

RUNNING HEAD: TRAUMA CHARACTERISTICS, PTSD, AND SUD

ACCEPTED MANUSCRIPT

The impact of trauma characteristics on posttraumatic stress disorder and substance use disorder
outcomes across integrated and substance use treatments

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Funding: This work was supported by the National Institute on Drug Abuse (NIDA R01 DA10843; Hien: PI). Aside from approving it for study funding, this funding source was not involved in the study design, data collection or analysis, interpretation of results, writing of report, or the decision to submit this article for publication.

Declarations of interest: none

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Abstract

Proponents of complex posttraumatic stress disorder (PTSD) constructs suggest that specific trauma characteristics, such as earlier age of first trauma (trauma age) and higher number of traumas (trauma count), may obstruct PTSD symptom reduction in treatment. PTSD and substance use disorders (SUD) commonly co-occur, but the impact of trauma age and count on PTSD treatment responses in a comorbid PTSD and SUD sample is unclear. Further, no studies have examined the impact of trauma characteristics on SUD treatment outcomes or whether their impact on either PTSD or SUD outcomes varies if PTSD is directly addressed. A secondary analysis of a randomized controlled trial was conducted to examine: (1) whether trauma age and count influence comorbid PTSD and SUD (PTSD+SUD) responses during and following treatment; and (2) whether these effects differed across an exposure-based, integrated PTSD+SUD treatment (Concurrent Treatment of PTSD and Substance Use Disorders using Prolonged Exposure; COPE) and a SUD-only focused treatment (Relapse Prevention Therapy; RPT). Individuals with PTSD+SUD randomized to COPE ($n = 39$) or RPT ($n = 43$) provided weekly measurements of PTSD and SUD. Across COPE and RPT, earlier trauma age predicted reduced SUD improvement ($B = -0.01$, standard error = 0.00). Trauma count did not predict changes in PTSD or SUD during or following treatment. These findings suggest that excluding individuals from exposure-based, integrated treatments on the basis of trauma characteristics is not empirically supported. However, individuals with earlier trauma ages may require additional or unique clinical attention to improve their SUD outcomes.

Keywords: posttraumatic stress disorder; substance use disorder; integrated treatment; trauma characteristics; childhood trauma

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1. Introduction

Posttraumatic stress disorder (PTSD) and substance use disorders (SUDs) often co-occur, with up to half of those with SUDs having a current or lifetime PTSD diagnosis (Jacobsen, Southwick, & Kosten, 2001; Reynolds et al., 2005) and 18% to 50% of those with SUDs having a lifetime diagnosis of PTSD (Back et al., 2000; Brown, Recupero, & Stout, 1995; Cacciola, Koppenhaver, Alterman, & McKay, 2009; Dansky et al., 1996; Mills, Lynskey, Teesson, Ross, & Darke, 2005). PTSD+SUD is characterized by a heightened severity of both disorders, poor functioning, and more suicide attempts (McCauley, Killeen, Gros, Brady, & Back, 2012). Some treatments target one disorder, often SUD, prior to targeting PTSD if at all (Flanagan et al., 2019). However, given the mutually exacerbating impact that each disorder has on the other, integrated interventions for PTSD+SUD have been developed and tested such as Concurrent Treatment of PTSD and Substance Use Disorders using Prolonged Exposure (COPE; Brady, Back, & Coffey, 2004; Back, Foa, Killeen, & Mills, 2014; McCauley et al., 2012). A meta-analysis revealed that, although integrated, trauma-focused interventions were most effective for PTSD+SUD, their effect sizes were small and limited to PTSD outcomes (Roberts, Roberts, Jones, & Bisson, 2015). Thus, there remains an urgent need to improve PTSD+SUD treatments. Identifying who is less likely to benefit from evidence-based PTSD+SUD interventions, and thus requires additional focus, could improve the treatment outcomes. This study examined whether trauma characteristics influenced PTSD+SUD outcomes after receiving an integrated (COPE), versus a SUD-only focused (Relapse Prevention Therapy; RPT, Carroll, 1996), treatment.

1.1. Associations Between Trauma Characteristics and PTSD

Although various traumas (i.e., direct or indirect exposure to, witnessing, or hearing about a close individual's experience of an event that involves real or threatened death, serious injury, or sexual violence; APA, 2013) can lead to PTSD, some trauma characteristics may be particularly important to PTSD+SUD treatment. Individuals' age of first trauma exposure (i.e., *trauma age*) and the number of traumas that they experienced (i.e., *trauma count*) are thought to disrupt the development of systems that regulate emotional responses and interpersonal relationships, complicating the presentation of PTSD in what some have called "complex PTSD" (e.g., Briere & Spinazzola, 2005; Cloitre, Miranda, Stovall-McClough, & Han, 2005; Cloitre et al., 2009; Herman, 1992; van der Kolk, Roth, Pelcovitz, Sunday, & Spinazzola, 2005).

