Journal of Substance Abuse Treatment 62 (2016) 38–48

Contents lists available at ScienceDirect

Journal of Substance Abuse Treatment

Integrated Cognitive Behavioral Therapy Versus Cognitive Processing Therapy for Adults With Depression, Substance Use Disorder, and Trauma

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ARTICLE INFO

Article history:
Received 12 June 2015
Received in revised form 3 November 2015
Accepted 11 November 2015

Keywords:
Cognitive processing therapy
Substance use disorder
PTSD
Depression
Integrated treatment

ABSTRACT

The comorbidity of substance use disorder (SUD), depression, and PTSD is common among veterans. Prior research has shown that among veterans with SUD and depression, those with PTSD did not maintain cognitive-behavioral treatment gains as well as those without PTSD. Thus, the current study was designed to evaluate whether adding trauma-focused treatment following an initial group-based integrated cognitive behavioral treatment (ICBT) for SUD and depression improved treatment outcomes. Participants were 123 veterans (89% male) recruited from the VA San Diego Healthcare System. All participants received ICBT in twice weekly, group-delivered sessions for 12 weeks (Phase 1). Participants were then randomized to receive 12 sessions of individual follow-up sessions (Phase 2) utilizing either ICBT or cognitive processing therapy that was modified to integrate SUD treatment (CPT-M). Results indicated that PTSD and depression symptoms slightly improved at the end of Phase 1 group ICBT and further improved through Phase 2 individual treatment (except for participants without PTSD who received CPT-M), with treatment gains maintained one year later. Substance use significantly improved at the end of Phase 1 group ICBT and these improvements were maintained through Phase 2 and the one year follow-up. Participants in the trauma-focused Phase 2 treatment (CPT-M) exhibited similar levels of symptom reduction and maintenance of treatment gains as those in the non-trauma-focused Phase 2 treatment (ICBT). However, there was a slight advantage for Phase 2 CPT-M over Phase 2 ICBT with respect to heavy drinking outcomes for individuals with PTSD. Overall, the combination of group ICBT followed by either CPT-M or ICBT individual therapy appears to be effective for veterans with depression, SUD, and trauma history.

1. Introduction

Substance use disorders (SUDs) and depressive disorders are highly prevalent (Grant et al., 2004) and frequently co-occur (Currie et al., 2005; Kessler et al., 2003). Integrated treatments have been shown to be helpful in reducing symptoms of both disorders (Kay-Lambkin, Baker, Lewin, & Carr, 2009; Lydecker et al., 2010). Studies have also shown high rates of Posttraumatic Stress Disorder (PTSD) in SUD treatment settings (37%: Bonin, Norton, Asmundson, Dicurzio, & Pidlubney, 2000; 63%: Stewart, Conrod, Samoluk, Pihl, & Dongier, 2000), depression treatment settings (13%: Felker, Kirchner, Chan, & Rubenstein, 2007; 36%: Carlier, Voerman, & Gersons, 2000), and in co-occurring SUD and depression clinical samples (38%: Norman, Tate, Wilkins, Cummins, & Brown, 2010). PTSD is associated with worse treatment response and poorer maintenance of treatment gains among substance dependent samples, depression samples, and co-occurring SUD and depression samples (Driessen et al., 2008; Green et al., 2006; Hegel et al., 2005; Holtzheimer, Russo, Zatzick, Bundy, & Roy-Byrne, 2005; Norman et al., 2010; Ouimette, Brown, & Najavits, 1998).

Although PTSD is associated with worse substance use and depression outcomes, clinicians have expressed concern that treating PTSD prior to substance use could lead to unsafe coping (i.e., substance use, suicidality), thereby increasing risk for clinical crises (Brady, Killeen, Brewerton, & Lucerini, 2000; Ford, Russo, & Mallon, 2007; Souza & Spates, 2008; Weis, 2010). A recent meta-analysis of psychological interventions for comorbid PTSD/SUD (Roberts, Roberts, Jones, & Bisson, 2015) contradicts this notion and found better PTSD and follow-up (5 to 7 months) substance use outcomes for exposure-based treatment compared to treatment as usual, but also found high treatment dropout across all studies and somewhat higher dropout for exposure-based interventions. Similarly, a review of PTSD/SUD treatment found the

Abbreviations: SUD, substance use disorder; ICBT, integrated cognitive behavioral therapy; CPT-M, cognitive processing therapy- modified; VASDHS, Veterans Affairs San Diego Healthcare System; HDRS, Hamilton Depression Rating Scale; PCL, PTSD checklist; PDA, percentage of days abstinent; LME, linear mixed effects.

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http://dx.doi.org/10.1016/j.jsat.2015.11.005
0740-5472/

strongest evidence for addressing PTSD and SUD concurrently rather than in a sequential fashion, and that this approach is also favored by patients (McCauley, Killeen, Gros, Brady, & Back, 2012). Yet, a systematic review of concurrent PTSD/SUD treatments found that concurrent treatments in general do not appear to be advantageous; rather, only those that are specifically trauma-focused show superior PTSD and SUD outcomes (van Dam, Vedel, Ehring, & Emmelkamp, 2012). The authors define trauma-focused approaches as those that focus on the memory and meaning of the traumatic event, whereas non-trauma focused therapies focus on present or past aspects of life other than the trauma. All the aforementioned reviews also make clear a need for future randomized controlled efficacy trials with adequate randomization, variant high-risk populations, long-term follow-ups, and active comparison groups (McCauley et al., 2012; Roberts et al., 2015; van Dam et al., 2012).

Some studies of PTSD/SUD treatments demonstrate that depression symptoms often improve along with PTSD and SUD improvements (see McCauley et al., 2012). Current PTSD treatment guidelines acknowledge that severe depression may limit the effectiveness of PTSD treatment, and that addressing depression first may sometimes be helpful (Foa, Keane, Friedman, & Cohen, 2009). However, a recent study (Hemmy Asamsama, Dickstein, & Chard, 2015) found PTSD treatment (i.e., Cognitive Processing Therapy) to be effective for PTSD even in cases of severe depression (changes in depressive symptoms were not reported). Little research has specifically examined how to best treat individuals who have co-occurring PTSD, SUD, and depression, and whether it may be helpful to address depression first.

In our previous research, we developed Integrated Cognitive Behavioral Therapy (ICBT) for treating veterans with both SUD and depression (Lydecker et al., 2010). ICBT aims to help individuals develop cognitive-behavioral skills that are useful for managing both SUD and depression (e.g., challenging maladaptive cognitions, increasing pleasant activities, building healthier social networks). This treatment was found to be efficacious, with greater attendance associated with more improvement in depression and substance use (Lydecker et al., 2010). Although ICBT successfully reduced substance use and depression symptoms, veterans with a comorbid PTSD diagnosis had worse substance use at the one year follow-up compared to individuals without co-occurring PTSD, despite similar improvements during and immediately following treatment (Norman et al., 2010). Thus, treatment gains were compromised over time when PTSD remained untreated. This research finding was the impetus for the current study.

