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RESEARCH ARTICLE

Posttraumatic stress disorder (PTSD) and complex PTSD in eating disorder treatment-seekers: Prevalence and associations with symptom severity

Sinead Day[1] | **Phillipa Hay**[2,3] | **Christopher Basten**[4] | **Susan Byrne**[5,6] | **Amanda Dearden**[7] | **Mandy Goldstein**[2,8] | **Amy Hannigan**[7] | **Gabriella Heruc**[9] | **Catherine Houlihan**[10] | **Marion Roberts**[11,12] | **W. Kathy Tannous**[13] | **Chris Thornton**[14] | **Natalie Valentine**[15] | **Deborah Mitchison**[16]

[1]Translational Health Research Institute, Western Sydney University, Penrith, Australia

[2]School of Medicine, Western Sydney University, Penrith, Australia

[3]Mental Health Services Camden and Campbelltown Hospitals, South West Sydney Local Health District, Sydney, Australia

[4]Department of Psychology, Macquarie University, Sydney, Australia

[5]School of Psychology, University of Western Australia, Perth, Australia

[6]The Swan Centre, Perth, Australia

[7]Queensland Eating Disorder Service, Indooroopilly, Australia

[8]everyBody Psychology & Wellbeing, Bondi Junction, Australia

[9]Appetite for Change, Crows Nest, Australia

[10]School of Health, University of the Sunshine Coast, Queensland, Australia

[11]Department of General Practice and Primary Healthcare, University of Auckland, Auckland, New Zealand

[12]Nurture Psychology, Auckland, New Zealand

[13]School of Business, Translational Health Research Institute, Western Sydney University, Penrith, Australia

[14]The Redleaf Practice, St. Leonards, Australia

[15]Wandi Nerida, Mooloolah Valley, Australia

[16]Graduate School of Health, University of Technology Sydney, Ultimo, Australia

Correspondence
Sinead Day, Translational Health Research Institute, Western Sydney University, Locked Bag 1797, Penrith NSW 2751, Australia.
Email: s.day2@westernsydney.edu.au

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Abstract

Although childhood trauma and posttraumatic stress disorder (PTSD) have been well-researched in eating disorder epidemiology, prevalence rates are unavailable for complex PTSD (CPTSD). Under recently introduced *ICD-11* criteria, individuals with CPTSD have both PTSD symptoms and additional disturbances in self-organization (DSO). Using *ICD-11* criteria, this study aimed to determine the prevalence of PTSD and DSO symptoms, diagnostic rates of PTSD and CPTSD, and childhood trauma exposure in eating disorder treatment-seekers. Participants (N = 217) were individuals attending residential, partial

hospitalization, and outpatient services who completed measures of eating disorder– and trauma-related symptoms and childhood adverse experiences. One third of participants reported PTSD symptoms, and over half reported DSO symptoms, with probable *ICD-11* diagnostic rates of 3.8% for PTSD and 28.4% for CPTSD. CPTSD was significantly more prevalent than PTSD and more common in higher levels of care. Both PTSD and DSO symptom severity were positively correlated with eating disorder symptoms and impairment, *r*s = .285–.642. DSO symptom severity was a significant and unique explanatory factor of eating disorder severity and impairment. The findings highlight the prevalence of CPTSD in eating disorder populations and the association between DSO symptoms and eating psychopathology independent of PTSD symptoms. Implications are discussed for adjunct treatment approaches for individuals with comorbid eating disorders and PTSD or CPTSD.

Exposure to traumatic experiences is common among individuals with eating disorders (EDs; e.g., Solmi et al., 2020; Trottier & MacDonald, 2017). EDs involve disturbances in eating-related behavior that impair physical or psychosocial functioning, including anorexia and bulimia nervosa, and binge eating disorder (American Psychiatric Association [APA], 2013). They are associated with a mortality risk up to twice as high as the general population (van Hoeken & Hoek, 2020). In most cases, traumatic events are found to predate ED onset (e.g., Kjaersdam Telléus et al., 2021), and traumatic events such as childhood abuse or neglect have been suggested to contribute to ED development via mechanisms including emotion dysregulation, dissociation, and maladaptive core beliefs, including a sense of defectiveness and abandonment (Trottier & MacDonald, 2017). Prior research has highlighted the prevalence of exposure to traumatic events (Rienecke et al., 2022; Tagay et al., 2014) and posttraumatic stress disorder (PTSD; Brewerton et al., 2020; Scharff et al., 2021) among ED populations. However, as conceptualizations of trauma continue to evolve, a more nuanced understanding is needed regarding which trauma-related presentations are most likely to occur in individuals with EDs.

Trauma is most broadly defined as both the experience of a threatening or aversive event or events and a corresponding intense emotional and biological stress response, including persistent symptoms that may meet the criteria for PTSD (Dalenberg et al., 2017). PTSD criteria vary between the two major diagnostic systems of the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*; APA, 2013) and the *International Statistical Classification of Diseases and Related Health Problems* (11th ed.; *ICD-11*; World Health Organization [WHO], 2019). The *DSM-5* diagnostic criteria were expanded from the previous edition to encompass four symptom clusters (i.e., intrusions, avoidance, negative alterations in cognition and mood, and alterations in arousal and reactivity) and an increased set of 20 possible symptoms (e.g., distressing thoughts, flashbacks, and sleep disturbance). Galatzer-Levy and Bryant (2013), among other researchers, have criticized these criteria for being overly broad and heterogeneous, with hundreds of thousands of potential symptom combinations.