In alignment with complex PTSD conceptualizations, research corroborates that individuals with PTSD, with and without SUDs, with early trauma ages and high trauma counts have more severe clinical presentations than those with later trauma ages and low trauma counts (e.g., Agorastos et al., 2014; Copeland et al., 2018; Evren, Kural, & Cakmak, 2006; Langeland, Draijer, & van den Brink, 2004; Schumm, Briggs-Phillips, & Hobfoll, 2006). In retrospective studies, a childhood trauma or "early" trauma age is associated with higher PTSD severity, increased risk for a PTSD diagnosis, and greater severity of "complex PTSD" symptoms (e.g., impaired self-reference, regulation, and interpersonal relations) in adulthood (Dias, Sales, Mooren, Mota-Cardoso, & Kleber, 2017; Ehring & Quack, 2010; Krammer, Kleim, Simmen-Janevska, & Maercker, 2016; Ogle, Rubin, & Siegler, 2013; Van Voorhees et al., 2012). Adulthood trauma survivors with a childhood maltreatment history also have a higher likelihood of developing PTSD in adulthood (Breslau et al., 2014) and higher trauma counts are linked to poorer functioning and higher dissociation, guilt, shame, and interpersonal difficulties (Copeland et al., 2018; Ehring & Quack, 2010; Green et al., 2000; Hagenaars, Fisch, & van Minnen, 2011;

Mullett-Hume, Anshel, Guevara, & Cloitre, 2008). Extant findings thus suggest that earlier trauma age and higher trauma counts are linked to many adverse correlates and outcomes.

1.2.1. Trauma Characteristics and PTSD Treatment Responses

Some proponents of the complex PTSD construct argue that survivors of early and repeated interpersonal traumas who subsequently develop complex PTSD may not fully benefit from frontline PTSD therapies and instead require specialized ones (e.g., Cloitre, 2015; Cloitre et al., 2011; Hassija, Garvert, & Cloitre, 2015). However, literature examining whether trauma characteristics predict distinct PTSD responses to frontline PTSD interventions remains unclear, and research examining their impact on SUD responses is minimal. Given that memories from early in the lifespan are less coherent than later ones (e.g., Hayne, 2004), exposure-based interventions that rely on traumatic memory processing such as prolonged exposure (PE; Foa, Hembree, & Rothbaum, 2007) could be less effective in early trauma survivors. Nevertheless, studies have not shown that childhood trauma survivors exhibit different PTSD treatment responses following PE (Jaycox, Foa, & Morral, 1998) and behavioral, psychoeducational, and expressive therapies (Ford & Kidd, 1998), compared to adulthood trauma survivors.

Conversely, studies on trauma count imply a potential influence of such experiences on treatment outcomes, although findings remain mixed. In one study, individuals with PTSD with a high trauma count had higher baseline PTSD severity and less PTSD improvement during an exposure-based PTSD treatment and treatment as usual compared to those with a single trauma (Priebe et al., 2018). Similarly, individuals with a high trauma count and early trauma age exhibited increased PTSD severity following PE compared to those with low trauma count/late trauma age. However, these effects disappeared at follow-up, suggesting delayed post-treatment

improvement (van Minnen, Arntz, & Keijsers, 2002). Collectively, data suggests that trauma age does not obstruct PTSD outcomes and that trauma count may, but more research is needed.

It is unclear if early trauma age and high trauma count impact PTSD treatment response in PTSD+SUD, wherein they are particularly prevalent (Evren et al., 2006; Langeland et al., 2004; Müller et al., 2015; Schumacher, Coffey, & Stasiewicz, 2006). Previous works also often studied the impact of one trauma characteristic on PTSD responses without controlling for the confounding influence of the other, despite their potential overlap (i.e., early trauma may also involve more of it). One study examined the impact of both trauma characteristics in 103 Australian participants with PTSD+SUD randomized to receive either COPE and treatment-as-usual or treatment-as-usual alone. Although trauma age did not affect response to either COPE or treatment-as-usual, a higher trauma count predicted smaller reductions in PTSD symptoms at post-treatment (Mills et al., 2016). This study corroborates extant literature, which suggests that trauma age may not influence PTSD treatment outcome, but those with higher trauma counts may indeed require unique clinical attention (e.g., a complex PTSD intervention). However, it remains unknown if either trauma characteristic influence SUD outcomes in PTSD+SUD.

1.2. Associations Between Trauma Characteristics and SUD

Patients with SUD who report an earlier trauma age (i.e., childhood trauma) and comorbid PTSD begin problematic drug use earlier, use more in the month prior to treatment, and have more lifetime drug overdoses compared to those without a co-occurring PTSD diagnosis and childhood trauma (Mergler et al., 2018). Although this finding conflates trauma age and PTSD, other work in PTSD+SUD samples shows that individuals with childhood trauma have more lifetime drug treatment episodes, poly-drug use, substance use dependency, and PTSD severity than those with only adulthood trauma (Farrugia et al., 2011). Similarly, survivors

of childhood trauma exhibit greater severity of PTSD and alcohol use disorder, first drink at an earlier age, and heavier drinking than adulthood trauma survivors (Müller et al., 2015; Schumacher et al., 2006; Waldrop, Santa Ana, Saladin, McRae, & Brady, 2007). Given that individuals with an earlier trauma age exhibit higher SUD severity, they may also show worse SUD outcomes and thus require additional clinical attention, but this is unknown.