Specifically, we were interested in testing a two-phased treatment approach in which veterans with SUD, depression, and trauma (most of whom met full PTSD criteria) were first provided with group ICBT during Phase 1 in order to address substance use and depression, and were then randomized in Phase 2 to receive individual therapy for PTSD or individual ICBT (reviewing the skills learned in Phase 1). We opted to include trauma-exposed individuals both with and without current PTSD, given that little is known about symptom trajectories for individuals with subthreshold PTSD, despite research indicating that individuals with subthreshold PTSD experience comparable functional impairments to those with PTSD (Norman, Stein, & Davidson, 2007). Our two-phased research design allowed us to test whether specifically addressing PTSD in Phase 2 improves outcomes for individuals with PTSD. Providing the interventions in this sequence capitalizes on the benefits of developing skills for reducing substance use and affective distress using a cost-effective group format prior to initiating a trauma-focused intervention delivered individually. Providing the Phase 2 intervention individually allowed for greater flexibility in scheduling in order to improve attendance as many veterans were expected to return to work or other responsibilities, and also allowed for discussion of variable trauma types and sensitive trauma issues in a private setting (we expected this to be important given the diversity of trauma types reported in veteran samples).

All participants received ICBT in twice weekly, group-delivered sessions for 12 weeks (Phase 1). We then randomized participants to receive follow-up ICBT or Cognitive Processing Therapy (CPT; Resick, Monson, & Chard, 2008) for 12 sessions (Phase 2). CPT was modified for this study (CPT-M) to also address cognitions relevant to SUD relapse prevention within the CPT framework. Treatment during Phase 2 (both ICBT and CPT-M) was delivered individually in once per week, one-hour sessions. We hypothesized that receiving CPT-M following Phase 1 ICBT treatment would result in greater reductions in substance use, depression symptoms, and PTSD symptoms, and better maintenance of treatment gains during the follow-up time period compared to receiving only ICBT treatment. We also hypothesized that greater attendance during Phase 2 would be associated with better outcomes and maintenance of treatment gains over time, and thus tested attendance as a moderator. This is consistent with our findings from our prior study providing ICBT and the documented association between treatment dose and outcomes (Lydecker et al., 2010). Finally, we also examined PTSD diagnosis as a moderator in order to examine whether treatment effects and symptom trajectories differed between those with current PTSD and those exposed to trauma without current PTSD.

2. Method

2.1. Participants

Participants were 123 outpatient veterans from the Veterans Administration San Diego Healthcare System (VASDHS). The study was approved by the University of California, San Diego and VASDHS Institutional Review Boards. This clinical trial is registered at www.ClinicalTrials.gov as NCT00958217. Participants were recruited from referrals to the outpatient dual diagnosis treatment program from October 2009 to October 2012. Inclusion criteria were: (1) presence of a current DSM-IV diagnosis of alcohol, cannabinol, or stimulant dependence with use in the past 90 days; (2) DSM-IV diagnosis of current major depressive disorder or dysthymia (with at least one lifetime episode occurring independent of alcohol or drug use); and (3) trauma exposure (with or without DSM-IV diagnosis of PTSD). Participants who had an abuse/dependence diagnosis from another drug category were included in the study as long as they also met criteria for an alcohol, cannabinoid, or stimulant disorder diagnosis. Exclusion criteria were the presence of a bipolar or psychotic disorder, living more than 50 miles away, memory deficits sufficient to impair accurate recall for assessments, life threatening or unstable medical illnesses, and participating in CPT within the past year.

Eligible participants were told about the study and provided informed consent to participate in 12 weeks of group ICBT treatment, as well as subsequent randomization to 12 sessions of either individual ICBT or individual CPT-M treatment. Participants were allowed up to 16 weeks to complete 12 sessions of Phase 2 ICBT or CPT-M. Randomization was stratified by gender and current PTSD diagnosis. Participants also consented to assessment interviews (at baseline [we use the term baseline to refer to the intake assessment prior to Phase 1], end of Phase 1, end of Phase 2, and quarterly during one year of follow-up), random toxicology screens, and to not participate in any other formal treatment for PTSD, depression, or substance dependence other than community mutual-help groups and pharmacotherapy. Participants were asked not to participate in other formal treatment only during the active treatment phases, and this was monitored during this timeframe. We did not have any participants who opted to drop out of the study in order to engage in other formal treatment.

A total of 154 veterans met initial screening criteria and completed informed consent. Of these, 123 (79.9%) were included in the present study. Participants were excluded from the present study ($n = 31$) if they were unable to be randomized into Phase 2 treatment because they died, moved, refused, were not psychiatrically or medically stable, were lost to follow-up before completing the baseline assessment, did not meet study criteria, or if recovery home requirements would not allow participation. Note that we did not have any exclusion criteria

pertaining to medication in order to make our study as naturalistic and generalizable as possible. Fig. 1 describes the flow of participants at each stage of the study. There were no significant differences between included and excluded participants on baseline PTSD symptoms, depression symptoms, percentage days abstinent (PDA), demographic variables, or prescribed medication, with the exception of monthly income (included participants had higher monthly incomes). Table 1 lists sample characteristics for the full sample and for each treatment group.

2.2. Procedure

In the first 12 weeks of treatment (Phase 1), all participants received the group-delivered ICBT intervention, which met twice weekly for 60 minutes. Participants were then randomized to receive 12 weekly 60-minute individual sessions of either ICBT or CPT-M (Phase 2). Participants in both interventions were offered pharmacotherapy appointments with VA psychiatric medical providers and psychotropic medications were prescribed using standard VA clinical protocol.

2.3. Interventions

The ICBT group intervention (Lydecker et al., 2010) included cognitive-behavioral strategies for substance use disorder from the Cognitive-Behavioral Coping Skills Training manual (Kadden et al., 1992), as well as cognitive-behavioral strategies for depression from the Group Therapy Manual for Cognitive-Behavioral Treatment of Depression (Muñoz & Miranda, 1996). The primary focus of treatment was on teaching participants to manage negative cognitions related to depression and substance use, increase pleasurable activities to promote positive mood and reduce substance relapse risk, and build healthy social networks. Specific trauma-related thoughts were not explored or challenged as part of ICBT. The ICBT individual intervention reviewed the skills taught during the group intervention. For both the