The *ICD-11*, in contrast, recently narrowed the criteria for PTSD to remove symptoms that may be common to other disorders and focus on disorder-specific symptoms, such as dissociative flashbacks and hypervigilance (WHO, 2019). These tighter *ICD-11* PTSD criteria have been found to lead to fewer diagnosed cases of PTSD than the *DSM-5* criteria (e.g., Shevlin et al., 2018). Moreover, the *ICD-11* has established complex PTSD (CPTSD) as a new trauma-related diagnosis. To be diagnosed with CPTSD, an individual must meet the criteria for PTSD and additionally experience disturbances in self-organization (DSO). DSO represents a new symptom category composed of affective dysregulation, negative self-concept, and disturbances in relationships (WHO, 2019). Thus, the *ICD-11* criteria for CPTSD overlap with both *ICD-11* and *DSM-5* PTSD criteria in requiring the presence of core PTSD symptoms, including avoidance, heightened threat perception, and reexperiencing symptoms (referred to as intrusions in the *DSM-5*). DSO symptoms as outlined in the *ICD-11* CPTSD criteria resemble some *DSM-5* PTSD criteria in terms of disturbances in affect and cognition.

CPTSD as a distinct diagnosis was considered for both the fourth edition of the *DSM* (*DSM-IV*; APA, 1994), under the closely related proposed diagnosis of disorders of

extreme stress not otherwise specified [DESNOS]), and the *DSM-5*; however, it was not included, citing insufficient empirical evidence (Resick et al., 2012). The *DSM-5* incorporated some elements of CPTSD into its definition of PTSD by broadening its criteria. In contrast, the *ICD-11* introduced PTSD and CPTSD as sibling disorders with simplified, limited symptomatology, which were hoped to improve clinical utility (Maercker, 2021) and reflected growing evidence for distinct symptom profiles (Karatzias et al., 2017).

The rationale for introducing CPTSD in the *ICD-11* also included highlighting the distinct symptoms seen in survivors of prolonged, repeated, and interpersonal traumatic experiences as opposed to single-incident traumatic events, particularly those that occur in childhood, such as verbal, physical, emotional, and sexual abuse and neglect (van der Kolk, 2007; Van Nieuwenhove & Meganck, 2019). These prolonged and interpersonal traumas often involve relational factors such as abandonment, helplessness, or betrayal, and are associated with pervasive developmental consequences for children (van der Kolk, 2007). Correspondingly, childhood interpersonal trauma has been found to be a bigger risk factor for CPTSD than for PTSD (Hyland et al., 2017). CPTSD may be particularly relevant to EDs due to their shared association with childhood maltreatment (Molendijk et al., 2017; Trottier & MacDonald, 2017). DSO symptoms also resemble some of the hypothesized mechanisms of trauma exposure in the development of EDs, particularly difficulties with emotion regulation.

Current trauma prevalence research in EDs focuses on PTSD and exposure to traumatic events. A systematic review and meta-analysis of 82 studies of childhood maltreatment (i.e., sexual, physical, and emotional abuse) in EDs found that 21%–59% of participants had experienced this form of trauma compared to 1%–35% of healthy controls (Molendijk et al., 2017). Research on exposure to traumatic events in adulthood is more sparse, but findings suggest higher rates of perinatal intimate partner violence (e.g., 10%–28% among women with a lifetime ED; Kothari et al., 2015) and sexual trauma during military service (e.g., 55% of female veterans with ED; Forman-Hoffman et al., 2012). A study of 4,524 individuals with EDs found that 19% of participants had experienced any kind of traumatic event, 62% of whom experienced trauma that occurred in childhood or adolescence, and 38% of whom reported adulthood trauma, with sexual trauma being the most common experience (Backholm et al., 2013). These findings highlight high rates of trauma exposure in ED populations, with substantial literature focused on interpersonal and childhood traumatic events.

Not everyone who experiences a traumatic event goes on to develop PTSD. In community samples, PTSD prevalence estimates range from less than 10% to nearly 50% among individuals exposed to trauma, with higher rates seen for those who are younger, female, have prior trauma exposure, and experience interpersonal trauma (Kessler et al., 2017; Shalev et al., 2019). However, previous research suggests that PTSD is relatively common among individuals with EDs. Most of this literature, including all research included here unless noted otherwise, has been based on the *DSM-IV* and *DSM-5* criteria for PTSD. In a recent review by Rijkers et al. (2019), the rate of comorbid PTSD was 9%–24% in the four included articles, with one study using *ICD-9* and *ICD-10* criteria, two using *DSM* criteria, and one study with unclear criteria specifications. A review by Ferrell et al. (2022) found that the rate of comorbid PTSD and ED was 28% across 29 included studies, with one study using *ICD-10* criteria, 22 studies using *DSM* criteria, and six studies with unclear criteria specifications. In this review, comorbid PTSD was found to be more common among individuals receiving inpatient (32%) versus outpatient treatment (24%). Similarly, PTSD has been found in 49% of individuals in residential ED treatment (Brewerton et al., 2020) and 53.5% of those receiving inpatient, residential, or partial hospitalization treatment for EDs (Rienecke et al., 2021). Moreover, both trauma exposure (Backholm et al., 2013) and PTSD (Brewerton et al., 2020; Scharff et al., 2021) have been found to be associated with more severe ED psychopathology.

