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Post traumatic stress disorder in anorexia nervosa

Mae Lynn Reyes-Rodríguez, Ph.D.[1], **Ann Von Holle, M.S.**[1], **T. Frances Ulman, Ph.D.**[1], **Laura M. Thornton, Ph.D.**[1], **Kelly L. Klump, Ph.D.**[2], **Harry Brandt, M.D.**[3], **Steve Crawford, M.D.**[3], **Manfred M. Fichter, M.D.**[4], **Katherine A. Halmi, M.D.**[5], **Thomas Huber, M.D.**[6], **Craig Johnson, Ph.D.**[7], **Ian Jones, M.D.**[8], **Allan S. Kaplan, M.D., F.R.C.P. (C)**[9,10,11], **James E. Mitchell, M.D.**[12], **Michael Strober, Ph.D.**[13], **Janet Treasure, M.D.**[14], **D. Blake Woodside, M.D.**[9,11], **Wade H. Berrettini, M.D.**[15], **Walter H. Kaye, M.D.**[16], and **Cynthia M. Bulik, Ph.D.**[1,17]

[1] Department of Psychiatry, University of North Carolina, Chapel Hill, NC

[2] Department of Psychology, Michigan State University, East Lansing, MI

[3] Department of Psychiatry, University of Maryland School of Medicine, Baltimore, MD

[4] Klinik Roseneck, Hospital for Behavioral Medicine, Prien and University of Munich (LMU), Munich, Germany

[5] New York Presbyterian Hospital-Westchester Division, Weill Medical College of Cornell University, White Plains, NY

[6] Klinik am Korso, Bad Oeynhausen, Germany

[7] Eating Recovery Center, Denver, CO

[8] Department of Psychological Medicine, University of Birmingham, United Kingdom

[9] Department of Psychiatry, The Toronto Hospital, Toronto, Canada

[10] Center for Addiction and Mental Health, Toronto, Canada

[11] Department of Psychiatry, Toronto General Hospital, University Health Network, Toronto, Canada

[12] Neuropsychiatric Research Institute and Department of Clinical Neuroscience, University of North Dakota School of Medicine and Health Sciences, Fargo, North Dakota

[13] Department of Psychiatry and Biobehavioral Sciences, David Geffen School of Medicine, University of California at Los Angeles, Los Angeles, CA

[14] Department of Academic Psychiatry, Kings College London, London, United Kingdom

[15] Department of Psychiatry, University of Pennsylvania, Philadelphia, PA

[16] Department of Psychiatry, University of California at San Diego, San Diego, CA

[17] Department of Nutrition, University of North Carolina, Chapel Hill, NC

Abstract

Objective—Comorbidity among eating disorders, traumatic events, and post traumatic stress disorder (PTSD) has been reported in several studies. The main objectives of this study were to describe the nature of traumatic events experienced and to explore the relation between PTSD and anorexia nervosa (AN) in a sample of women.

Correspondence to: Dr. Bulik, Department of Psychiatry, University of North Carolina at Chapel Hill, 101 Manning Drive, CB #7160, Chapel Hill, NC 27599-7160, Voice: (919) 843 1689 Fax: (919) 843 8802, cbulik@med.unc.edu.

Methods—Eight hundred twenty-four participants from the National Institutes of Health funded Genetics of Anorexia Nervosa Collaborative Study were assessed for eating disorders, PTSD, and personality characteristics.

Results—From a final sample of 753 women with AN, 13.7% (n=103) met DSM-IV criteria for PTSD. The sample mean age was 29.5 years (SD=11.1). In pairwise comparisons across AN subtypes, the odds of having a PTSD diagnosis were significantly lower in individuals with restricting AN (RAN) than individuals with purging AN without binge eating (PAN) (OR=0.49, 95% CI=0.30, 0.80). The majority of participants with PTSD reported the first traumatic event before the onset of AN (64.1%, n=66). The most common traumatic events reported by those with a PTSD diagnosis were sexual related traumas during childhood (40.8%) and during adulthood (35.0%).

Conclusions—AN and PTSD do co-occur and traumatic events tend to occur prior to the onset of AN. Clinically, these results underscore the importance of assessing trauma history and PTSD in individuals with AN and raise the question of whether specific modifications or augmentations to standard treatment for AN should be considered in a subgroup to address PTSD-related psychopathology.