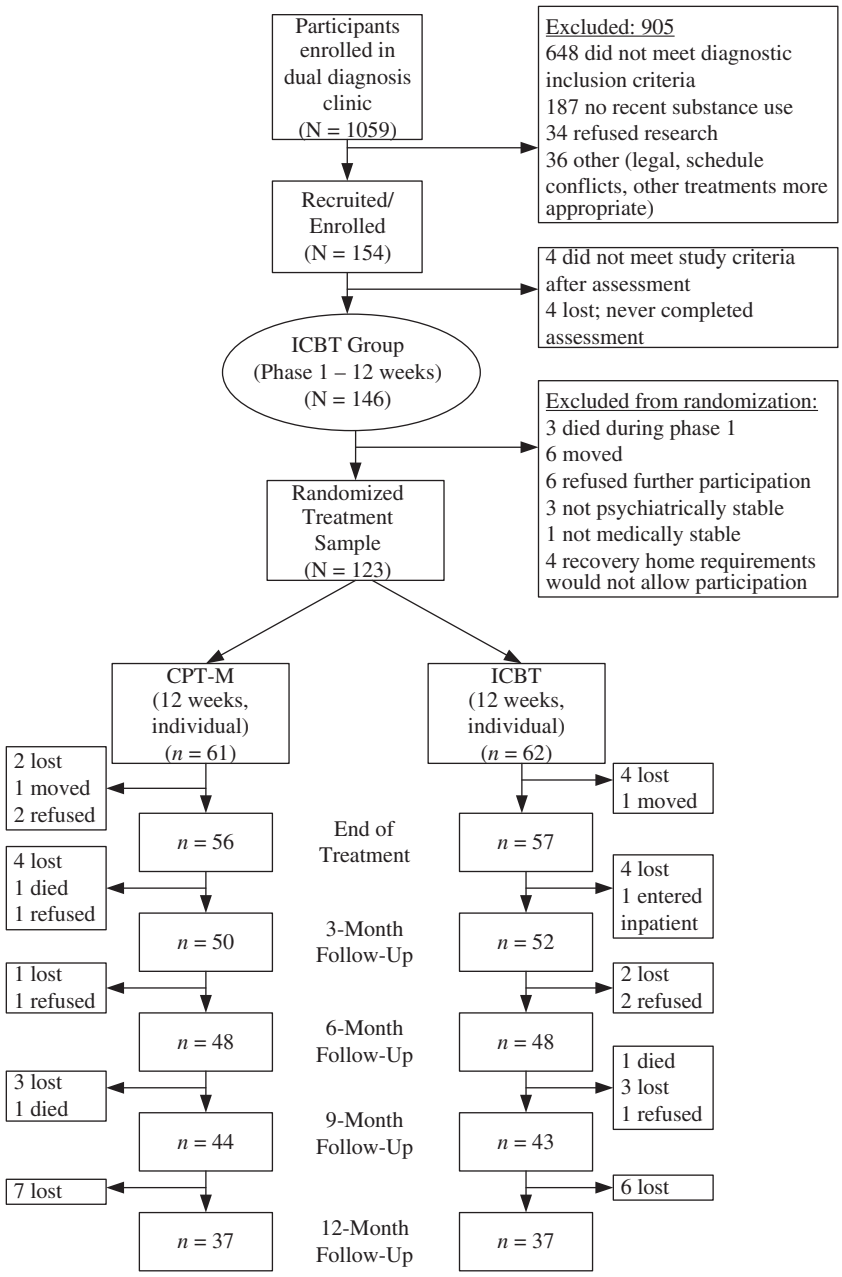

Fig. 1. Participant flow chart. CPT-M = cognitive processing therapy-modified; ICBT = integrated cognitive behavioral therapy.

Table 1
Pre-treatment characteristics of sample (prior to treatment).

Characteristic	Overall	CPT-M	ICBT
	(*N* = 123)	(*n* = 61)	(*n* = 62)
Gender, % male	88.6	90.2	87.1
Mean (*SD*) age (years)	47.26 (11.97)	47.20 (12.12)	47.32 (11.92)
Marital status, %			
Married	21.1	21.3	21.0
Divorced/widowed/separated	54.5	59.0	50.0
Never married	24.4	19.7	29.0
Mean (SD) education (years)	12.96 (3.02)	13.11 (2.38)	12.81 (3.55)
Ethnicity, %			
Non-Hispanic Caucasian	64.2	65.6	62.9
Hispanic	16.2	14.7	17.7
African-American	9.8	9.8	9.7
Asian or Pacific Islander	4.9	4.9	4.8
Other	4.8	4.9	4.8
Mean (*SD*) monthly income ($)	2268 (2263)	2057 (2070)	2478 (2440)
Treatment history			
Mean (*SD*) lifetime substance treatments	2.09 (2.25)	2.20 (2.08)	1.98 (2.42)
Mean (*SD*) lifetime psychiatric treatments	1.76 (2.90)	1.93 (3.15)	1.58 (2.65)
PTSD diagnosis, %			
Never met criteria	14.6	11.5	17.7
Lifetime but not current	3.3	4.9	1.6
Current	82.1	83.6	80.6
Trauma type			
Combat, %	44.2	42.4	45.9
Sexual, %	32.5	28.8	36.1
Other, %	28.3	33.9	23.0
Substance use disorder diagnosis, %			
Current alcohol disorder only	41.5	39.3	43.5
Current drug disorder only	13.8	13.1	14.5
Current alcohol and drug disorder	44.7	47.5	41.9
Mean (*SD*) percent days abstinent (PDA)	.43 (.27)	.40 (.25)	.47 (.29)
Mean (*SD*) PCL score	56.99 (13.30)	58.38 (12.37)	55.53 (14.19)
Mean (*SD*) HDRS score	33.00 (10.86)	33.37 (9.70)	32.63 (11.99)

Note. *Ns* vary slightly across assessments due to missing data. CPT-M = cognitive processing therapy-modified; ICBT = integrated cognitive behavioral therapy; PCL = PTSD checklist; HDRS = Hamilton Depression Rating Scale

group and individual ICBT interventions, the therapist and patients used the same manual.

The CPT-M intervention modified trauma-focused CPT (Resick et al., 2008) to include substance relapse prevention. CPT addresses trauma-related thoughts, whether these thoughts are consistent or inconsistent with patients' prior schemas about the world, and the impact these thoughts have on emotions and behaviors. Patients are taught to recognize and challenge maladaptive thought patterns and common issues associated with trauma responses (e.g. safety, trust, power/control, esteem, and intimacy). CPT has been used successfully with patients exposed to combat trauma and other types of trauma (e.g., Monson et al., 2006; Resick & Schnicke, 1992). We modified CPT specifically for this study in order to include brief check-ins regarding any recent substance use or cravings at the beginning of sessions, as well as incorporation of substance use cognitions in examples within the CPT framework (e.g., using Challenging Belief Worksheets). Therapists had a brief supplemental manual for the CPT-M condition in addition to the standard CPT manual. No other modifications were made to CPT. We did not modify CPT to address depression because depression was already addressed in Phase 1, and because CPT has been found to produce improvements in depression along with PTSD symptoms (Resick & Schnicke, 1992). Also, note that recent research indicates that CPT is effective even in cases of severe co-occurring depression (Hemmy Asamsama et al., 2015). Although CPT is an intervention specifically for PTSD, we expected that CPT-M may have benefits for trauma-exposed participants without current PTSD by addressing subthreshold PTSD symptoms and preventing relapse, both of which may also benefit depression. However, it should be noted that no studies have tested or demonstrated efficacy of CPT for individuals without PTSD.

Thirteen therapists provided the study interventions. Therapists were trained in and provided both therapies. Therapists included the Principal Investigator, clinical psychology postdoctoral fellows, interns, or advanced (4th year) graduate students. For CPT-M, therapists completed the standard training for CPT and received additional training related to the modifications for substance relapse prevention. For ICBT, therapists received training using the standard procedures in the dual diagnosis outpatient clinic. Group ICBT is/was the standard treatment used in the dual-diagnosis clinic for patients with substance disorders and depression, and has been so since March 2000. Therapists received both individual and group supervision, and also received CPT-M consultation from a VA national CPT trainer. All therapy sessions were recorded and available for review in supervision; a random selection (10%) were reviewed to ensure fidelity to the manual and avoid contamination of content. Weekly clinical supervision was provided to all therapists by staff clinicians experienced in the interventions.