To our knowledge, no previous studies have examined the prevalence of CPTSD using the recently introduced *ICD-11* criteria in a sample of individuals with EDs. Given that the types of trauma exposure most common in EDs—childhood maltreatment and adult interpersonal trauma—are also those most likely to result in CPTSD (Hyland et al., 2017), this raises the question of whether CPTSD may be highly prevalent in individuals with EDs. As a CPTSD diagnosis requires both PTSD symptoms and additional DSO symptoms, studies of PTSD prevalence in EDs based on *DSM* criteria or previous editions of the *ICD* will have included cases of CPTSD. With the advent of the *ICD-11* CPTSD criteria, establishing separate prevalence rates for PTSD and CPTSD is important to recognize the presence and impact of DSO symptoms, allowing clinicians to make informed decisions about targeting these in treatment. This may include considering trauma-informed principles in the therapeutic relationship and incorporating therapies that have been found to be effective for DSO symptoms.

The current study aimed to investigate the prevalence and clinical correlates of *ICD-11* PTSD, CPTSD, and exposure to childhood trauma in three treatment settings for

EDs: residential, partial hospitalization, and outpatient. We hypothesized that (a) CPTSD would be more prevalent than *ICD-11* PTSD; (b) trauma-related disorders would be more prevalent in individuals who presented to higher levels of care for EDs (i.e., residential and partial hospitalization services); and (c) trauma exposure, PTSD, and CPTSD would each be associated with more severe ED psychopathology and ED-related impairment.

METHOD

Participants

A total of 312 individuals were invited to participate across three treatment groups. Residential treatment was delivered at Wandi Nerida, the first ED residential service in Australia, and involved 2–3 months of treatment in a live-in, home-like setting. Partial hospitalization took place at the Queensland Eating Disorders Service's (QuEDS) Day Program, a state-wide public health service running 4 days per week for 8 weeks. Both services include individual and group psychological therapies, nutritional rehabilitation, and family support. Outpatient treatment consisted of regular (typically weekly or fortnightly) consultations with a psychologist at one of seven private clinics. All services primarily relied on cognitive behavioral therapeutic modalities, such as enhanced cognitive behavioral therapy for EDs (CBT-E), as well as other evidence-supported modalities (e.g., dialectical behavioral therapy [DBT]). Partial hospitalization and residential sites were located in Australia; outpatient services were in Australia and New Zealand.

Of the invited individuals, 33 (10.6%) did not consent to participate (residential: $n = 1$, outpatient: $n = 32$). A total of 62 participants (partial hospitalization: $n = 3$, outpatient: $n = 59$) were excluded for not answering either trauma measure. One-way analyses of variance (ANOVAs) showed that excluded participants did not significantly differ on any of the other variables, $ps > .05$. The remaining sample included 217 participants: 66 who were receiving residential treatment, 21 who were receiving partial hospitalization, and 130 who were receiving outpatient treatment. Eligible participants were 16 years of age or older, had a primary ED diagnosis, were proficient in English, and were able to consent to study participation.

Most participants identified as female (90.1%), and a minority identified as male (8.0%) or another gender (1.9%). Participants ranged in age from 16 to 77 years, with a mean age of 27.2 years ($SD = 9.95$). The average ED age of onset was 16.4 years ($SD = 6.50$). Most participants were born in Australia (60.6%) or New Zealand (24.4%). All treatment groups included participants with a variety of ED diagnoses. The most common presentation was anorexia nervosa restrictive subtype (43.5%), followed by atypical anorexia (15.9%), anorexia nervosa binge–purge subtype (14.5%), binge eating disorder (8.7%), and bulimia nervosa (6.5%).

Procedure

The present study used a cross-sectional, between-subjects design. Data were collected between January 2022 and August 2023. All treatment sites were part of the TrEAT Registry, which allows ED services to elect to participate in collecting selected outcomes data for clinical monitoring, research, and their own quality assurance purposes (for a further description, see Babbott et al., 2022). Participants were recruited on admission to each service. Participants self-referred or were referred by their general practitioner (GP) and were assessed for treatment suitability by program staff. Participants completed the measures via an online survey, using the Qualtrics survey platform, on admission except the ACEs Questionnaire for outpatient treatment. For the outpatient treatment group, the ACEs measure was completed in Week 8 due to clinical staff being unable to directly supervise the admission survey for these participants. As such, staff at this treatment site requested delayed administration of this measure to be able to promptly address any risk issues arising from participants' responses. However, as this is a measure of historical experiences rather than current symptoms, the difference in administration timing was not expected to influence the findings. The partial hospitalization group elected not to complete the ACEs Questionnaire at the request of clinical staff.

