudinal association with associated DE symptoms in childhood. The association of sexual trauma trended toward significance for childhood BN symptoms. Unexpectedly, no type of adult trauma exposure had significant associations with any subsequent DE symptom in adulthood.

Aim 3: Assess Longitudinal Associations of PTSD Symptoms on DE

Across childhood observations, endorsing any PTSD symptom was not significantly related to any form of DE at the subsequent wave after controlling for DE at the prior observation, sex, race, and SES. Due to the low prevalence of PTSD symptoms in childhood, we could not parse associations of specific PTSD symptom clusters on DE during this period.

Across adulthood observations, endorsing any PTSD symptom was significantly associated with increased BN symptoms at the subsequent wave, again controlling for previous DE, sex, race/ethnicity, and SES. (See Table 5). When we examined each cluster individually, only associations with painful recall were significant. The association with hyperarousal trended toward significance. PTSD symptoms had no significant longitudinal associations with symptoms of AN or associated DE.

Discussion

The unique attributes of the Great Smoky Mountains Study allowed for prospective assessment of trauma exposure on subsequent DE, extending a literature that has largely focused on cross-sectional associations of these constructs. In the current study, we aimed to confirm previously documented associations of different trauma exposures with DE in a large community-based sample, as well examine the longitudinal associations of trauma exposure and PTSD specifically with DE. First, we observed associations of multiple forms of trauma exposure to DE outcomes in childhood and adulthood. Second, we found evidence for longitudinal associations of trauma exposure with DE among children but limited evidence for this relation among adults. Conversely, PTSD symptoms had significant associations with subsequent DE among adults, but we did not observe this relation in childhood.

The association of DE (particularly binge-purge symptoms) with exposure to both violent and sexual trauma has been documented among children, adolescents, and adults (e.g., Dansky, Brewerton, Kilpatrick, & O'Neil, 1997; Smolak & Murnen, 2002). Consistent with this, we saw significant associations of violent trauma with each DE symptom cluster assessed in childhood and adulthood. Sexual trauma was linked with all outcomes assessed in childhood but with only BN symptoms, sustained appetite changes, and preoccupation with eating in adulthood. This may further suggest specific associations of sexual trauma with BN symptoms (i.e., versus AN symptoms) but could also reflect lower frequencies of lifetime sexual trauma and AN symptoms in this sample.

We did not anticipate the association of non-violent, non-sexual trauma in childhood with multiple forms of DE in that developmental period or the relation of cumulative lifetime exposure to such trauma with some forms of DE in adulthood. We note that non-violent, non-sexual trauma as defined here included instances of observing death or injury to others. It thus may have inadvertently captured the impact of witnessing violence (e.g., family violence).

Contrary to hypothesis, childhood exposure to non-sexual, non-violent trauma was related to some forms of DE in adulthood, whereas we observed no significant relations between violent or sexual trauma in childhood and adulthood DE. These results contrast slightly with those of Johnson, Cohen, Kasen, and Brook (2002), which found associations of childhood sexual abuse and physical neglect with both DE and diagnosable eating disorders in adulthood. Differences in follow-up period (i.e., through age 30 here versus age 22 in the earlier work) may have contributed to the discrepancy. We also controlled for childhood DE symptoms in our analyses; the impact of childhood trauma exposures on DE in adulthood may be mediated by childhood DE. Further research should continue to target high-risk groups to prospectively assess the relation of maltreatment and other childhood trauma exposures on adulthood DE.

Within developmental periods, trauma exposures but not PTSD symptoms had significant associations with DE in childhood. The reverse was true in adulthood. This suggests possible differential pathways from trauma exposure to DE by developmental time period. Among youth, mood and anxiety symptoms may be even more common following trauma exposure than PTSD symptoms (Copeland, Keeler, Angold, & Costello, 2007). Future research should test whether these mediate the relation between trauma exposure and DE in childhood and adolescence. PTSD symptoms may represent a more important target among adults. Although we cannot rule out that the longer timeframe between assessments in adulthood impacted our findings, our results echo previous work showing that BN was more prevalent among adult women sexual assault survivors with a lifetime history of PTSD versus those who did not experience PTSD (Dansky, Brewerton, Kilpatrick, & O'Neil, 1997).

Trim, Galovski, Wagner, & Brewerton (2016) suggested potential functions of DE symptoms among individuals with PTSD, including downregulating hyperarousal or responding to negative cognitions about shape and weight stemming from trauma experiences. We found significant relations for painful recall (which encompasses distress associated with this reexperiencing) and trending relations for hyperarousal, suggesting that BN symptoms may function to counter-act intrusive traumatic memories and to downregulate arousal. If confirmed, these initial results support the importance of examining specific PTSD symptom clusters for differential effects on DE.

A key strength of this study is its use of a diverse, community-based sample that participated in repeated assessments over a 16-year follow-up period. Trained interviewers gathered the data, and childhood data collection included parent report, likely mitigating some of the bias inherent in self-report. We were also able to clarify the relation of trauma exposure versus PTSD symptoms on DE and examine the relations of specific PTSD symptom clusters with DE in adulthood.