2.4. Measures

2.4.1. Clinical diagnoses

Baseline DSM-IV PTSD diagnoses were assessed using the Clinician Administered PTSD Scale (CAPS; Blake et al., 1995), a semi-structured interview with excellent psychometric properties (Weathers, Keane, & Davidson, 2001). The CAPS assesses for the type of trauma experienced and the severity of PTSD symptoms. Symptoms were considered present if clinicians gave a frequency rating of one or more and an intensity rating of two or more (Weathers et al., 2001). Interrater reliabilities for the CAPS are typically close to .90 (Weathers et al., 2001). The average total score on the CAPS was 70.31 (*SD* = 24.97; range: 13.00–116.00).

The Composite International Diagnostic Interview (CIDI; Robins et al., 1988) was used to assess for other baseline DSM-IV diagnoses, including depression and SUDs. It has been used to diagnose depression and substance use in national and international epidemiological studies (e.g., Darves-Bornoz et al., 2008; Kessler, Chiu, Demler, & Walters, 2005), and distinguishes between substance-induced and independent psychiatric diagnoses.

2.4.2. Psychological symptoms and substance use

Measures of PTSD symptoms, depression symptoms, and substance use were administered at baseline and at quarterly intervals throughout the study (through the one-year follow-up).

2.4.2.1. Depression symptoms. The Hamilton Depression Rating Scale (HDRS; Hamilton, 1960) is a 21-item structured clinical interview assessment of depression symptom items over the past week rated on a 0 (none) to 4 (most severe) scale. The HDRS has been found to have good sensitivity and specificity among SUD populations (Willenbring, 1986).

2.4.2.2. PTSD symptoms. PTSD symptoms were measured via the 17-item PTSD Checklist–Civilian. (PCL-C; Weathers, Litz, Huska, & Keane, 1994), a self-report measure of PTSD symptoms over the last month rated on a 1 (not at all) to 5 (extremely) scale. The civilian version was chosen because it does not restrict the nature of the trauma.

2.4.2.3. Substance use. Frequency of alcohol and drug use and quantity of alcohol use (past 90 days) were assessed using the Timeline Followback (TLFB; Sobell & Sobell, 1992), a calendar-assisted structured clinical interview. The TLFB has documented validity and reliability in individuals with psychiatric disorders and comorbid alcohol and/or substance dependence (e.g., Carey, Carey, Maisto, & Henson, 2004). Random toxicology screens were conducted to maximize the reliability of the participants' self-reported substance use. The TLFB was corrected based on positive toxicology screens and subsequent discussions with participants in order to get a more accurate timeline. Summary PDA scores were calculated at each time point. Trajectory analyses examined two substance use outcomes:

(1) probability of any alcohol or drug use on a given day, and (2) probability of heavy drinking (5 or more drinks consumed in a day) on a given day.

2.5. Data analytic strategy

SPSS Version 20 was used to conduct all preliminary and Phase 1 analyses, and R Version 3.1 and Stata Version 14 were used to conduct Phase 2 trajectory analyses. Linear mixed effects (LME) models (Frees, 2004; West, Welch, & Galecki, 2007) were used to ascertain trajectories for PTSD symptoms (PCL scores) and depression symptoms (HDRS scores). Trajectories of substance use (any alcohol or drug use on a particular day) and heavy drinking (>5 drinks on a particular day) were modeled as dichotomous outcomes, using logit links to predict the probability of substance use or heavy drinking on a particular day. Participant level intercepts were modeled as random effects in all the models. Models were estimated with maximum likelihood methods. Sets of parameter estimates were collectively tested for significance with likelihood ratio tests, which are more reliable than score or Wald tests (Harrell, 2001). Tests for interactions were based on comparing models with and without the first and second order time interactions. The trajectories modeled responses from the end of Phase 1 (point of randomization) to the one year follow-up. Thus, these models included data from six different assessments that span approximately 15 months: end of Phase 1 (i.e., Time 0 in our clinical trajectory analyses), end of Phase 2 (3–4 months later), and four follow-up assessments conducted approximately 3 months apart. This analytic approach was advantageous in that it allowed for examination of change in treatment outcomes across all of Phase 2 and the one year follow-up.

Because follow-up assessments were not always conducted on exactly the desired date, we computed the number of days that had elapsed since the end of Phase 1 assessment for each additional assessment and converted this variable into months to facilitate interpretation. Thus, our time variable reflected months elapsed since Phase 1. We modeled time as a second-order polynomial in order to allow for curvilinear trajectories, as we expected the temporal dynamics to be complex. In models where diagnostics revealed problematic collinearity, time was re-parameterized as an orthogonal polynomial (Bright & Dawkins, 1965; Narula, 1978).

For each outcome, we tested whether treatment group interacted with time in order to examine whether trajectories differed across treatment condition (Model 1). Separate models examined attendance (number of Phase 2 therapy sessions attended; Model 2), and baseline PTSD diagnosis (Model 3) as predictors. Note that attendance was modeled as a continuous variable but that interactions were probed by examining outcomes at high (11 or more sessions) and low (3 or fewer sessions) attendance. Additional models also tested the effect of gender; however, gender did not significantly alter symptom trajectories in any model for any outcome and thus these results are not reported. Models included interactions between the predictor, time, and treatment group. Intercepts were specified as random variables and slopes as fixed variables. Cases with incomplete data were included as LME models can accommodate missing data under the missing-at-random assumption (Singer & Willett, 2003).

3. Results

3.1. Group equivalence and sample characterization

Participants' pretreatment (i.e., prior to Phase 1 treatment) substance use levels, depression levels, and PTSD levels are shown in Table 1. The CPT-M and ICBT groups were similar (see Table 1) with respect to demographic variables, substance use at baseline, PTSD symptoms (PCL scores), and depression symptoms (HDRS scores). On average, participants were abstinent fewer than half of the past 90 days at baseline (mean PDA = .43, SD = .27), had clinically elevated (i.e., above 50 on PCL) PTSD symptoms (mean PCL = 56.99, SD = 13.30), and severe depression symptoms (mean HDRS = 33.00, SD = 10.86).

3.2. Attendance

All but three participants attended at least one group ICBT session (Phase 1). On average, those who attended group ICBT attended 14.83 sessions (SD = 6.25) out of 24 possible sessions. Baseline substance use (r = .03, p = .739), PTSD symptoms (r = .02, p = .859), PTSD diagnosis (r = .07, p = .448), and depression symptoms (r = −.11, p = .247) were all unrelated to group attendance during Phase 1. Partial correlations indicated that participants who attended more group ICBT sessions had increased days abstinent from substance use during Phase 1 (pr = .56, p < .001) controlling for baseline levels. However, group ICBT attendance was not significantly related to changes in depression (pr = −.09, p = .348) or PTSD symptoms (pr = −.13, p = .16) at the end of Phase 1.

Following randomization, there were four CPT-M participants who did not attend any individual sessions, and eight ICBT participants who did not attend any individual sessions. Among those who attended at least one individual session, the average number of sessions attended was 7.00 (SD = 4.10) for CPT-M participants and 7.63 (SD = 3.97) for ICBT participants.

For the CPT-M condition, partial correlations indicated that attending a greater number of individual sessions during Phase 2 was associated with decreased PTSD symptoms (pr = −.45, p = .002) and depression symptoms (pr = −.31, p = .049) at the end of Phase 2, controlling for end of Phase 1 scores on these variables. PDA scores were unrelated to CPT-M attendance (pr = .08, p = .577).