All procedures were approved by the ethics committees of Western Sydney University (H14478, H14742) and Royal Brisbane Hospital (EC00172). All participants provided informed consent to participate in the study.

Measures

Demographic and clinical characteristics

Participants reported demographic characteristics. Clinicians at all treatment sites assessed participants' body mass index (BMI; a common clinical correlate) and ED diagnosis (based on admission interviews).

ED symptoms

ED psychopathology was measured using the Eating Disorder Examination Questionnaire (Version 6; EDE-Q; Fairburn, 2008). Scores are averaged to provide index scores for global ED psychopathology and four subscales (i.e., Restraint, Eating Concerns, Weight Concerns, Shape Concerns). Global and subscale scores range from 0 to 6,

with higher scores representing more severe symptoms. The EDE-Q has been psychometrically validated in Australian community samples, yielding good validity (Mond et al., 2004b) and internal consistency (Mond et al., 2004a), with a global score cut-off of 2.8 shown to reliably screen for EDs (Mond et al., 2008). The scale had excellent internal consistency in the current sample, Cronbach's $\alpha = .94$.

ED-related impairment was assessed using the Clinical Impairment Assessment (CIA; Bohn & Fairburn, 2008), with scores ranging from 0 to 48 and higher scores reflecting greater impairment. This scale has been found to have acceptable reliability and a clinical cutoff score of 16 (Raykos et al., 2019; Reas et al., 2010). The CIA demonstrated excellent internal consistency in the current sample, Cronbach's $\alpha = .95$.

Trauma-related symptoms and disorders

PTSD and CPTSD symptoms were measured using the International Trauma Questionnaire (ITQ; Cloitre et al., 2018). This 20-item measure is derived from *ICD-11* diagnostic criteria. Although the instrument is not a diagnostic tool for PTSD and CPTSD on its own, the ITQ indicates probable diagnoses. The validity of the ITQ in distinguishing between PTSD and CPTSD under *ICD-11* diagnostic criteria has been established in multiple studies (Camden et al., 2023; Murphy et al., 2020). For a probable diagnosis of PTSD, participants must endorse at least one symptom from each PTSD symptom domain (i.e., reexperiencing, avoidance, sense of threat) at a level of *moderately* or higher and report at least moderate resulting functional impairment. For a probable diagnosis of CPTSD, participants must endorse at least one symptom from each PTSD and DSO symptom domain (i.e., affective dysregulation, negative self-concept, disturbances in relationships) at a level of *moderately* or higher and report functional impairment from DSO symptoms. Individuals who report both threshold PTSD and DSO symptoms receive a probable diagnosis of CPTSD (i.e., under the *ICD-11*, CPTSD supersedes PTSD such that they are never comorbid). ITQ scores can be summed to create continuous indices of PTSD and DSO symptom severity, ranging from 0–24, with higher scores representing more severe symptoms. In the current sample, the ITQ demonstrated good internal consistency for both the PTSD, Cronbach's $\alpha = .92$, and CPTSD subscales, Cronbach's $\alpha = .88$.

Childhood trauma exposure

Childhood trauma history was measured using the Adverse Childhood Experiences (ACEs) Questionnaire (Felitti et al., 1998), which is used to assess the occurrence of 10 discrete traumatic experiences, including forms of abuse and neglect. It provides a continuous indicator of the number of ACEs experienced (range: 0–10) and a categorical indicator of specific experiences. The psychometric properties have been supported in clinical and community samples (Bruskas & Tessin, 2013; Dube et al., 2004). In the present sample, the measure demonstrated adequate internal consistency, Cronbach's $\alpha = .78$.

Data analysis

Data were analyzed using SPSS (Version 29). Descriptive statistics were calculated for each of the outcome and demographic variables. As data were not normally distributed, Spearman correlations were used to investigate associations between continuous variables, and Kruskal–Wallis tests were used to compare differences by treatment group, with effect sizes reported as eta-squared, calculated from the Kruskal-Wallis H statistic (see Tomczak & Tomczak, 2014). Post hoc comparisons were conducted using Mann–Whitney tests with Bonferroni-adjusted p values. Differences in prevalence rates of probable PTSD and CPTSD, including by treatment group and ED diagnosis, were assessed using chi-square tests; significant differences were further probed using z tests with Bonferroni-adjusted p values, and effect sizes were reported as Cramer's V. Hierarchical regressions assessed the level of variance explained in ED psychopathology and ED-related impairment by the trauma-related variables, with variables entered in four blocks: (a) age, gender, and BMI (covariates, categorical variables dummy-coded); (b) total ACEs; (c) PTSD symptom severity; and (d) DSO symptom severity. Missing data were handled listwise for regression analyses, as this method has been shown to perform well when the pattern of missing data is not random (Kromrey & Hines, 1994; missing ACEs data was largely based on treatment site).