It also remains unknown whether trauma characteristics only impact treatment outcomes when directly targeted (i.e., via integrated PTSD+SUD interventions) or pervasively (i.e., even in SUD only treatments) given the impact of trauma age on severity of both disorders (e.g., Ehring & Quack, 2010; Farrugia et al., 2011). Trauma characteristics may be a useful prognostic indicator for both PTSD and SUD outcomes, even in SUD-focused interventions. However, they may also inadvertently conflate trauma characteristics and the demographic and socioeconomic indicators that they are linked with. For example, there are robust associations between some forms of early trauma exposure (e.g., domestic violence) and lower socioeconomic status indicators (e.g., Buka, Stichick, Birdthistle, & Earls, 2001; Fantuzzo & Fusco, 2007), which have also been associated with poorer SUD outcomes (e.g., McCaul, Svikis, & Moore, 2001; McKay & Weiss, 2001). Further, young girls are more likely than boys to experience specific interpersonal traumas, namely sexual assault (Finkelhor, Turner, Ormrod, & Hamby, 2009), which are theoretically particularly likely to lead to complex PTSD and obstruct treatment outcomes (Cloitre, 2015; Cloitre et al., 2011; Hassija et al., 2015). Controlling for socioeconomic status and sex in the study of the impact of trauma characteristics on outcomes is thus important.

The present study therefore aimed to replicate research examining the impact of trauma characteristics (trauma age and count) on changes in PTSD during and following treatment and extend it by examining their impact on changes in SUD during and following treatment in

PTSD+SUD, after controlling for socioeconomic indicators and sex. We also studied whether the relationship of trauma characteristics to PTSD and SUD treatment responses differed in an integrated PTSD+SUD treatment (COPE) versus a SUD-focused treatment (RPT; Carroll, 1996; Marlatt & Donovan, 2007). Consistent with previous studies (e.g., Ford & Kidd, 1998; Jaycox et al., 1998; Priebe et al., 2018; van Minnen et al., 2002), we hypothesized that, trauma age would not predict changes in PTSD severity, after accounting for the impact of trauma count, but that trauma count would predict less reduction in PTSD over treatment, controlling for trauma age. Furthermore, given the role of trauma age in exacerbating SUD severity (e.g., Farrugia et al., 2011; Müller et al., 2015; Schumacher et al., 2006; Waldrop et al., 2007), we hypothesized that, early trauma age would result in less reduction in SUD over treatment. We considered examinations of the impact of trauma count on SUD responses, comparisons of all study relationships across COPE and RPT, and examinations of the impact of trauma characteristics on post-treatment responses, to be exploratory given a lack of research in this area.

2. Material and Methods

2.1. Participants

This study is a secondary analysis of a randomized controlled trial of integrated treatment of PTSD+SUD (Ruglass et al., 2017). Analyses for the present study focus only on the active intervention arms of COPE (n = 39) or RPT (n = 43), and not the active monitoring control group. Participants were recruited from New York City through advertisements and referrals. Informed written consent was obtained prior to assessments. Inclusion criteria were: 1) PTSD as defined by *Diagnostic and Statistical Manual of Mental Disorders-IV-TR* (American Psychiatric Association [APA], 2000) or subthreshold PTSD (Grubaugh et al., 2005) defined as having experienced a traumatic event (Criteria A), meeting Criteria B (re-experiencing symptoms),

Criteria E (one-month symptom duration), and Criteria F (significant distress or functioning impairment) and either Criteria C (avoidance or numbing symptoms) *or* Criteria D (hyperarousal symptoms); 2) past or current alcohol or substance dependence as defined by *DSM-IV-TR* (APA, 2000); and 3) use of primary problematic substance in the past 3 months. Exclusion criteria were: 1) history of psychotic or bipolar disorder; 2) current severe depression (i.e., a score of 30 or greater on Beck Depression Inventory; Beck, Steer, & Brown, 1996); 3) imminent suicide risk; 4) current involvement in an abusive relationship; 5) ongoing participation in PTSD treatment; 6) initiation of or dosage change in psychotropic medication within eight weeks of participation; 7) evidence of organic mental syndrome; or 8) refusal to be audio/video-recorded.

Randomization to COPE, RPT, or an assessment-only control group was conducted using an urn randomization approach and stratified by baseline PTSD severity, baseline substance dependence severity, and sex. Assessors blind to group assignment collected clinical assessments at baseline, at each weekly visit during the 12-week active intervention study period, and at four post-treatment follow-ups which occurred one-week and one-, two-, and three-months after the completion of the active intervention period. Participation was compensated at various points throughout the study: \$35 USD for baseline assessments; \$15 USD per weekly completion of active study period assessments; and \$20 USD for each follow-up.

2.1.1. Power analyses. We conducted power analyses in G*Power to examine the number of individuals required to identify a conservative, small effect size ($f = .10$; Cohen, 1988) in a between-within interaction in an analysis of variance with two groups and 12 measurements per subject (see below), and a $r = .60$ correlation amongst repeated measures. Our results suggested that a sample size of 58 (29 individuals per group) would yield 80% power to detect a small effect size ($f = .10$) in a between-within interaction such as the one outlined by primary

study hypotheses. Based on this, we identified that our sample of $N = 82$ was likely sufficient to detect the two-way interaction effects under investigation.