Keywords

PTSD; anorexia nervosa; trauma; prevalence; comorbid; epigenetic

Introduction

Associations among eating disorders, traumatic events, and post traumatic stress disorder (PTSD) have been reported in several studies (1–3). The prevalence of PTSD in eating disorder samples has ranged widely from 1% to 52% (1, 2, 4, 5). The breadth of estimates might reflect differences in assessment and recruitment methods, criteria used to define PTSD, and whether lifetime or current PTSD was assessed. The National Women's Study reported a lifetime prevalence of PTSD in 36.9% of women with bulimia nervosa (BN), in 21.0% of women with binge eating disorder (BED), and 11.8% in women with no eating disorder (1). The prevalence of PTSD in clinical samples of individuals with anorexia nervosa (AN) has been estimated at 10% (5) and 47% (4). Furthermore, some studies have found that traumatic events are more commonly associated with BN and AN binge-purge type than with AN restricting type (6, 7). Typically, the lifetime prevalence of PTSD is lower in individuals with eating disorders ascertained from the community than those ascertained in inpatient treatment facilities (1, 3).

Somewhat more challenging is characterizing and quantifying the frequency and nature of traumatic events in individuals with eating disorders because the definition and classification of traumatic events vary considerably across studies. Broadly, the occurrence of childhood sexual abuse (1, 6, 8, 9), emotional abuse (10), accidents, and interpersonal loss or separation (11) have been reported in individuals with eating disorders. Understanding the relation among traumatic events, PTSD, and eating disorder symptoms could assist with refining theories of shared genetic, epigenetic, and neural processes across disorders, leading to the development of testable hypotheses.

The current study represents a secondary data analysis of the National Institute of Mental Health (NIMH)-funded Genetics of Anorexia Nervosa Collaborative (GAN) Study. This study presents a comprehensive description of PTSD in individuals with AN and explores the relation between PTSD and AN. The four aims of the present study were: 1) to assess the prevalence of PTSD in a sample of women with AN by AN subtype; 2) to document the frequency and nature of traumatic events associated with PTSD in women with AN by AN

subtype; 3) to present the time course of AN onset relative to the first reported traumatic event in those participants with PTSD, by AN subtype; and 4) to evaluate the relation between PTSD and various defining characteristics of AN (i.e., BMI, anxiety, concern over mistakes, age of menarche, obsessions, harm avoidance).

Methods

Participants

Participants were from the NIH funded Genetics of Anorexia Nervosa Collaborative Study with data collection occurring between January 2003 and June 2007. The complete methodology for this investigation has been presented in a separate publication (12). All participants provided informed consent and the study was approved by the local Institutional Review Boards/Ethics Boards. Multiplex families were ascertained through a proband with AN who provided permission to contact other willing affected relatives and parents in accordance with Institutional Review Board requirements of each participating site. A brief screen was administered to probands and affected relatives to establish an initial diagnosis of AN. If screened eligible, probands and affected relatives then underwent an extensive diagnostic assessment to confirm the diagnosis of AN and to establish all other study inclusion and exclusion criteria.

To be considered for the study, probands were male or female, age 16 or older, ill or recovered. They must have met a lifetime diagnosis of modified DSM-IV AN, with or without amenorrhea, at least 3 years prior to study entry, and by age 45. Low weight for probands was defined as a body mass index (BMI) at or below 18 kg/m^2 for females and 19.6 kg/m^2 for males. These BMI values correspond to the 5^{th} percentile BMI values of the National Health and Nutrition Examination Survey (13) epidemiological sample of females and males, respectively, for the average age range (27–29 years) of probands in our previous studies. All probands were required to have at least one first, second, or third degree relative with AN, excluding parents and MZ twins, who was willing to participate in the study. All participants had to speak either English or German. Potential probands were excluded from the study if they had any of the following: regular binge eating, defined as at least twice a week for at least three months; a history of severe central nervous system trauma; psychotic disorders or developmental disability; or a medical, neurological or substance use disorder that could confound the diagnosis of AN or interfere with their responding cogently to assessments. Individuals with a maximum lifetime BMI exceeding 30 kg/m^2 were excluded.