RESULTS

Table 1 presents descriptive statistics and results from Kruskal–Wallis tests for each continuous variable. There were significant differences between treatment settings for each of the ED-related variables and PTSD and CPTSD symptom severity. Post hoc comparisons showed that compared to outpatient participants, those in the residential, $p < .001$, and partial hospitalization groups, $p = .006$, reported more severe ED psychopathology, as well as dietary restraint, $p < .001$, $p = .005$; eating concerns, $p < .001$, $p = .045$; weight concerns, $p < .001$, $p = .033$;

TABLE 1 Comparison of mean scores on demographic and outcome variables

	Total sample ($N = 217$)		Residential ($n = 66$)		Partial hospitalization ($n = 21$)		Outpatient ($n = 130$)					
Variable	***M***	***SD***	***M***	***SD***	***M***	***SD***	***M***	***SD***	***p***	***df***	η^2_H	**Post hoc comparisons**
Age (years)	27.21	9.95	25.49	6.63	27.19	7.68	28.12	11.54	.832	2	0.008	
Eating disorder age of onset (years)	16.37	6.50	15.23	5.63	16.11	8.25	17.01	6.61	.072	2	0.016	
BMI (kg/m^2)	21.25	7.21	19.00	6.08	21.19	4.67	24.52	8.44	< .001	2	.150	OP > R PH > R
Global eating disorder symptoms	4.02	1.38	4.78	1.07	4.51	1.12	3.56	1.36	< .001	2	.216	R, PH > OP
Restraint	3.41	1.84	4.62	1.47	4.09	1.52	2.70	1.70	< .001	2	.242	R, PH > OP
Eating concerns	3.54	1.39	4.09	1.10	4.04	1.31	3.19	1.44	< .001	2	.098	R, PH > OP
Shape concerns	4.71	1.46	5.38	1.04	5.12	0.85	4.30	1.57	< .001	2	.161	R > OP
Weight concerns	4.41	1.56	5.03	1.25	4.80	1.24	4.04	1.63	< .001	2	.106	R, PH > OP R > OP
Eating disorder-related impairment	33.50	11.58	41.82	7.92	38.38	7.61	28.28	10.84	< .001	2	.334	R, PH > OP
Total ACEs	2.48	2.42	2.38	2.40	–	–	2.82	2.51	.461	1	−.006	
PTSD symptom severity	9.73	7.27	11.53	7.39	12.00	6.52	8.42	7.08	.006	2	.039	R > OP
DSO symptom severity	15.40	6.19	19.00	4.51	18.33	4.53	12.98	6.08	< .001	2	.220	R, PH > OP

Note: BMI = body mass index; ACEs = adverse childhood experiences; PTSD = posttraumatic stress disorder; DSO = disturbances in self-organization; *df* = degrees of freedom; R = residential; OP = outpatient; PH = partial hospitalization.

[a]Small: .06, moderate: .14, large: > .14.

ED-related impairment, $p < .001$, $p = .001$; and DSO symptoms, $p < .001$, $p = .001$. Residential participants reported higher shape and weight concerns, $ps < .001$; more severe PTSD symptoms, $p = .017$; and lower BMI, $p < .001$, than outpatient participants. Outpatient and partial hospitalization groups did not significantly differ in BMI, weight concerns, shape concerns, or PTSD symptoms, $p = .032–334$. Participants in the residential and partial hospitalization treatment groups only significantly differed in BMI, which was lower for those in residential treatment, $p = .044$.

A chi-square test showed significant differences in the rates of probable PTSD, CPTSD, and having neither PTSD nor CPTSD, $\chi^2(2, N = 211) = 131.84$, $p < .001$, $V = .56$. A subsequent binomial test pairwise comparison showed that rates of probable CPTSD were statistically significantly higher than rates of probable PTSD, $p < .001$. Additional chi-square tests found that neither PTSD prevalence, $\chi^2(8, N = 138) = 14.78$, $p = .064$, $V = 0.27$, nor CPTSD prevalence, $\chi^2(8, n = 137) = 10.19$, $p = .252$, $V = 0.33$, significantly differed by ED diagnosis, with no further pairwise comparisons conducted.

As shown in Table 2, across the total sample, DSO symptoms showed large positive associations with all measures of ED symptoms, $r_\rho = .503–.642$, except for dietary restraint, with which it had a medium positive association, $r_\rho = .490$. PTSD symptoms had a medium positive association with all measures of ED symptoms, $r_\rho = .304–.373$, except for eating concerns, for which there was a small positive association $r_\rho = .285$. PTSD and DSO symptoms were strongly positively correlated, and ACEs were positively and significantly correlated with PTSD but not DSO symptom severity.