For the ICBT condition, partial correlations indicated that attending a greater number of individual sessions during Phase 2 was associated with increased percentage days abstinent (pr = .42, p = .002) and decreased PTSD symptoms (pr = −.29, p = .049) at the end of Phase 2, controlling for end of Phase 1 scores on these variables. There were marginally significant associations between individual ICBT attendance and changes in depression symptoms (pr = −.26, p = .082).

PTSD diagnosis was unrelated to attendance for both CPT-M (r = .11, p = .410) and ICBT (r = .07, p = .585) conditions.

3.3. Pharmacotherapy

Across the course of the study, the percentage of participants taking prescribed medications ranged from 64–73% for antidepressant/PTSD medications, 10–21% for addiction medications, and 12–26% for sleep medications. Percentage of participants taking prescribed medications did not differ between the CPT-M and ICBT conditions. Further, we examined whether there were changes in prescribed medication (antidepressant/PTSD medications, sleep medications, and addictions medications) between the following time points: (1) baseline and end of Phase 1, (2) end of Phase 1 and end of Phase 2, and (3) end of Phase 2 and one year follow-up. The percentage of participants who reported changes in prescribed medications ranged from 4 to 11 percent across these time points and types of medications, indicating that medication use was relatively stable over the course of the study. Further, changes in prescribed medications did not significantly differ between the CPT-M and ICBT conditions at any time point or for any medication type.

3.4. Group ICBT (Phase 1) outcomes

Prior to randomization, participants engaged in 12 weeks of group ICBT, and we hypothesized that there would be significant improvements in substance use, PTSD symptoms, and depression symptoms by the end of Phase 1. The mean PDA score significantly increased from .43 (SD = .27) at baseline to .82 (SD = .28) at the end of Phase 1 treatment ($t(113)$ = −13.77, p < .001, Cohen's d = 1.29, a large effect size). Thus, participants cut the frequency of their substance use by

more than two-thirds, as they went from using 57% of days prior to treatment to using 18% of days at the end of Phase 1. This replicates findings from the Lydecker et al. (2010) study, which found the average PDA score for those who participated in group ICBT to be .84 at the end of treatment. There were no significant associations between demographic variables (gender, age, ethnicity, marital status, education, or income) and PDA scores at the end of Phase 1, controlling for baselines PDA scores.

At the end of Phase 1, mean PCL scores significantly decreased from 56.99 ($SD = 13.30$) to 50.67 ($SD = 15.72$), $t(110) = 5.61$, $p < .001$. A change of 5 points is recommended as a minimum threshold for determining whether an individual has responded to treatment, and a 10 point change is considered clinically meaningful (National Center for PTSD, 2014). The average decrease at the end of Phase 1 was 6.32 points (Cohen's $d = 0.46$, a medium effect size), which may be considered to be a reliable decrease. Although a decrease of 6.32 points on the PCL may not be a clinically meaningful reduction for an individual, this decrease represents a significant overall group reduction in severity of PTSD symptoms. Note that at the end of Phase 1, the average PCL score of 50.67 approached the recommended threshold of 50 for clinically significant PTSD symptoms for a VA mental health population (National Center for PTSD, 2014).

At the end of Phase 1, mean HDRS scores significantly decreased from 33.00 ($SD = 10.86$) to 30.39 ($SD = 12.35$), $t(102) = 3.40$, $p = .001$. The average decrease of 2.61 points on the HDRS is small (Cohen's $d = 0.27$), but does nonetheless indicate an overall significant group reduction in severity of depression (note that the United Kingdom National Institute for Clinical Excellence (2004) defined an improvement of 3 HDRS points as a significant benefit for antidepressants over placebos). However, a decrease of this magnitude would not be considered clinically meaningful for an individual. The small decrease in depression symptoms replicates the previous study (Lydecker et al., 2010), which did not assess PTSD symptoms. Again, no demographic variables were found to be significantly associated with changes in symptoms.

3.5. Phase 2 outcomes

3.5.1. Substance use outcomes

3.5.1.1. Summary statistics. The mean PDA score in the CPT-M condition was .84 ($SD = .26$) at the end of Phase 1 (i.e., prior to starting CPT-M), .81 ($SD = .28$) at the end of individual CPT-M treatment (i.e., Phase 2), and .73 ($SD = .32$) at the one year follow-up. Average PDA scores followed a similar pattern in the ICBT condition: .81 ($SD = .30$) at the end of Phase 1, .79 ($SD = .29$) at the end of individual ICBT treatment (i.e., Phase 2), and .72 ($SD = .40$) at the one year follow-up. Therefore, in both treatment conditions, the percentage days abstinent from substance use stayed almost the same at the end of Phase 2 as compared to the end of Phase 1, but decreased 5% (ICBT) to 8% (CPT-M) by one year after Phase 2 ended. Although PDA scores decreased at the one-year follow-up, reflecting an increase in substance use, participants were still abstinent on approximately 30 percent more days at the one year follow-up than they were prior to treatment (mean baseline PDA = .43).

3.5.1.2. Clinical trajectories of substance use over Phase 2. Table 2 presents results from models predicting trajectories of substance use (any alcohol or drug use), as well as heavy drinking (5 or more drinks in a day). Model predicted probabilities of substance use on a given day were generally low at 4.9% (CI = 2.3%, 7.5%) and 10.7% (CI = 5.5%, 15.8%) at randomization and the end of follow-up, respectively. In Model 1 (see Table 2), there was no evidence to support the hypothesis that CPT-M trajectories demonstrated less frequent substance use than ICBT trajectories. We did find a difference in treatment responses when considering attendance. Attendance was a highly significant moderator of the effect of treatment on substance use trajectories (treatment by time by attendance interaction; see Table 2 Model 2), such that participants with lower attendance in the ICBT condition had worse substance use trajectories over time (see Table 3 for model predicted probabilities of substance use, and Fig. 2 for a graphical depiction of this pattern). PTSD diagnosis also moderated the effect of treatment on substance use trajectories, as reflected in a significant 3-way interaction (treatment by time by PTSD; see Table 2, Model 3). Model predicted probabilities of substance use were consistently lower for participants with PTSD compared to participants without PTSD (see Table 3). Substance use trajectories did not significantly differ across treatment conditions for participants with PTSD but did differ for those without PTSD. Specifically, the probability of substance use stayed about the same across Phase 2 and the one year follow-up for CPT-M participants without PTSD, whereas ICBT participants without PTSD were more likely to use substances over time (see Table 3).

With respect to models predicting trajectories of heavy drinking, we found evidence of more preferable trajectories of heavy drinking in the CPT-M condition as compared to the ICBT condition over time, as indicated by a treatment by time interaction (see Table 2, Model 1). That is, both conditions showed similar probabilities of heavy drinking during Phase 2 treatment, but heavy drinking increased more during the one-year follow-up in the ICBT condition compared to the CPT-M condition. A significant 3-way interaction between treatment, attendance, and time (see Table 2, Model 2) sheds light on the higher probability of heavy drinking in the ICBT condition during the follow-up period. This interaction is depicted in Fig. 3. ICBT participants with low attendance had increasing probabilities of heavy drinking over time, whereas CPT-M participants with low attendance were initially similar but had decreased likelihood of heavy drinking over time (see Table 3). Consistent with substance use findings, trajectories for heavy drinking were low for participants with higher attendance at the end of Phase 2 and remained consistently low over time (see Table 3 and Fig. 3). Trajectory patterns of heavy drinking were also differentially associated with PTSD diagnosis across treatment conditions, as indicated by a time by treatment by PTSD interaction (see Table 2, Model 3). As indicated in Table 3, this interaction appeared to be driven largely by the fact that participants in the CPT-M condition without PTSD had slightly higher heavy drinking trajectories across time (.047 at 3 months follow-up, $SE = 0.050$) relative to all other participants, but declined by the one year follow-up (0.030 at 12 months follow-up, $SE = 0.033$). Participants in the CPT-M condition with PTSD maintained consistently low probability of heavy drinking across all of Phase 2 and the one year follow-up (see Table 3).