All affected relatives were required to meet the same inclusion criteria as probands with the exception that regular binge eating was permitted and they need not have met AN criteria three years prior to the study. They were, however, required to have had a minimum duration of at least three months at low weight as outlined above. Affected relatives could have had an additional lifetime diagnosis of BN. If a family had a proband and an affected paired relative, additional affected relatives with the diagnosis of AN, BN, or eating disorder not otherwise specified (EDNOS) were permitted into the study.

Assessments

Clinical assessments were conducted by masters or doctoral level psychologists or other mental health specialists. Training for all interviewers included 4-day centralized training, didactic sessions with a recorded interview sample, discussion, role playing, and follow-up diagnostic consensus teleconferences. All interviewers had to be certified prior to conducting interviews. Interviewers from the German site were certified by a native German speaking psychiatrist trained in all clinical interviews. Monthly consensus calls were

conducted to discuss diagnostic issues. Final eating disorders diagnoses were reviewed by each site's principal investigator.

Eating Disorder Pathology—To assess eating disorder pathology, three clinical interviews were used. To establish AN diagnosis and to assess inclusion and exclusion criteria, we used an expanded modified version of Module H of the Structured Clinical Interview for Axis I Disorders (SCID-I) (14, 15). The Structured Interview for Anorexia Nervosa and Bulimic Syndromes (SIAB) (16–17) was administered to confirm the eating disorder diagnosis and to collect additional information regarding core eating disorder behaviors. The Yale-Brown-Cornell Eating Disorder Scale (18)(YBC-EDS) was administered to evaluate current and lifetime eating disorder severity and to assess specific core obsessions and compulsions related to eating disorders. AN subtypes were defined as restricting type AN (RAN) characterized by restrictive behaviors, no binge eating or purging behaviors reported; AN with purging and without binge eating (PAN); AN with binge eating, with or without purging (BAN); and individuals with a lifetime diagnosis of both AN and BN (ANBN).

Post Traumatic Stress Disorder—The SCID-I was used to establish a lifetime diagnosis of PTSD according to DSM-IV criteria. Participants completed the PTSD section if they indicated that they had experienced a traumatic event and their response to the event involved intense fear, helplessness, or horror. Information about each traumatic event and the age at which each event occurred was collected.

Personality and Symptom Assessments—Data for lifetime lowest illness-related BMI and age at menarche were obtained from the SIAB. Trait anxiety was assessed using the 40-item self-report questionnaire: The State-Trait Anxiety Inventory Form Y (STAI-Y) (19). The concern over mistakes subscale of The Multidimensional Perfectionism Scale (20) was used. This subscale reflects the tendency to react negatively to mistakes. The anticipatory worry and pessimism vs. uninhibited optimism subscale of harm avoidance (HA1) was assessed using the Temperament and Character Inventory (TCI) (21, 22). This subscale was selected to explore potential traits related to eating disorders symptoms and temperament and personality (23). Food obsessions were evaluated using the worst total score from The Yale-Brown-Cornell Eating Disorder Scale (YBC-EDS), which assesses the severity and types of core obsessions and compulsions specific to eating disorders (18). Obsessions were assessed using the obsessions subscale of the Yale-Brown Obsessive Compulsive Scale (Y-BOCS), a semi-structured interview designed to rate the presence and severity of obsessive thoughts and compulsive behaviors typically found among individuals with obsessive-compulsive disorder (24).

Statistical Analysis

All analyses were performed using SAS/STAT software, Version 9.1 of the SAS System for Windows (25). Preliminary analysis assessing PTSD prevalence across centers found no significant differences (chi-sq statistic=13.2, df=9, p-value=0.16), thus center was not included in any analysis as a confounding variable. Descriptive analyses were conducted to characterize the sample in regard to PTSD prevalence across AN subtypes; counts and types of traumatic events across AN subtypes; and timing of PTSD relative to AN onset. Using logistic regression analysis, odds of having a PTSD diagnosis were calculated, comparing pairs of AN subtypes. To test differences in counts of traumatic events across AN subtypes, we used a non-parametric Cochran-Mantel-Haenszel (CMH) chi-square test. Differences in the mean number of years from the first reported traumatic event to AN onset across subtypes were tested using an analysis of variance (ANOVA). Model diagnostics included

examination of residuals. All pairwise comparisons within the ANOVA included the Tukey-Kramer adjustment.