As depicted in Table 3, 32.1% of participants reported (above) threshold PTSD symptoms and 58.3% reported threshold DSO symptoms. After accounting for impairment criteria and trumping rules (i.e., with CPTSD superseding a diagnosis of probable PTSD if the thresholds for both PTSD and DSO symptoms were met), overall, 28.4% of the sample met the criteria for a probable CPTSD diagnosis, and only 3.8% met the criteria for a probable PTSD diagnosis. The rates of both threshold PTSD symptoms and a probable PTSD diagnosis did not significantly differ by treatment setting, but the prevalence both of DSO symptoms and probable CPTSD diagnosis differed by treatment group. Post hoc comparisons showed that threshold DSO symptoms were significantly more common in residential (78.8%) and partial hospitalization (81.0%) treatment than in outpatient participants (43.5%), but the prevalence did not differ between residential and partial hospitalization samples. Probable CPTSD was more common in partial hospitalization compared with residential and outpatient treatment.

TABLE 2 Correlations between outcome variables across the sample

Variable	PTSD symptoms	DSO symptoms	Global eating disorder symptoms	Restraint	Eating concerns	Shape concerns	Weight concerns	Eating disorder-related impairment	BMI
Total ACEs	.416***	.091	.001	.022	−.046	.018	.058	−.010	.147
PTSD symptoms	–	.548***	.361***	.304***	.285***	.321***	.347***	.373***	−.194*
DSO symptoms		–	.585***	.490***	.508***	.514***	.503***	.642***	−.256**
Global ED symptoms			–	.861***	.828***	.881***	.864***	.728***	−.242**
Restraint				–	.569***	.635***	.592***	.606***	−.334***
Eating concerns					–	.695***	.685***	.648***	−.141
Shape concerns						–	.884***	.663***	−.163
Weight concerns							–	.646***	−.114

Note: ACEs = adverse childhood experiences; PTSD = posttraumatic stress disorder; DSO = disturbances in self-organization; ED = eating disorder; BMI = body mass index.
*$p < .05$. **$p < .01$. ***$p < .001$.

TABLE 3 Prevalence of posttraumatic stress disorder (PTSD) and complex PTSD (CPTSD) across treatment groups

	Total sample ($N = 212$)		Residential ($n = 66$)		PH ($n = 21$)		Outpatient ($n = 125$)				
Variable	***n***	**%**	***n***	**%**	***n***	**%**	***n***	**%**	***p***	$\chi^2(2)$	V[b]
Threshold PTSD symptoms	68	32.1	22	33.3	11	52.4	35	28	.083	4.98	.15
Probable PTSD diagnosis	8	3.8	0	0.0	1	4.8	7	5.6	.150	3.79	.13
Threshold DSO symptoms[a,b]	123	58.3	52	78.8	17	81.0	54	43.5	< .001	26.93	.36
Probable CPTSD diagnosis[a,b]	60	28.4	22	33.3	10	47.6	28	22.6	.036	6.66	.18

Note: PH = partial hospitalization; DSO = disturbances in self-organization.

[a]Total sample: $N = 211$, outpatient subsample: $n = 124$.

[b]Weak: .20, moderate: .60, strong: > .60.

TABLE 4 Prevalence of specific adverse childhood experiences

	Total sample ($N = 85$)	Residential ($n = 68$)	Outpatient ($n = 17$)		
Variable	**%**	**%**	**%**	***p***	$\chi^2(1)$[a]
Verbal abuse	34.9	28.8	58.8	.021	5.37
Physical abuse	20.5	16.7	35.3	.090	2.88
Sexual abuse	16.9	15.2	23.5	.411	0.68
Emotional abuse	43.4	40.9	52.9	.372	0.80
Neglect	8.4	7.6	11.8	.579	0.31
Parental divorce/separation	34.9	36.4	29.4	.592	0.29
Domestic violence toward mother	14.5	16.7	5.9	.260	1.27
Family member using drugs	27.7	30.3	17.6	.298	1.08
Family member with mental illness	41.0	40.9	41.2	.984	0.00
Family member went to prison	6.1	6.2	5.9	.967	0.00

Note: A Bonferroni correction was applied to p values.

[a]Pearson's chi-square test.

As seen in Table 4, there were no differences in the prevalence of experiencing each form of ACE except for verbal abuse, which was more commonly reported in outpatient treatment (58.8%) than residential treatment (28.8%). ACE frequency varied from 6.1% for having a family member incarcerated to 43.4% for emotional abuse.

The hierarchical regressions for both ED psychopathology and ED-related impairment in the overall sample were statistically significant (Tables 5 and 6). Gender was not retained in either of the final models. The first hierarchical regression was significant at Step 4, with the final model explaining 20.3% of the variance in ED psychopathology, $F(5, 62) = 4.412$, $p = .002$. After accounting for age, BMI, and number of ACEs, PTSD symptoms accounted for 7.9% of the variance in ED psychopathology, $p = .019$. DSO symptoms accounted for an additional 12.4% of the variance in ED psychopathology, $p = .002$.

The second hierarchical regression was significant at each step, with the final model explaining 27.9% of the variance in ED-related impairment, $F(5, 62) = 6.194$, $p < .001$. Age and BMI accounted for 12.7% of the variance in ED-related impairment, $p = .004$. Total ACEs and PTSD symptom severity did not statistically significantly add to the model. After accounting for covariates, ACEs, and PTSD symptoms, DSO symptom severity uniquely explained an additional 12.3% of the variance in ED-related impairment, $p = .001$.