The community-based sample also reflects a limitation, however, in that prevalence of DE symptoms was relatively low. Such low prevalence (and resulting reduction in power) is consistent with the relatively low base rate of DE symptoms in the population as a whole and reflects the challenge of completing population-based, longitudinal research on DE. Although trauma exposure was unfortunately widespread in the sample, PTSD symptoms were less common, particularly during childhood. It is possible that the prevalence of PTSD symptoms in childhood was underestimated due to the screening process for such symptoms. Additionally, both PTSD and DE symptoms were assessed using DSM-IV criteria rather than DSM-5, and neither the CAPA or YAPA disordered eating modules were independently validated. Replication of the findings using established DE and PTSD measures for DSM-5 and in clinical simples are critical next steps. Another important limitation was that time between assessments and screening criteria for the full PTSD module differed from childhood to adulthood observations. We thus cannot directly compare the findings across developmental periods.

Despite these limitations, the study provides new longitudinal evidence for associations of trauma exposure and PTSD symptoms with later DE. The findings also point to the impact that PTSD symptoms have on symptoms of BN among adults. Continued efforts to understand the cognitive, affective, and interpersonal mechanisms connecting trauma exposure to specific DE symptoms among children and adults will be critical to developing prevention and clinical intervention techniques that are both developmentally sensitive and effective.

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Clinical Implications

- Age-related differences emerged between trauma and disordered eating.
- Both sexual and non-sexual traumas were linked to childhood disordered eating.
- Posttraumatic stress disorder symptoms did not predict childhood disordered eating.
- Posttraumatic stress disorder symptoms predicted adult bulimia nervosa symptoms.
- Reexperiencing symptoms specifically predicted adult bulimia nervosa symptoms.

Table 1

Frequency of trauma exposures and disordered eating symptoms reported in childhood, through adulthood

Trauma Exposure

Category	Example exposures	Childhood Frequency[1] (*N*, %)	Cumulative Lifetime Frequency[1] (*N*, %)
Any	Any form of trauma exposure	1007 (67.7)	1222 (86.1)
Sexual	Rape, sexual abuse	184 (11.0)	313(20.6)
Violent	Physical abuse, violent death of loved one, experiencing physical violence, war or terrorism, captivity	419 (25.0)	692 (45.4)
Other	Witnessing trauma with potential to cause death or injury, learning about a loved one's trauma exposure, natural disaster, fire, serious unintentional injury, diagnosis of serious illness, exposure to noxious agent	840 (56.5)	1074 (75.8)

Frequency of Eating Disorder Symptoms

Category	Definition	Childhood Frequency (*N*, %)	Cumulative Lifetime Frequency (*N*, %)
Any AN symptom	Endorsed any of the following: • Underweight • Fear of gaining weight • Distorted body image	274 (19.7)	375 (26.5)
Any BN symptom	Endorsed any of the following: • Binge eating • Any attempts to reduce weight (via self-induced vomiting, dieting, excessive exercise) • Overconcern with body image	673 (42.2)	792 (50.4)
Any Associated DE symptom	Endorsed any of the following: • Sustained increased appetite • Sustained decreased appetite • Preoccupation with food/eating	966 (69.8)	1096 (78.5)

Note. All *N*s are unweighted. Categories are consistent with those outlined in Copeland et al. (2015).

[1]Dichotomous variable based on whether participants reported exposure at any available observation during the time period.

Table 2

Associations of trauma exposure and DE symptoms by age group and trauma type

	No trauma *M* (*SD*)	Any Trauma *M* (*SD*)	MR (95% CI)	Sexual Trauma *M* (*SD*)	MR (95% CI)	Violent Trauma *M* (*SD*)	MR (95% CI)	Other Trauma Exposure *M* (*SD*)	MR (95% CI)
Childhood Trauma with Childhood DE Symptoms									
Mean AN sx	0.12 (0.36)	0.25 (0.50)	1.9** (1.2–3.0)	0.40 (0.59)	2.3** (1.3–4.0)	0.26 (0.48)	2.0* (1.2–3.5)	0.26 (0.50)	1.9** (1.2–3.0)
Mean BN sx	0.35 (0.59)	0.58 (0.72)	1.7*** (1.3–2.2)	0.69 (0.71)	1.6** (1.1–2.3)	0.63 (0.63)	1.8*** (1.3–2.4)	0.60 (0.69)	1.7*** (1.3–2.2)
Mean Associated sx	0.61 (0.57)	0.84 (0.54)	1.4*** (1.2–1.6)	0.78 (0.58)	1.4** (1.1–1.9)	0.80 (0.51)	1.4** (1.1–1.6)	0.85 (0.53)	1.4*** (1.2–1.6)
Cumulative Lifetime Trauma with Adulthood DE Symptoms[1]									
Mean AN sx	0.10 (0.30)	0.18 (0.50)	1.6 (0.9–2.7)	0.34 (0.69)	1.9 (0.9–3.9)	0.23 (0.54)	1.9* (1.0–3.5)	0.16 (0.47)	1.4 (0.8–2.5)
Mean BN sx	0.20 (0.43)	0.39 (0.61)	1.8** (1.2–2.6)	0.58 (0.72)	1.9 (1.2–2.9)**	0.36 (0.58)	1.7** (1.1–2.5)	0.40 (0.62)	1.8** (1.3–2.8)
Mean Associated sx.	0.28 (0.45)	0.44 (0.55)	1.5** (1.1–2.0)	0.64 (0.56)	1.8*** (1.3–2.7)	0.52 (0.58)	1.7*** (1.3–2.4)	0.46 (0.56)	1.5** (1.1–2.0)