3.5.2. PTSD symptoms

3.5.2.1. Summary statistics. The mean PCL score in the CPT-M condition was 51.46 ($SD = 15.48$) at the end of Phase 1 treatment, 49.62 ($SD = 14.04$) at the end of CPT-M Phase 2 treatment, and 48.33 ($SD = 17.14$) at the one year follow-up. In the ICBT condition, the mean PCL score was 49.88 ($SD = 16.06$) at the end of Phase 1 treatment, 46.69 ($SD = 15.74$) at the end of ICBT Phase 2 treatment, and 39.47 ($SD = 16.46$) at the one year follow-up. A one-way ANOVA indicated that PCL scores were significantly lower at the one year follow-up for participants in the ICBT condition, compared to the CPT-M condition, $F(1,71) = 5.58$, $p = .023$. However, this result was based on only those participants who were assessed at the one year follow-up; results from trajectory models that account for missing values (see below) do not indicate significant differences between treatment conditions over time.

We also examined correlations between PTSD diagnosis and PTSD symptoms over time in order to examine whether participants who started the study with PTSD remained significantly elevated on PTSD throughout the study. Participants who began the study with PTSD had higher PTSD symptoms at every time point, including end of Phase 1 treatment ($r = .470$, $p < .001$), end of Phase 2 treatment ($r = .388$, $p < .001$), and one year follow-up ($r = .304$, $p = .009$).

3.5.2.2. Clinical trajectories of PTSD symptoms over Phase 2. Table 4 presents results from models predicting PTSD symptoms from Phase 2

Table 2
Models predicting substance use and heavy drinking trajectories.

Model and term	Model 1 Base model	Model 2 Attendance model	Model 3 PTSD diagnosis model
Probability of substance use			
Intercept	−2.833 (0.390)***	−2.232 (0.734)**	−2.094 (0.908)*
Treatment	−0.254 (0.551)	−0.337 (1.040)	0.305 (1.405)
Time	0.095 (0.014)***	0.453 (0.025)***	0.116 (0.029)***
Time^2	−0.003 (0.001)***	−0.02 (0.001)***	−0.001 (0.001)
Treatment × time	−0.032 (0.020)	−0.304 (0.035)***	−0.013 (0.043)
Treatment × time^2	0.002 (0.001)	0.005 (0.002)**	−0.004 (0.002)
Attendance	—	−0.082 (0.090)	—
Attendance × treatment	—	−0.011 (0.128)	—
Attendance × time	—	−0.052 (0.003)***	—
Attendance × time^2	—	0.003 (0.000)***	—
Attendance × treatment × time	—	0.044 (0.004)***	—
Attendance × treatment × time^2	—	−0.001 (0.000)***	—
PTSD diagnosis	—	—	−0.932 (1.022)
PTSD diagnosis × treatment	—	—	−0.630 (1.559)
PTSD diagnosis × time	—	—	−0.021 (0.033)
PTSD diagnosis × time^2	—	—	−0.002 (0.002)
PTSD × treatment × time	—	—	−0.023 (0.048)
PTSD × treatment × time^2	—	—	0.008 (0.003)**
Probability of Heavy Drinking			
Intercept	−4.687 (0.464)***	−4.373 (0.884)***	−5.975 (0.991)***
Treatment	0.056 (0.652)	0.491 (1.270)	2.869 (1.486)
Time	0.040 (0.018) *	0.384 (0.054)***	0.195 (0.039)***
Time^2	0.000 (0.001)	−0.014 (0.003)***	−0.005 (0.002)*
Treatment × time	0.027 (0.024)	−0.261 (0.095)**	−0.143 (0.052)**
Treatment × time^2	−0.004 (0.001)***	−0.002 (0.005)	0.000 (0.003)
Attendance	—	−0.023 (0.109)	—
Attendance × treatment	—	−0.103 (0.159)	—
Attendance × time	—	−0.055 (0.008)***	—
Attendance × time^2	—	0.002 (0.000)***	—
Attendance × treatment × time	—	0.052 (0.014)***	—
Attendance × treatment × time^2	—	−0.001 (0.001)	—
PTSD diagnosis	—	—	1.573 (1.106)
PTSD diagnosis × treatment	—	—	−3.405 (1.638)*
PTSD diagnosis × time	—	—	−0.184 (0.044)***
PTSD diagnosis × time^2	—	—	0.005 (0.002)*
PTSD × treatment × time	—	—	0.204 (0.059)***
PTSD × treatment × time^2	—	—	−0.005 (0.003)

Note. N = 123. Substance use refers to any alcohol or drug use. Heavy drinking refers to 5 or more drinks in a day. For treatment, CPT-M was coded as 1 and ICBT was coded as 0.
*p<.05, **p<.01, ***p<.001.

through the one year follow-up. There was no evidence to support our hypothesis that those receiving CPT-M would have greater reductions or better maintenance of PTSD symptom improvements than those receiving ICBT (see Table 4, Model 1), as trajectories of PTSD symptoms did not differ between treatment conditions after randomization. Further, we did not find strong evidence that either attendance (Table 4, Model 2) or PTSD diagnosis (Table 4, Model 3) influenced trajectories of PTSD symptoms. Although participants without a PTSD diagnosis

Table 3
Model predicted probability of substance Use and heavy drinking.

		Model Predicted Probability of Any Substance Use		
		At randomization	At end of Phase 2	At 12-month follow-up
ICBT	Low attendance	0.077 (0.038)	0.183(0.079)	0.28(0.107)
CPT-M	Low attendance	0.055 (0.27)	0.075(0.036)	0.03(0.016)
ICBT	High Attendance	0.041 (0.021)	0.03(0.015)	0.049(0.024)
CPT-M	High Attendance	0.026 (0.013)	0.036(0.019)	0.158(0.071)
ICBT	No PTSD	0.11 (0.081)	0.161 (0.112)	0.367 (0.193)
CPT-M	No PTSD	0.143 (0.118)	0.188 (0.146)	0.177 (0.140)
ICBT	PTSD	0.046 (0.019)	0.062 (0.025)	0.081 (0.032)
CPT-M	PTSD	0.034 (0.014)	0.042 (0.017)	0.079 (0.03)
		Model Predicted Probability of Heavy Drinking		
		At randomization	At end of Phase 2	At 12-month follow-up
ICBT	Low attendance	0.011(0.007)	0.023(0.014)	0.054(0.031)
CPT-M	Low attendance	0.013(0.008)	0.017(0.01)	0.004(0.002)
ICBT	High Attendance	0.009(0.005)	0.005(0.003)	0.004(0.003)
CPT-M	High Attendance	0.005(0.003)	0.007(0.004)	0.013(0.008)
ICBT	No PTSD	0.002(0.002)	0.005(0.005)	0.016(0.015)
CPT-M	No PTSD	0.031(0.043)	0.046(0.049)	0.030(0.033)
ICBT	PTSD	0.011(0.006)	0.012(0.006)	0.015(0.007)
CPT-M	PTSD	0.007(0.003)	0.008(0.004)	0.007(0.003)

Note. N = 123. Attendance was modeled as a continuous variable, with interactions probed by examining outcomes at low (3 or fewer individual sessions) and high (11 or more individual sessions) levels of attendance.