DISCUSSION

This paper investigated the prevalence of PTSD and CPTSD within and across three different treatment settings for EDs. Approximately one third of individuals reported threshold PTSD symptoms, and over half reported DSO

TABLE 5 Results from hierarchical regression explaining eating disorder psychopathology

Variable	Step 1	Step 2	Step 3	Step 4	*SE*	95% CI
Age	−0.02	−0.02	−0.02	−0.02	0.01	[−0.05, 0.01]
BMI	−0.02	−0.02	−0.01	0.00	0.02	[−0.04, 0.04]
Total ACEs		0.05	−0.00	0.01	0.06	[−0.11, 0.13]
PTSD symptom severity			0.05*	0.01	0.02	[−0.04, 0.05]
DSO symptom severity				0.11**	0.03	[0.04, 0.17]
$R^2_{adjusted}$	0.02	0.02	0.08	0.20		
ΔR^2	0.05	0.01	0.08*	0.12**		

Note. Coefficients are unstandardized, and categorical variables are dummy-coded. CI = confidence interval; ACEs = adverse childhood experiences; BMI = body mass index; DSO = disturbances in self-organization.
*$p < .05$. **$p < .01$.

TABLE 6 Results from hierarchical regression explaining eating disorder-related impairment

Variable	Step 1	Step 2	Step 3	Step 4	*SE*	95% CI
Age	−0.28	−0.29	−0.30	−0.27	0.11	[−0.49, −0.04]
BMI	−0.30	−0.33	−0.29	−0.17	0.16	[−0.49, 0.15]
Total ACEs		0.45	0.09	0.21	0.48	[−0.75, 1.16]
PTSD symptom severity			0.31	−0.02	0.18	[−0.38, 0.34]
DSO symptom severity				0.89**	0.26	[0.36, 1.41]
$R^2_{adjusted}$	0.13	0.13	0.16	0.28		
ΔR^2	0.15**	0.01	0.05	0.12**		

Note. Coefficients are unstandardized, and categorical variables are dummy-coded. CI = confidence interfval; ACEs = adverse childhood experiences; BMI = body mass index; DSO = disturbances in self-organization.
*$p < .05$. ** $p < .01$.

symptoms. When diagnostic criteria were applied, there were overall point prevalence rates of 3.8% for probable PTSD and 28.4% for probable CPTSD, with CPTSD being significantly more prevalent than PTSD. DSO symptoms—the symptom cluster that distinguishes CPTSD from PTSD—uniquely accounted for 12.4% of the variance in ED psychopathology and 12.3% of the variance in ED-related impairment.

PTSD symptom prevalence was similar to that reported in previous literature (e.g., Ferrell et al., 2022), but the prevalence of a probable PTSD diagnosis in the current study was considerably lower than in prior research. As outlined previously, existing PTSD prevalence literature has largely used measures based on *DSM-IV* and *DSM-5* diagnostic criteria, which will capture cases of CPTSD due to overlapping symptoms. As such, our findings indicate that there is a lower prevalence of PTSD in EDs when *ICD-11* criteria are used to differentiate PTSD from CPTSD, with a probable PTSD diagnosis only being applied to individuals who present with PTSD symptoms in the absence of DSO symptoms. We found that PTSD symptoms were more severe in residential treatment than in outpatient treatment, which is consistent with prior research (Ferrell et al., 2022). However, when PTSD was distinguished from CPTSD by an absence of DSO symptoms, the rates of probable PTSD diagnosis did not differ by treatment setting. CPTSD diagnosis, in contrast, was more prevalent in partial hospitalization than residential and outpatient treatment, and when isolating DSO symptoms, this distinct CPTSD symptom cluster was more prevalent and more severe at higher levels of care. As such, the prevalence rates were consistent with previous research as well as with our hypotheses regarding CPTSD diagnosis and DSO symptoms but not PTSD symptoms alone.

As predicted, PTSD and DSO symptoms correlated with more severe ED symptoms and ED-related impairment. This replicates previous findings for PTSD (Brewerton et al., 2020; Scharff et al., 2021; Tagay et al., 2014) and extends them to DSO symptoms but with generally large, as opposed to medium, effect sizes. Similarly, regression findings established DSO symptoms as a significant explanatory factor of ED psychopathology and impairment, accounting for more variance than PTSD symptoms. In contrast, PTSD symptoms were significant in explaining variance in ED psychopathology but not ED-related impairment, and the total number of reported ACEs explained variance in neither ED psychopathology nor impairment. Interestingly, the number of reported ACEs

was correlated with PTSD but not DSO symptoms; however, the frequency of discrete types of childhood traumatic events may not indicate the severity or impact of these experiences. Overall, the findings support previous literature on the prevalence of childhood maltreatment in EDs (e.g., Molendijk et al., 2017) but highlight PTSD and DSO symptoms as stronger correlates of ED symptom severity.