2.2. Procedure

Participants received 12 sessions of COPE or RPT in weekly, 90-minute individual sessions over the course of 12 weeks. COPE integrates two evidence-based approaches, PE for PTSD and relapse prevention for SUD (Back et al., 2014), in the treatment of PTSD+SUD. COPE employs psychoeducation about the relationship between PTSD and SUD and in vivo and imaginal exposure exercises in conjunction with RPT strategies. RPT is a cognitive-behavioral intervention for SUDs that focuses on skill-building to minimize the risk of relapse. Neither traumatic events nor PTSD are discussed within RPT. If participants raised trauma- or PTSD-related issues in RPT, therapists redirected clients back to focus on the RPT curriculum. All study procedures were approved by the City College of New York Institutional Review Board.

2.3. Measures

2.3.1. Demographics. Age, education, sex, race/ethnicity, employment for the past 30 days, years of education, and marital status were collected at the time of the baseline interview.

2.3.2. Trauma characteristics and PTSD. Frequency of exposure to *DSM*-defined traumatic events were measured using the Life Events Checklist (LEC; Gray, Litz, Hsu, & Lombardo, 2004). Age of earliest trauma exposure and diagnosis of PTSD or subthreshold PTSD were assessed by the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995). Severity of self-reported PTSD symptoms was measured by the modified PTSD Symptom Scale Self-Report (MPSS-SR; Falsetti, Resnick, Resick, & Kilpatrick, 1993) at baseline, weekly during the active intervention period, and then at one-week, one-month, two-month, and three-month follow up assessment visits. The Cronbach alpha at the randomization week for this measure was .91.

2.3.3. Substance use. Lifetime alcohol and SUD diagnoses were assessed with the Structured Clinical Interview for *DSM-IV* for Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 2002). The type of primary problematic substance was determined by SCID SUD criteria and the total days of use in 30 days prior to baseline interview was assessed with the Addiction Severity Index – Lite (Cacciola, Alterman, McLellan, Lin, & Lynch, 2007). SUD severity was defined as the number of days of use of the problematic substance in past 7-days and measured weekly during treatment, and then at each follow up assessment visit, via self-report by the Substance Use Inventory (SUI; Weiss, Hufford, Najavits, & Shaw, 1995).

2.4. Data Analysis

Generalized Estimating Equations (GEE) (e.g., Burton, Gurrin, & Sly, 1998) using SPSS Version 25 was used because it allows for repeated measures and uses a semi-parametric method to estimate regression parameters via within-cluster similarity of residuals. Participants with missing data are retained in study analyses, and GEE is robust to covariance structure misspecification. As is common in SUD populations (Delucchi & Bostrom, 2004), weekly SUD data was positively skewed, overdispersed, and zero-inflated. We therefore used a negative binomial distribution to accommodate the SUD data (Hilbe, 2011).

Two primary GEE analyses were run with PTSD and SUD outcomes focusing on the active treatment phase and using the available weekly PTSD and SUD measures collected during the course of treatment to examine the impact of trauma characteristics on treatment response. Two secondary GEE analyses examining the impact of trauma characteristics on post-treatment PTSD and SUD responses, which included data from the last week of treatment and the one-week and the one-, two-, and three-month follow-up visits. For all of these analyses, treatment group was entered as a categorical predictor with time, trauma age, and trauma count as

continuous ones. Given the relationship between PTSD and trauma characteristics (e.g., Ehrling & Quack, 2010) and substance use (Walton et al., 2018), PTSD severity was entered as a covariate in SUD analyses. Sex, years of education, and the baseline number of days of employment in the past 30 days were entered into models as continuous demographic and socioeconomic covariates. Trauma characteristics predictors and covariates were grand-mean centered. Time \times trauma age and time \times trauma count were entered to examine if trauma characteristics predicted weekly changes in PTSD or SUD severity during or following treatment. Group \times time \times trauma age and group \times time \times trauma count interaction terms were entered to examine if the two-way interactions varied by treatment, and all subsidiary interactions required to build these interactions were entered. Autoregressive, exchangeable, and unstructured covariance structures were examined, retaining the model with the lowest corrected Quasilikelihood under the Independence Model Criterion value.

3. Results

3.1. Descriptive Information

See Ruglass et al. (2017) for descriptive and demographic information regarding study samples. The most common substances used by individuals in COPE and RPT were alcohol (48.7% and 41.9%, respectively) and alcohol and stimulants together (20.5% and 30.2%, respectively). Approximately 26.7% of entries across all measures (including but not limited to weekly measures) were missing, and a Little's Missing Completely at Random Test was not statistically significant, $\chi^2(1704) = 1608.15, p = .952$. Approximately 35.9% of those in COPE and 32.6% of those in RPT had subthreshold (as opposed to full) PTSD. Independent samples *t*-tests and chi-square tests suggest that the COPE and RPT groups did not differ in age, $t(80) = -0.54, p = .592$, years of education, $t(80) = 0.37, p = .715$, days of employment, $t(76) = 1.67, p$