Note. AN = Anorexia nervosa, Associated symptoms = Sustained appetite change, preoccupation with food/eating, BN = Bulimia nervosa, DE = Disordered eating, MR = Mean ratio, Sx = Symptoms. Values adjusted for sex, race, and family SES. Reference group for all analyses is no trauma.

[1] Dichotomous variable based on reported exposure at any available observation.

* $p < .05$

** $p \leq .01$

*** $p < .001$.

Table 3

Association of childhood trauma exposure with adult DE symptoms, by trauma type

	No trauma *M* (*SD*)	Any Trauma *M* (*SD*)	MR (95% CI)	Sexual Trauma *M* (*SD*)	MR (95% CI)	Violent Trauma *M* (*SD*)	MR (95% CI)	Other Trauma Exposure *M* (*SD*)	MR (95% CI)
Mean AN sx	0.15 (0.40)	0.17 (0.46)	1.1 (0.7–1.9)	0.16 (0.34)	0.8 (0.4–1.7)	0.19 (0.48)	1.2 (0.6–2.5)	0.16 (0.45)	1.1 (0.6–1.9)
Mean BN sx	0.29 (0.57)	0.35 (0.56)	1.3 (0.9–1.7)	0.41 (0.55)	1.2 (0.8–1.8)	0.34 (0.49)	1.2 (0.8–1.7)	0.35 (0.56)	1.2 (0.9–1.7)
Mean Assoc. sx	0.31 (0.53)	0.43 (0.52)	1.4* (1.0–1.8)	0.41 (0.49)	1.1 (0.8–1.6)	0.43 (0.49)	1.3† (1.0–1.8)	0.44 (0.53)	1.4* (1.0–1.9)

Note. AN = Anorexia nervosa, Associated symptoms = Sustained appetite change, preoccupation with food/eating, BN = Bulimia nervosa, DE = Disordered eating, MR = Mean ratio, Sx = Symptoms. Values adjusted for sex, race, and family SES. Reference group for all analyses is no trauma.

† p = .05

* $p < .05$

Table 4

Longitudinal association of trauma exposure with disordered eating symptoms across waves, by trauma type and developmental period

	Cumulative Trauma MR	95% CI	Sexual Trauma MR	95% CI	Violent Trauma MR	95% CI	Other Trauma Exposure MR	95% CI
				Childhood DE				
AN sx	1.1	0.9–1.3	1.0	0.6–1.6	1.4	0.8–2.3	1.0	0.7–1.4
BN sx	1.1	1.0–1.2	1.4†	1.0–1.8	1.4**	1.1–1.7	0.9	0.7–1.1
Associated sx	1.1**	1.0–1.2	0.9	0.7–1.3	1.3*	1.0–1.6	1.2*	1.0–1.3
				Adulthood DE				
AN sx	1.0	0.7–1.6	0.5	0.2–1.7	0.7	0.3–1.6	1.1	0.7–1.8
BN sx	1.1	0.9–1.3	0.9	0.5–1.6	1.1	0.7–1.6	1.1	0.8–1.5
Associated sx.	1.1	0.9–1.3	0.9	0.7–1.3	0.9	0.6–1.3	1.2	0.9–1.6

Note. AN = Anorexia nervosa, Associated symptoms = Sustained appetite change, preoccupation with food/eating, BN = Bulimia nervosa, DE = Disordered eating, MR = Mean ratio, Sx = Symptoms. Values adjusted for DE symptoms at previous observation, sex, race, and family SES. Reference group for all analyses is no trauma.

† p = .05

* $p < .05$

** $p \leq .01$.

Table 5

Longitudinal associations of DSM-IV PTSD symptoms with DE in adulthood

	Any PTSD Sx		Painful Recall		Avoidance		Hyperarousal	
	MR	**95% CI**	**MR**	**95% CI**	**MR**	**95% CI**	**MR**	**95% CI**
AN sx	1.0	0.7–1.3	1.0	0.5–1.8	1.1	0.4–2.8	1.0	0.5–2.0
BN sx	1.1 *	1.0–1.2	1.2 *	1.0–1.5	1.3	0.9–2.0	1.3 †	1.0–1.6
Associated sx.	1.0	0.9–1.2	1.0	0.8–1.3	1.2	0.7–1.8	1.0	0.8–1.4

Note. AN = Anorexia nervosa, Associated symptoms = Sustained appetite change, preoccupation with food/eating, BN = Bulimia nervosa, DE = Disordered eating, MR = Mean ratio, PTSD = Posttraumatic stress disorder, Sx = Symptoms. Values adjusted for DE symptoms at previous wave, sex, race, and family SES.

† $p = .05$

* $p < .05$.