To estimate the relative risk of a PTSD diagnosis for characteristics associated with AN, we used a Poisson regression including a dichotomous PTSD diagnosis as a response variable and several covariates representing characteristics of AN: 1) lowest lifetime BMI; 2) trait anxiety; 3) harm avoidance; 4) concern over mistakes; 5) age at menarche; 6) obsessions (Y-BOCS); 7) worst food obsessions total score (YBC-EDS). In this model, the risk of PTSD is meant in a general sense as the probability of experiencing a PTSD event spanning times before and/or after initiation of the AN characteristics. Approximately 40% of the sample reported a zero for the Y-BOCS obsessions score, therefore the obsessions score was dichotomized to indicate a score of zero or greater than zero. Poisson regression with generalized estimating equations (GEE) is a method to estimate relative risks, which allows for more accurate variance estimates. Given that multiple family members participated, family status was used as a variable in determining the covariance matrix. Age at interview was also included as a confounding variable. We evaluated variance inflation scores of the seven predictor variables and found no evidence of collinearity (26–28).

Results

Participants

The initial sample comprised 824 participants. For the purposes of this study the following not mutually exclusive groups were removed from analyses: males (n=33); individuals with a diagnosis of PTSD but no description of a PTSD-related event (n=2); individuals with a missing PTSD diagnosis (n=21); individuals with missing eating disorder subtype information (n=1); and individuals who did not have a diagnosis of AN (n=17). After applying the exclusion criteria, 753 participants remained in the analysis sample with a mean age of 29.5 years (SD=11.1).

Prevalence of Lifetime PTSD

Table 1 presents the prevalence of PTSD diagnosis by AN subtype. Of the 753 individuals, 13.7% (n=103) met DSM-IV criteria for PTSD and 86.3% (n=650) reported either no event or insufficient symptoms to assign a possible diagnosis. In pairwise comparisons across AN subtypes, the odds of having a PTSD diagnosis were significantly lower in individuals with RAN than individuals with PAN (OR=0.49, 95% CI=0.30, 0.80). No other significant differences were found.

Traumatic Events and PTSD

Over one third of women [39.0% (n=294)] reported one or more traumatic events in their lifetime. The percentage of women with PTSD endorsing one or more traumatic event by AN subtype is presented in Table 2. No significant differences were found in the number of reported events across AN subtypes (CMH chi-square=0.59, degrees of freedom=1, $p<0.44$). Types of traumatic events experienced (see Table 3) are presented separately by the presence or absence of PTSD and by AN subtype. The most common traumatic events reported by those with a PTSD diagnosis were: childhood sexual abuse between the ages of 6 and 17 (n=42; 40.8%); sexual abuse or rape in adulthood (n=36; 35.0%); and death or illness of a family member or significant other (n=18; 17.5%).

Time course of AN onset relative to the age at first reported event in participants with PTSD

The majority of participants with PTSD reported the first traumatic event before the onset of AN (64.1%, n=66). The percentage of participants who experienced the first traumatic event

before AN, by AN subtype, is as follows: RAN=69.7% (n=23); PAN=60.0% (n=24); BAN=57.9% (n=11); and ANBN=72.7% (n=8). No significant pairwise differences were found between AN subtypes in mean time to AN onset from the first reported traumatic event (data not shown). For those with a PTSD diagnosis, the mean time in years (standard deviation) from the first traumatic event to AN onset was: RAN=8.0 (5.9); PAN=12.3 (6.9); BAN=7.8 (3.9); and ANBN=10.0 (4.5).

Association between PTSD and characteristics of AN

In the model estimating the relative risk of PTSD by AN characteristics, only food obsessions total score from the YBC-ED was significantly positively associated with the probability of PTSD (See Table 4). A one unit increase in the food obsessions total score resulted in a 6.7% increase in probability of PTSD ($p<0.006$). This positive association persisted after adjustment for age at interview ($p<0.003$). No other predictors were significantly associated with PTSD in the model.

Discussion

In our exploration of associations between lifetime PTSD and AN, the observed lifetime prevalence of PTSD (13.7%) in the present sample is lower than one previous report (47.0%) (4), but similar to a study of female British patients with AN (10.0%) (29). Similar to other studies (6, 7, 30), the prevalence of PTSD was higher in individuals with PAN than in individuals with RAN.