= .098, race/ethnicity, $\chi^2(3) = 2.86, p = .413$, sex $\chi^2(1) = 0.75, p = .386$, employment status, $\chi^2(2) = 3.54, p = .171$, or marital status, $\chi^2(1) = 1.49, p = .223$. The groups also did not differ on the frequency of different forms of trauma such as physical assault, accident/disaster, or sexual assault (p s ranged from .12 to .71), or in alcohol dependence, $\chi^2(1) = 0.25, p = 0.618$, drug dependence, $\chi^2(1) = 0.30, p = .586$, or both diagnoses, $\chi^2(1) = 1.79, p = .181$. The mean trauma age in COPE and RPT were 17.90 (SD = 13.64), and 18.49 (SD = 14.13), respectively, and did not differ, $t(80) = -0.19, p = .848$. However, trauma count was higher in the RPT (M = 2.12, SD = .70) than COPE (M = 1.74, SD = .79) group, $t(80) = -2.28, p = .025$. Table 1 presents correlations between primary study variables.

3.2. Trauma Characteristics and PTSD Responses During Treatment

Tables 2 and 3 present primary study findings. With respect to primary study analyses during the active treatment phase, there was a main effect of time on PTSD symptoms such that, across both conditions, PTSD symptoms decreased over the course of treatment (B = -2.15, SE = 0.37), 95% CI [-2.87, -1.42], $\chi^2(1) = 60.79, p < .001$. There were no main effects of trauma age or trauma count, or interactions involving these variables, predicting PTSD symptoms.

3.3. Trauma Characteristics and SUD Responses During Treatment

For SUD outcomes during the active treatment phase, the only significant interaction was a two-way time \times trauma age interaction (B = -0.01, SE = 0.00), 95% CI [-0.01, -0.002], $\chi^2(1) = 10.92, p = .001$. Figure 1 reflects this interaction wherein average days of past week substance use are represented at weeks 1 through 14 at three levels of trauma age: one standard deviation or more below mean trauma age, one standard deviation within mean trauma age, and one standard deviation or more above mean trauma age. Interpretation of parameter estimates and this plot suggest that earlier trauma ages predicted less reduction in SUD during the treatment phase.

3.3. Trauma Characteristics and PTSD and SUD Responses After Treatment

With respect to PTSD outcomes during the post-treatment phase, there was a trend-level, but ultimately not statistically significant, three-way group \times time \times trauma count interaction ($B = -3.28$, $SE = 1.74$), 95% CI $[-6.70, 0.14]$, $\chi^2(1) = 3.54$, $p = .06$. There was a significant group \times trauma count interaction predicting PTSD symptoms, which suggested that a higher trauma count was associated with higher PTSD severity over the follow-up period in the COPE condition more so than in the RPT condition, ($B = 58.46$, $SE = 27.27$), 95% CI $[5.01, 111.91]$, $\chi^2(1) = 4.60$, $p = .03$, but was not related to the magnitude of change in PTSD following treatment. With respect to SUD outcomes during the post-treatment phase, there were no main effects of trauma age or count, or interactions involving them, predicting SUD symptoms.

4. Discussion

This study examined the role of two trauma characteristics associated with the construct of complex PTSD—age and count—by investigating in PTSD+SUD treatment. It also examined if these trauma characteristics functioned differentially in an exposure-based, integrated treatment for PTSD+SUD versus a SUD-focused intervention. Contrary to hypotheses, findings showed that neither trauma characteristic predicted PTSD responses during treatment for COPE or RPT. However, earlier trauma age, but not higher trauma count, predicted less reduction in substance use during treatment across both treatment conditions. Although there was a stronger relationship between trauma count and PTSD following COPE compared to RPT, trauma count and age did not influence the rate or magnitude of PTSD or SUD change after either treatment.

4.1. Impact of Trauma Characteristics on PTSD Responses

The lack of an association between trauma age and PTSD response is consistent with literature suggesting that trauma age does not influence PTSD symptom change during or after

treatment (e.g., Ford & Kidd, 1998; Jaycox et al., 1998). Trauma count also did not influence change in PTSD during treatment, although a higher trauma count was associated with more severe PTSD in the post-treatment phase to a greater extent following COPE than RPT. The exposure component of COPE involves actively processing trauma memories and memory recall (Foa et al., 2007). Given that COPE processes one traumatic experience at a time, having a higher number of traumatic experiences that are not addressed in COPE may have a greater impact on PTSD severity during follow-up than it would in a treatment that does not directly process trauma memories or discuss PTSD content at all (e.g., RPT). However, it is important to note that even though trauma count was more strongly associated with PTSD symptoms in COPE than in RPT during follow-up, it did not influence the magnitude of change in PTSD symptoms during or following treatment. Across both RPT and COPE, trauma age and count did not influence the magnitude of change in PTSD symptoms before and after treatment. Together, these findings suggest that the possibility that a trauma memory may be less intact due to age, or that it is only one of many available memories, does not compromise efficacy of COPE. Our findings thus fit in a broader literature that does not support excluding patients from trauma-processing models like PE on the basis of trauma characteristics and with concern for their potential “complexity” (e.g., Tripp, Jones, Back, & Norman, 2019; van Minnen et al., 2002).