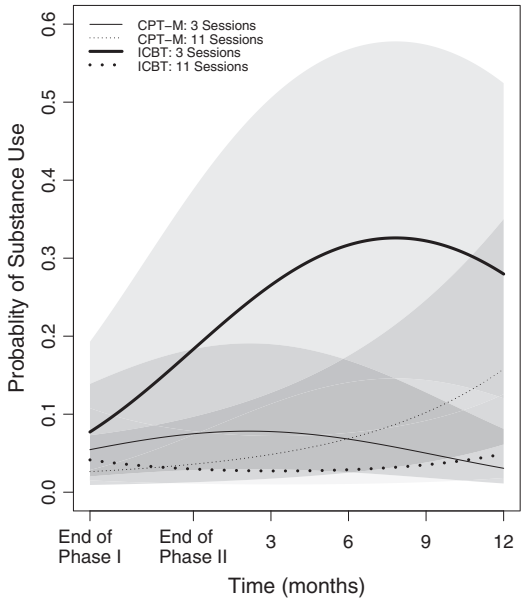

Fig. 2. Trajectories of substance use probability moderated by attendance.

scored on average 16.6 points lower on the PCL than did participants with a PTSD diagnosis prior to beginning Phase 2 treatment, we did not detect differences in the impacts of the treatments on patients with and without PTSD (see Fig. 4).

3.5.3. Depression symptoms

3.5.3.1. Summary statistics. The mean HDRS score in the CPT-M condition was 31.92 ($SD = 12.37$) at the end of Phase 1 treatment, 27.83 ($SD = 11.55$) at the end of CPT-M Phase 2 treatment, and 27.81 ($SD = 14.41$) at the one year follow-up. In the ICBT condition, the mean HDRS score was 28.85 ($SD = 12.25$) at the end of Phase 1 treatment, 26.14 ($SD = 12.58$) at the end of ICBT Phase 2 treatment, and 23.06 ($SD = 14.10$) at the one year follow-up.

The association between depression symptoms and PTSD diagnosis was significant prior to randomization (i.e., following Phase 1 ICBT; $r = .413$, $p < .001$) and following individual Phase 2 treatment ($r = .296$, $p = .003$), but was non-significant at the one year follow-up ($r = .121$, $p = .311$), indicating that participants with PTSD were indistinguishable from those without PTSD in terms of depression symptoms by the one year follow-up.

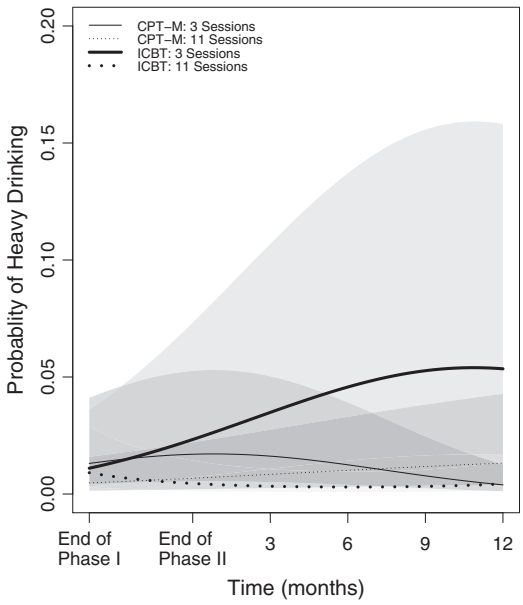

Fig. 3. Trajectories of heavy drinking probability moderated by attendance.

3.5.3.2. Clinical trajectories of depression symptoms over Phase 2. Table 4 presents results from models predicting depression symptoms from Phase 2 through the one year follow-up. Similar to the PTSD model, trajectories of depression symptoms did not significantly differ between treatment groups after randomization (i.e., over Phase 2 and the one year follow-up). Again, there was no evidence that CPT-M resulted in greater reductions or better maintenance of depression symptoms compared to ICBT (see Table 4, Model 1). Further, there was no strong evidence that attendance influenced trajectories of depression symptoms over time (see Table 4, Model 2). However, there was an interaction between PTSD diagnosis, treatment condition, and time (see Table 4, Model 3). This interaction is depicted in Fig. 5. For participants with PTSD, the ICBT and CPT-M conditions showed similar decreases in depression symptoms. For participants without PTSD, the ICBT condition also exhibited decreases in depression symptoms, but the CPT-M condition exhibited temporary increases in depression symptoms over the course of Phase 2 and the first 5 months of follow-up. However, the difference in depression symptoms between the ICBT and CPT-M participants without PTSD was negligible by the one year follow-up (see Fig. 5).

4. Discussion

Utilizing a sample of veterans with SUD, depression, and trauma history, the present study examined the utility of providing follow-up trauma-focused therapy (Phase 2 CPT-M) after initially providing group-based cognitive-behavioral therapy for SUD and depression (i.e., Phase 1 ICBT). Our study design was unique in that participants engaged in 12 weeks of twice-weekly group ICBT in Phase 1 prior to being randomized to receive either individual CPT-M or individual ICBT in Phase 2. Our primary hypothesis was that receiving Phase 2 CPT-M would result in better treatment outcomes over time (substance use, depression symptoms, and PTSD symptoms) compared to receiving Phase 2 ICBT.

Results indicated that there were slight improvements in PTSD and depression symptoms at the end of Phase 1 group ICBT, and that there were additional small improvements after Phase 2 individual treatment (except for depression for individuals in the CPT-M condition without PTSD). These small treatment gains in PTSD and depression were maintained one year later. There were more substantial improvements in substance use after Phase 1 ICBT, with improvements largely maintained over the course of Phase 2 and the one year follow-up. However, inconsistent with our hypotheses, we did not find evidence that engaging in trauma-focused treatment (Phase 2 CPT-M) resulted in greater reductions of PTSD or depression symptoms, or better maintenance of treatment gains, compared to treatment that is not trauma focused (Phase 2 ICBT). With respect to substance use, those receiving Phase 2 CPT-M had less heavy drinking (5 or more drinks on a given day) over time than those assigned to Phase 2 ICBT, but treatment conditions did not differ when predicting overall substance use (i.e., the probability of any alcohol or drug use on a given day). Notably, participants with lower attendance in the ICBT condition had the worst substance use and heavy drinking outcomes over time. Findings are consistent with a study providing an integrated cognitive-behavioral therapy for PTSD-SUD (McGovern, Lambert-Harris, Alterman, Xie, & Meier, 2011), which showed decreases in PTSD symptoms and drug use relative to addiction counseling/standard care and, although not the focus of treatment, depression symptoms decreased over time but did not differ across treatment conditions.