Differences in some personality traits among AN subtypes may explain differences in PTSD prevalence. For example, greater impulsivity has been associated with BAN and PAN subtypes compared with RAN (7). In addition, impulsivity has been associated with trauma (31) and PTSD (32). Some specific eating disorders behaviors (e.g., binge eating and purging) associated with impulsivity, could be mechanisms to avoid awareness of the trauma (31). Considering the AN characteristics studied, only greater food obsessions were associated with the presence of PTSD. Intrusive and obsessive thoughts have been identified as a reaction to traumatic experiences which enable the avoidance or inhibition of intense affect related to the trauma (33). Because impulsivity and obsessions related to trauma have been associated with treatment resistance (33), suicide risk (32), and self-destructive behaviors (31), the treatment of AN patients with comborbid PTSD may benefit from focused attention to address impulsivity and obsessive features that are associated with PTSD.

Consistent with other studies (5, 29, 34, 35), sexual-related traumas were the most commonly reported type of trauma in the current sample. However, the diversity of traumas reported by participants underscores the importance of assessing a wide range of traumatic events (5, 11). Childhood sexual abuse is associated with increased risk for both PTSD (36) and eating disorders (6); however, given that it is also associated with several other psychiatric disorders, childhood sexual abuse is generally considered to be a nonspecific risk factor for later psychopathology (6, 37, 38).

Although the nature of the relation between PTSD and eating disorders has not been fully elucidated, examining commonalities in neural processing of stressors may provide insight into shared mechanisms that drive symptom expression. One potential commonality may be aberrant fear conditioning (39). Individuals with PTSD show extreme fear conditioning (40) and delayed fear extinction (40–43). Similarly, Strober (39) theorizes that the core features of AN are influenced by the presence of extreme fear conditioning and a greater than typical resistance to fear extinction. Strober's theory remains to be tested empirically and behavioral evidence supporting the theory remains circumstantial (44–49).

Commonalities in aberrant fear conditioning between disorders may be associated with atypical adaption to stressors via the amygdala and HPA axis. In PTSD, hyperactivity in the amygdala plays a critical role in abnormal fear conditioning (50) and is also associated with emotion dysregulation (51). Although the association of fear conditioning and the amygdala within eating disorders has not been directly tested, evidence of atypical amygdala activity in AN exists. Miyake et al. (47) reported higher activation of the right amygdala when processing negative words associated with body image in individuals with RAN, PAN, and BAN compared with individuals with BN and control participants with no eating disorders. In PTSD, hyperactivity is found in the HPA axis compared with controls when presented with a stressor (48, 49, 52) and hyperactivity of the HPA axis is also seen in AN (48, 49, 53), although confounders related to the effects of starvation limit inferences about HPA functioning in AN. A fruitful avenue of future inquiry could be exploration of the role of the amygdala and HPA axis in fear conditioning and maintenance in both disorders and in their comorbid presentation.

Examining similarities in neural processing of aversive stimuli in individuals with AN and PTSD who have experienced a childhood trauma may help in the refinement of our understanding of comorbidity. Neurobiological research underscores the role of childhood trauma in the onset and/or maintenance of adult psychopathology (54–56) and genetic and epigenetic studies suggest that early life experiences can alter neural functioning in a way that increases vulnerability for the development of AN or PTSD in adulthood. Adverse experiences in childhood can lead to a more reactive amygdala and HPA axis, increasing the vulnerability for developing PTSD after experiencing a trauma in adulthood (57–60). Results of genetic association studies have implicated polymorphisms in the *CRHR1* and *FKBP5* genes as mediators of long term changes in the HPA axis and PTSD in individuals who have experienced childhood abuse (61–64). Gillespie et al., (61) synthesize this literature and suggest that individuals with “risk” gene variations combined with child maltreatment may be at increased risk for PTSD if faced with a trauma in adulthood. Whether similar gene × environment interactions operate in influencing risk for later post-trauma psychopathology, including AN, is worthy of investigation.