It is also possible that other trauma characteristics relevant to the construct of complex PTSD that were not measured influence PTSD responses. For example, proponents of complex PTSD constructs suggest that PTSD presentations are complicated by early and repeated *interpersonal* traumas that disrupt developing attachment systems (e.g., Briere & Spinazzola, 2005; van der Kolk et al., 2005). The extent to which a trauma is interpersonal could moderate PTSD responses more than age or count, but evidence is mixed (e.g., van Minnen et al., 2002).

4.2. Impact of Trauma Characteristics on SUD Responses

That individuals with an earlier trauma age, but not a higher trauma count, exhibited less improvement in substance use during treatment is consistent with our hypotheses and research suggesting that earlier trauma ages are linked with heightened SUD severity (e.g., Farrugia et al., 2011; Müller et al., 2015; Schumacher et al., 2006; Waldrop et al., 2007). This effect was found across COPE and RPT, suggesting that trauma age influences SUD outcomes regardless of whether or not trauma is directly targeted in treatment. Earlier trauma ages are positively correlated with heightened PTSD severity (Langeland et al., 2004), and PTSD severity, in turn, is associated with worse SUD outcomes (e.g., Ouimette, Ahrens, Moos, & Finney, 1997). It is possible that this heightened PTSD severity hinders SUD improvement even if the rate of change in PTSD during treatment is comparable to those with later trauma ages. However, the absence of a main effect of trauma age on PTSD severity suggests that the impact of trauma age on PTSD severity is unlikely to fully account for its influence on SUD responses.

Visual inspection of our figure shows that individuals with earlier trauma ages may exhibit mild worsening of symptoms throughout treatment, although this remains speculative. Perhaps this is because individuals with earlier trauma ages exhibit distinct SUD profiles than those with older trauma ages with comorbid PTSD+SUD. Trauma survivors with PTSD often seek out substances to manage PTSD-related distress (Haller & Chassin, 2014). Accordingly, earlier trauma is associated with an earlier age of first substance use (Waldrop et al., 2007), and may contribute to use of a greater variety of substances (Farrugia et al., 2011). If individuals with earlier trauma ages also began using substances earlier and use a variety of substances to regulate PTSD symptoms (i.e., polysubstance use), then, regardless of SUD severity, their SUD may be more longstanding, complex, and difficult to treat. Alternatively, although we controlled for

some socioeconomic status indicators, we did not control for all. Trauma age may not directly influence SUD outcomes, but socioeconomic correlates other than education and employment (i.e., neighborhood location) may. Future research should disentangle the effects of other socioeconomic indicators from trauma characteristics on SUDs.

Although trauma age predicted less SUD reduction during treatment, it did not predict SUD symptom change post-treatment. This suggests that trauma age may not negatively affect the maintenance of gains, nor does it result in delayed improvements after treatment. Individuals with early trauma age thus appear to benefit less during treatment, and then maintain their benefits or lack thereof posttreatment compared to others. Identifying ways to optimize within-treatment change in substance use for those with early trauma ages is thus indicated.

In the current study, trauma count did not influence SUD outcomes. Whereas earlier trauma ages may directly influence the temporal onset of substance use in the lifespan, trauma count may not. Therefore, trauma count may not necessarily lead to more ingrained, long-standing, SUD problems that respond less favorably to treatment. Further, it is also possible that early trauma age disrupts developing emotion and interpersonal regulation systems that could obstruct SUD treatment responses, but that a higher trauma count does not *necessarily* do this. However, as we did not examine the mediating role of emotion regulation problems between trauma characteristics and SUD treatment responses, this assertion remains conjecture.

4.3. Limitations

Several limitations in the present study should be noted. The lack of significant effects may be due to low statistical power, given the small sample size in both treatment arms. Despite this, Mills and colleagues (2016) demonstrated significant effects of trauma count on PTSD responses in COPE with similar sample sizes to the present study. Nevertheless, future research

should replicate this study's questions in a larger sample. There are also trauma characteristics beyond trauma age and count (i.e., duration and severity of trauma; relationship with perpetrator, response of social network) that influence PTSD (e.g., Ullman, Filipas, Townsend, & Starzynski, 2007; Ullman & Peter-Hagene, 2014). Investigating more trauma characteristics, their correlates, and the way that they operate on outcomes would illuminate how these variables do and do not impact PTSD+SUD treatment. In addition, it is likely that some participants used more than their primary problematic substance during and following the treatment period, but we did not examine the impact of trauma characteristics on concurrent or other substance use beyond the primary substance. Future researchers are therefore advised to investigate the impact of trauma treatment on substance use outcomes in general, rather than solely the substance of most problematic use, particularly since many use multiple substances concurrently. Finally, we did not examine whether trauma characteristics predict treatment dropout and, as is typical with PTSD+SUD samples (Roberts et al., 2015), dropout rates in this study were high. It therefore remains unclear whether (a) trauma characteristics influence dropout and (b) study findings generalized to those who dropped out.