This was the first study of which we are aware to analyze the prevalence and correlates of CPTSD in EDs. By using *ICD-11* criteria to differentiate PTSD and DSO symptoms, this study may provide a clearer indication of the prevalence of PTSD in EDs and suggests that previous literature on comorbid PTSD encapsulates *ICD-11* CPTSD cases. Differentiating between these symptom clusters allows for a more accurate understanding of the types of trauma presentations in ED samples and may enable more targeted adjunct treatments. However, this study did not assess PTSD prevalence based on *DSM-5* criteria, which should be included in future research alongside *ICD-11* criteria to provide more contextualization of findings within previous literature and diagnostic debates. In addition, self-report measures are not sufficient to confirm a clinical diagnosis, and, as such, future research would benefit from incorporating standardized interview tools.

Other limitations of this study include variation in sample sizes between treatment groups and a lack of available ACE data for the partial hospitalization group. Further research is also needed regarding how these trauma-related disorders impact treatment outcomes for EDs. A recent systematic review identified that both trauma exposure and PTSD diagnosis negatively affect ED treatment outcomes (Day et al., 2024). Further research is needed to determine whether these findings extend to, or indeed may be attributable to, the presence of DSO symptoms as in cases of CPTSD.

Establishing prevalence rates for CPTSD in EDs is important in understanding the need for adjunct therapies with approaches that may need to differ from those for PTSD. A systematic review of PTSD treatment guidelines indicated that CBT in various forms, including cognitive processing therapy (CPT; Resick et al., 2017) and prolonged exposure (PE; Foa et al., 2007), was recommended as the first-line psychological treatment in all but one of 14 guidelines (Martin et al., 2021). There are also emerging findings that these PTSD treatments can be integrated with ED therapies to improve symptoms of both disorders, such as CPT (for PTSD) with CBT for EDs (Trottier et al., 2017). Regarding CPTSD, a systematic review and meta-analysis of 51 randomized controlled trials found that although PTSD treatments were shown to benefit individuals with CPTSD, childhood trauma moderated outcomes for CPTSD symptoms such that individuals with CPTSD who had experienced childhood trauma showed smaller improvements following existing therapies for PTSD (Karatzias et al., 2019). Targeted treatments for CPTSD are undergoing evaluation, such as enhanced skills training in affective and interpersonal regulation (ESTAIR; Karatzias et al., 2023). Other findings indicate that existing interventions, such as DBT (Bohus, 2022) and CPT (Eilers et al., 2021), can be adapted to reduce CPTSD symptoms. Thus, individuals with comorbid CPTSD and EDs may benefit from incorporating therapies that have been developed or adapted specifically for CPTSD symptoms.

ED clinicians should consider assessing for trauma-related disorders and using measurement tools that are able to differentiate between PTSD and DSO symptom clusters. The ITQ (Cloitre et al., 2018), which was used in this study, is a freely available self-report screening instrument and is currently the most thoroughly researched and validated measure for CPTSD (for a review of this and other CPTSD measures, see Seiler et al., 2023).

Given the high prevalence of trauma-related symptoms and probable diagnoses found in the current study, there is a need for the development of practice guidelines that account for this comorbidity. As Brewerton (2023) noted, existing practice guidelines for EDs rarely do more than refer readers to the separate guidelines for these disorders. Integrated practice guidelines would assist with providing more effective, evidence-based, and coordinated care to the many individuals who experience both disorders. Such guidelines should include key principles of trauma-informed care, such as safety, collaboration, and empowerment, with additional potential considerations, such as avoiding retraumatization in restrictive treatment settings and offering informed choices around weighing (Brewerton, 2018). Brewerton (2023) recently put forward recommendations for integrated treatment planning for EDs and PTSD, many of which may be applicable to CPTSD.

Debates about appropriate PTSD diagnostic criteria and conceptualizations of CPTSD are ongoing and have been discussed in detail elsewhere (e.g., Cloitre, 2021; Cloitre et al., 2020). Although *ICD-11* CPTSD criteria were introduced to capture the effects of prolonged, inescapable stressors—including, but not exclusively, childhood trauma—some researchers advocate for a purely developmental approach to the diagnosis (e.g., see Kerig, 2023). In contrast, others have argued against differentiating CPTSD and PTSD entirely (e.g., Resick et al., 2012). Future research on variations of trauma exposure and trauma-related symptoms will continue to add clarity to the assessment and treatment of PTSD and CPTSD in EDs.

CPTSD appears to be common across multiple ED treatment settings. The recent establishment of CPTSD as a formal diagnosis in the *ICD-11* provides the

opportunity for more precise assessment and treatment of comorbid trauma-related disorders in ED populations by differentiating PTSD and DSO symptoms. Future research and clinical practice in the ED field should assess these symptom profiles separately and consider the utility of incorporating adjunct trauma-focused treatment approaches.

OPEN PRACTICES STATEMENT

The study reported in this article was not formally pre-registered. Neither the data nor the materials have been made available on a permanent third-party archive; requests for the data or materials should be sent via email to the lead author at s.day2@westernsydney.edu.au.

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ORCID

Sinead Day https://orcid.org/0000-0001-7634-035X

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