The fetal programming hypothesis (65) suggests that a shared vulnerability to develop PTSD and AN may begin even before birth. Neonatal dysmaturity (66) and low birth weight (65) are associated with maternal undernutrition and stress, and these developmental markers are associated with epigenetic changes in neural programming that later in life lead to an exaggerated behavioral stress response from increased HPA axis and amygdala activity. Although these epigenetic changes are theorized to increase the reproductive fitness of an organism (65, 67), they are also associated with increased avoidance responses to stress (65) and vulnerability to psychiatric disorders in adulthood (68, 69). Preliminary evidence suggests that epigenetic processes affecting the development of the HPA axis in the fetus mediates the relation between trauma and PTSD (69, 70). Similarly, one study found that childhood abuse was only associated with AN in the presence of neonatal dysmaturity, suggesting that epigenetic processes occurring in the prenatal environment combined with exposure to childhood maltreatment are related to an increased vulnerability for AN (66). In summary, PTSD and AN in adulthood may be related to the development of the brain’s stress response system including the HPA axis and amygdala with vulnerability for developing one or both of these disorders beginning during fetal development and subsequent early childhood experiences imparting either greater risk or resilience (61).

The results of this study should be considered in context of its limitations. First, our results represent a secondary analysis of data from a genetic study; therefore each exclusion criterion in the original study potentially influences the extent to which the results can generalize to other samples. Second, because reporting of traumatic events (71) is influenced

by several factors including age at which the trauma occurs, intensity of emotions experienced, and whether the trauma is directly experienced or witnessed (72), trauma history collection is susceptible to errors in recall. A comprehensive and structured approach to the assessment of traumatic events that combines self-report and in depth-interview data may optimize the accuracy of the collected data (72). Third, our sample population only included women. Thus, the results may not be generalizable to men with eating disorders. Fourth, it is possible that our sampling strategy (>1 affected relative) could limit the generalizability of the results. However, previous analyses of this dataset have yielded estimates of eating disorder severity and comorbid psychopathology that are well within the bounds of other samples in the literature. Fifth, we were limited by the absence of either pathological or control comparison groups.

Conclusion

AN and PTSD co-occur and traumatic events tend to occur prior to the onset of AN. Clinically, these results underscore the importance of assessing trauma history and PTSD in individuals with eating disorders (3, 5). In addition, they raise the question of whether specific modifications or augmentations to standard treatment for AN should be considered to address PTSD-related psychopathology.

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Glossary

AN	anorexia nervosa
ANBN	lifetime diagnosis of both anorexia and bulimia nervosa
ANOVA	analysis of variance
BAN	anorexia nervosa with binge, with or without purging
BED	binge eating disorders
BN	bulimia nervosa
BMI	body mass index
CNS	central nervous system
DSM	Diagnostic and Statistical Manual
EDNOS	eating disorders not otherwise specified
GEE	generalized estimating equations
HA1	anticipatory worry and pessimism vs. uninhibited optimism subscale of harm avoidance
HPA axis	hypothalamic-pituitary-adrenal axis
MZ	monozygotic
NIH	National Institutes of Health

PAN	purging anorexia nervosa without binge eating
PTSD	post traumatic stress disorder
RAN	restricting anorexia nervosa
SAS-STAT	Statistical Analysis Software
SIAB	Structured Interview for Anorexia Nervosa and Bulimic Syndromes
SCID-1	Structured Clinical Interview for Axis I Disorders
STAI-Y	State-Trait Anxiety Inventory Form Y
TCI	Temperament and Character Inventory
Y-BOCS	Yale-Brown Obsessive Compulsive Scale
YBC-EDS	Yale-Brown-Cornell Eating Disorder Scale

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Table I

Number (%) of women with post traumatic stress disorder (PTSD) by anorexia nervosa (AN) subtype.

	PTSD Diagnosis Number (%)	
AN subtype [a]	**No PTSD**	**DSM-IV PTSD**
RAN (n=332)	299 (90.1)	33 (9.9)
PAN (n=217)	177 (81.6)	40 (18.4)
BAN (n=115)	96 (83.5)	19 (16.5)
ANBN (n=89)	78 (87.6)	11 (12.4)
TOTAL (n=753)	650 (86.3)	103 (13.7)

[a] RAN-restricting AN; PAN-AN with purging and without binge eating; BAN-AN with binge eating, with or without purging; ANBN-lifetime diagnosis of both AN and bulimia nervosa

Table II

Of patients with a post traumatic stress disorder (PTSD) diagnosis (n=103), number (%) of women reporting one or more than one traumatic event by anorexia nervosa (AN) subtype

	Number of traumatic events Number (%)[a]	
AN subtype[b]	One Event	More Than One Event
RAN	15 (45.5)	18 (54.5)
PAN	22 (55.0)	18 (45.0)
BAN	12 (63.2)	7 (36.8)
ANBN	5 (45.5)	6 (54.5)

[a] Cochran-Mantel-Haenszel (CMH) chi-square statistic indicates no significant location shift in distribution of number of events across eating disorder subtype (CMH chi-square=0.59, df=1, p<0.44).