4.4. Conclusions

Despite these limitations, there are several conclusions that can be drawn from study findings. Our findings suggest that excluding individuals from exposure-based treatments and assuming that they instead need specialized treatment for a complex form of PTSD on the basis of trauma characteristics is not empirically supported. However, future studies should seek to replicate our results with larger samples. Individuals with earlier trauma ages may require additional attention or specialized treatment strategies to show comparable SUD responses to others, and future researchers should investigate this possibility further.

5. References

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Table 1

Correlations between baseline primary variables of interest

	1.	2.	3.	4.	5.	6.	7.
1. Baseline past week substance use	--						
2. Baseline past week PTSD severity	0.16	--					
3. Sex	-0.05	-0.03	--				
4. Baseline days of employment (past 30 days)	-0.04	-0.23*	0.04	--			
5. Years of education completed	-0.03	-0.01	-0.10	0.05	--		
6. Trauma age	0.24*	0.02	0.09	-0.23*	-0.08	--	
7. Trauma count	-0.11	0.01	-0.09	0.07	0.15	-0.39***	--

Note. * $p < .05$; ** $p < .01$; *** $p < .001$. All variables measured at baseline.

Table 2

Generalized Estimating Equations Examining if Trauma Characteristics Predict PTSD Response

	B (SE)	χ^2	df	p-value
Intercept	57.15 (4.83)	328.27	1	<.001
Education (years)	-1.16 (1.06)	1.19	1	.257
Days of employment (past 30 days)	-0.66 (0.27)	5.92	1	.015
Sex	-1.52 (5.05)	0.09	1	.764
Group	0.01 (6.65)	0.00	1	.999
Time	-2.15 (0.37)	60.79	1	<.001
Trauma age	0.14 (0.28)	0.01	1	.912
Trauma count	-1.72 (6.72)	0.00	1	.977
Time × group	-0.12 (0.56)	0.05	1	.826
Time × trauma age	-0.01 (0.02)	0.10	1	.758
Time × trauma count	-0.35 (0.54)	0.01	1	.921
Group × trauma age	-0.24 (0.39)	0.37	1	.545
Group × trauma count	3.19 (9.26)	0.12	1	.730
Group × time × trauma age	0.00 (0.04)	0.01	1	.933
Group × time × trauma count	0.77 (0.78)	0.97	1	.324

Note. Statistically significant effects are bolded. Group = Co-Occurring Prolonged Exposure and Relapse Prevention (COPE; Back et al., 2014) versus Relapse Prevention Therapy (RPT; Carroll, 1998; Marlatt & Donovan, 2007); trauma age and count were grand mean centered. RPT = reference category.

Table 3

Generalized Estimating Equations Examining if Trauma Characteristics Predict SUD Response

	B (SE)	χ^2	df	p-value
Intercept	1.36 (0.14)	176.25	1	<.001
Sex	0.15 (0.22)	0.45	1	.502
Education (years)	-0.01 (0.04)	0.03	1	.863
Days of employment (past 30 days)	0.01 (0.01)	0.16	1	.689
PTSD severity	0.00 (0.00)	4.13	1	.042
Group	-0.08 (0.18)	0.20	1	.657
Time	-0.17 (0.03)	42.31	1	<.001
Trauma age	0.02 (0.01)	5.93	1	.015
Trauma count	0.14 (0.18)	0.03	1	.875
Time × group	0.09 (0.04)	5.65	1	.017
Time × trauma age	-0.01 (0.00)	10.92	1	.001
Time × trauma count	-0.02 (0.04)	0.20	1	.654
Group × trauma age	-0.01 (0.01)	1.12	1	.290
Group × trauma count	-0.23 (0.26)	0.79	1	.375
Group × time × trauma age	0.00 (0.00)	2.06	1	.152
Group × time × trauma count	0.02 (0.05)	0.22	1	.639

Note. Statistically significant effects are bolded. Group = Co-Occurring Prolonged Exposure and Relapse Prevention (COPE; Back et al., 2014) versus Relapse Prevention Therapy (RPT; Carroll, 1998; Marlatt & Donovan, 2007); trauma age and count were grand mean centered. RPT = reference category.

Table 4

Generalized Estimating Equations Examining if Trauma Characteristics Predict PTSD Response from the Last Session Across Follow Up Periods

	B (SE)	χ^2	df	p-value
Intercept	34.37 (9.79)	8.02	1	.005
Sex	-2.80 (5.24)	0.29	1	.593
Group	-4.66 (21.86)	0.05	1	.831
Time	-0.17 (0.63)	0.01	1	.937
Education (years)	-2.59 (1.13)	5.30	1	.021
Days of employment (past 30 days)	-0.74 (0.29)	6.51	1	.011
Trauma age	-0.69 (0.62)	0.53	1	.465
Trauma count	-23.59 (16.85)	0.17	1	.683
Time × group	0.23 (1.32)	0.03	1	.861
Time × trauma age	0.05 (0.05)	0.62	1	.430
Time × trauma count	1.36 (1.14)	0.10	1	.748
Group × trauma age	-0.28 (2.26)	0.02	1	.903
Group × trauma count	58.46 (27.27)	4.60	1	.032
Group × time × trauma age	0.01 (0.14)	0.00	1	.952
Group × time × trauma count	-3.28 (1.74)	3.54	1	.060

Note. Statistically significant effects are bolded. Group = Co-Occurring Prolonged Exposure and Relapse Prevention (COPE; Back et al., 2014) versus Relapse Prevention Therapy (RPT; Carroll, 1998; Marlatt & Donovan, 2007); trauma age and count were grand mean centered. RPT = reference category.

Table 5

Generalized Estimating Equations Examining if Trauma Characteristics Predict SUD Response from the Last Session Across Follow Up Periods

	B (SE)	χ^2	df	p-value
Intercept	-1.10 (1.15)	0.34	1	.559
Sex	0.68 (0.33)	4.15	1	.042
Education (years)	0.11 (0.07)	2.81	1	.093
Days of employment (past 30 days)	0.00 (0.02)	0.05	1	.829
PTSD severity	0.00 (0.00)	1.00	1	.319
Group	0.70 (1.42)	0.24	1	.625
Time	0.04 (0.08)	0.91	1	.341
Trauma age	-0.04 (0.05)	1.07	1	.302
Trauma count	0.42 (1.71)	0.01	1	.942
Time × group	0.02 (0.09)	0.03	1	.864
Time × trauma age	0.00 (0.00)	0.40	1	.529
Time × trauma count	0.01 (0.12)	0.06	1	.810
Group × trauma age	-0.01 (0.09)	0.00	1	.948
Group × trauma count	-0.98 (1.98)	0.24	1	.623
Group × time × trauma age	0.00 (0.01)	0.07	1	.789
Group × time × trauma count	0.02 (0.13)	0.02	1	.900

Note. Statistically significant effects are bolded. Group = Co-Occurring Prolonged Exposure and Relapse Prevention (COPE; Back et al., 2014) versus Relapse Prevention Therapy (RPT; Carroll,

1998; Marlatt & Donovan, 2007); trauma age and count were grand mean centered. RPT = reference category.

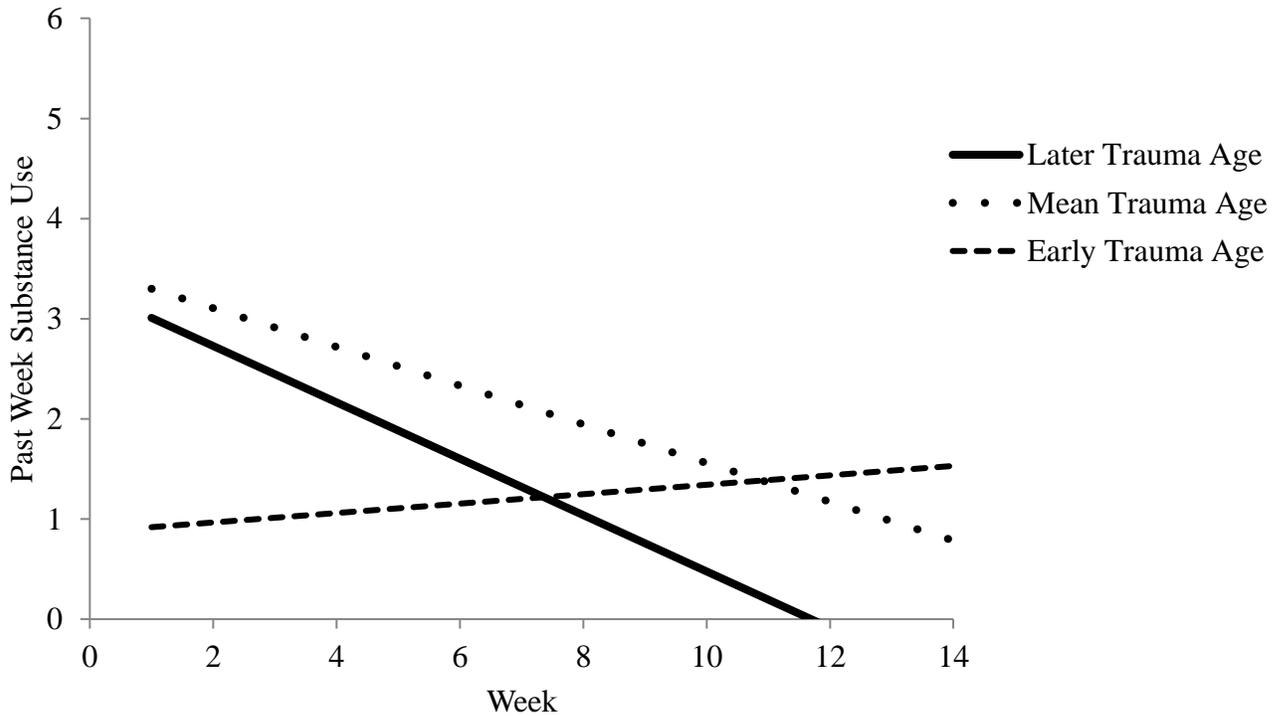


Figure 1. Graphical representation of time \times trauma age interaction on weekly substance use frequency (mean days of use).

Note. X-axis values reflect study week. Each data series reflects values for individuals one standard deviation or more below mean trauma age (Early Trauma Age), one standard deviation within mean trauma age (Mean Trauma Age), and one standard deviation or more above mean trauma age (Later Trauma Age)