[b] RAN-restricting AN; PAN-AN with purging and without binge eating; BAN-AN with binge eating, with or without purging; ANBN-lifetime diagnosis of both AN and bulimia nervosa.

Table 3

Percent of women experiencing each type of traumatic event[a], by post traumatic stress disorder (PTSD) diagnosis status and anorexia nervosa (AN) subtype[b]

	PTSD Absent					PTSD Present				
Traumatic Events	**RAN (n=299)**	**PAN (n=177)**	**BAN (n=96)**	**ANBN (n=78)**	**Total (n=650)**	**RAN (n=33)**	**PAN (n=40)**	**BAN (n=19)**	**ANBN (n=11)**	**Total (n=103)**
Early Child Sexual Abuse/ Molestation (5 years or younger)	0.0	0.0	2.1	1.3	0.5	15.2	12.5	10.5	27.3	14.6
Childhood Sexual Abuse (6–17 years)	1.7	4.0	3.1	2.6	2.6	42.4	37.5	42.1	45.5	40.8
Sexual Abuse, Rape, Sexual Attack (18 or older)	4.0	5.1	0.0	5.1	3.8	33.3	37.5	26.3	45.5	35.0
Death/Sickness of Family Member or Significant Other	5.0	11.3	2.1	9.0	6.8	24.2	22.5	0.0	9.1	17.5
Domestic Violence, Adult	0.0	1.1	0.0	1.3	0.5	6.1	5.0	0.0	0.0	3.9
Domestic Violence, Child	1.0	2.3	3.1	1.3	1.7	12.1	15.0	10.5	0.0	11.7
Crime or Violence/Terrorism	3.7	3.4	3.1	2.6	3.4	6.1	10.0	15.8	0.0	8.7
Car/Motor Accident	6.0	3.4	3.1	11.5	5.5	9.1	10.0	5.3	9.1	8.7
Natural Disaster	0.0	2.3	0.0	2.6	0.9	6.1	5.0	0.0	0.0	3.9
Witness of Death or Someone at Immediate Risk of Dying	4.0	3.4	1.0	7.7	3.8	12.1	2.5	5.3	0.0	5.8
Other	10.0	10.2	8.3	5.1	9.2	15.2	7.5	31.6	18.2	15.5

[a]Women can report more than one event. If a one type of event was reported more than once, only the first incident was tallied in this table.

[b]RAN-restricting AN; PAN-AN with purging and without binge eating; BAN-AN with binge eating, with or without purging; ANBN-lifetime diagnosis of both AN and bulimia nervosa

Table IV

Relative risk of post traumatic stress disorder for characteristics associated with anorexia nervosa

	Ratio of Probabilities	
Parameter	**Unadjusted**	**Adjusted[a]**
Low BMI	1.00 (0.91, 1.10)	1.01 (0.92, 1.11)
Trait anxiety	1.02 (0.99, 1.04)	1.02 (1.00, 1.04)
Harm avoidance	1.04 (0.95, 1.15)	1.04 (0.94, 1.15)
Concern over mistakes	1.00 (0.98, 1.03)	1.00 (0.98, 1.03)
Age at menarche	1.02 (0.92, 1.12)	1.01 (0.92, 1.12)
Obsessions score >0[b]	1.15 (0.75, 1.74)	1.16 (0.77, 1.75)
YBC-EDS total Score[c]	**1.07 (1.02, 1.12)**	**1.08 (1.03, 1.13)**

[a] Adjusted for age at interview.

[b] Dichotomous Yale-Brown Obsessive Compulsive Scale score not equal 0

[c] Yale-Brown-Cornell Eating Disorders Scale

Note: Values in bold are significant at an alpha level=0.05