Participants with a PTSD diagnosis had higher PTSD symptoms throughout treatment and the one year follow-up than did participants without a PTSD diagnosis, but PTSD diagnosis did not influence

Table 4
Models predicting PTSD symptoms and depression symptom trajectories.

Model and term	Model 1 Base Model	Model 2 Attendance Model	Model 3 PTSD diagnosis Model
PTSD Symptoms (PCL)			
Intercept	49.349 (2.050)***	46.258 (3.900)***	36.001 (4.206)***
Treatment	2.270 (2.905)	4.368 (5.525)	0.659 (6.494)
Time	−0.358 (0.343)	0.392 (0.689)	−0.823 (0.696)
Time^2	0.001 (0.021)	0.001 (0.042)	0.042 (0.042)
Treatment × time	−0.677 (0.489)	−1.151 (1.019)	0.862 (1.194)
Treatment × time^2	0.051 (0.030)	0.052 (0.062)	−0.074 (0.076)
Attendance	—	0.406 (0.474)	—
Attendance × treatment	—	−0.269 (0.681)	—
Attendance × time	—	−0.094 (0.081)	—
Attendance × time^2	—	0.000 (0.005)	—
Attendance × treatment × time	—	0.057 (0.119)	—
Attendance × treatment × time^2	—	0.000 (0.007)	—
PTSD diagnosis	—	—	16.637 (4.707)***
PTSD diagnosis × treatment	—	—	0.880 (7.13)
PTSD diagnosis × time	—	—	0.660 (0.799)
PTSD diagnosis × time^2	—	—	−0.056 (0.049)
PTSD × treatment × time	—	—	−1.853 (1.312)
PTSD × treatment × time^2	—	—	0.150 (0.083)
Depression symptoms (HDRS)			
Intercept	29.72 (1.757)**	30.465 (3.543)***	22.743 (3.702)***
Treatment	3.320 (2.482)	5.228 (4.908)	−5.145 (5.785)
Time	−0.686 (0.318)*	−0.506 (0.665)	−1.233 (0.648)
Time^2	0.031 (0.019)	0.039 (0.040)	0.064 (0.039)
Treatment × time	−0.112 (0.456)	−0.474 (0.98)	2.841 (1.121)*
Treatment × time^2	−0.006 (0.028)	0.004 (0.059)	−0.162 (0.071)*
Attendance	—	−0.118 (0.421)	—
Attendance × treatment	—	−0.271 (0.594)	—
Attendance × time	—	−0.021 (0.078)	—
Attendance × time^2	—	−0.001 (0.005)	—
Attendance × treatment × time	—	0.047 (0.114)	—
Attendance × treatment × time^2	—	−0.001 (0.007)	—
PTSD diagnosis	—	—	8.771 (4.155)*
PTSD diagnosis × treatment	—	—	9.165 (6.349)
PTSD diagnosis × time	—	—	0.745 (0.742)
PTSD diagnosis × time^2	—	—	−0.045 (0.045)
PTSD × treatment × time	—	—	−3.514 (1.228)**
PTSD × treatment × time^2	—	—	0.187 (0.077)*

Note. N = 123.
*p<.05, **p<.01, ***p<.001. For treatment, CPT-M was coded as 1 and ICBT was coded as 0.

trajectories of PTSD symptoms over time. That is, both participants with and without PTSD exhibited similar declines in PTSD symptoms and maintained Phase 1 treatment gains during treatment and the one year follow-up. With respect to depression symptoms, participants with PTSD also reported more depression than did those without PTSD, but depression trajectories did not significantly differ across

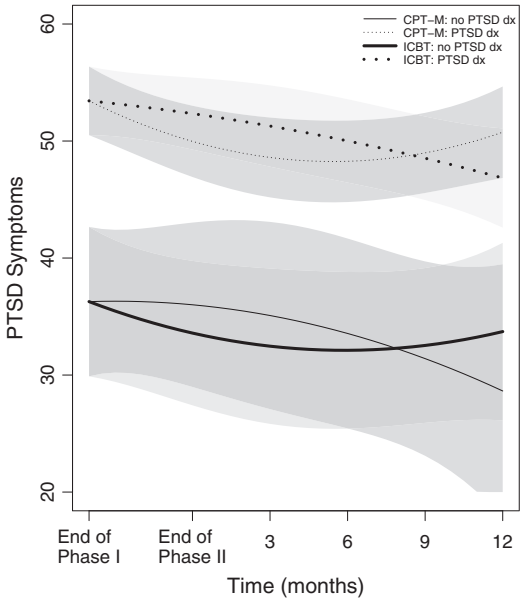

Fig. 4. Trajectories of PTSD symptoms moderated by PTSD diagnosis.

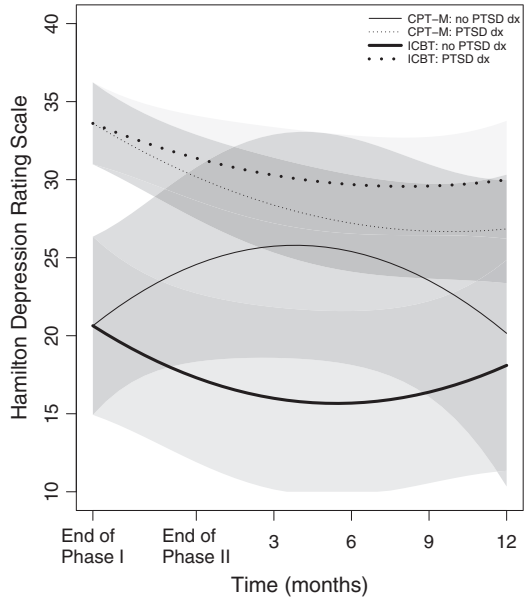

Fig. 5. Trajectories of depression symptoms moderated by PTSD diagnosis.

treatment conditions. There was, however, some evidence that depression symptom trajectories differed across treatment conditions for participants without PTSD, such that individuals in the CPT-M condition without PTSD had increases in depression symptoms during treatment. Although this increased depression was negligible by the one year follow-up, it appears that CPT-M was not helpful for individuals without PTSD. Thus, our theory that PTSD treatment would be helpful even for those with subthreshold PTSD was not supported.

Results of models examining moderation by PTSD diagnosis when predicting substance use and heavy drinking were more complex. Both models showed significant treatment by PTSD diagnosis by time interactions, which appeared to be driven by differences between treatment conditions for participants without PTSD. Among participants without PTSD, those in the ICBT condition had the worst/least preferable substance use trajectories, but those in the CPT-M condition without PTSD had the worst/least preferable heavy drinking trajectories (thus, contrasting with our theory that CPT-M would be helpful for those without PTSD). Our results also unexpectedly indicated that substance use outcomes were better for those with PTSD as compared to those without PTSD. However, confidence in these findings is limited, since only 18% of the sample (12 ICBT and 10 CPT-M participants) did not meet criteria for current PTSD.

Findings from this study extend those of Lydecker et al. (2010) by demonstrating the efficacy of ICBT within a trauma-exposed SUD sample. Findings also extend research by Norman et al. (2010), who found that individuals with SUD, depression, and PTSD exhibited declines in depression following group ICBT or 12-Step Facilitation interventions but remained elevated on depression relative to those without PTSD. Norman and colleagues also found that substance use treatment gains were not maintained over time for those with a PTSD diagnosis (note that PTSD symptom severity was not assessed in that study). In light of these findings, we examined whether PTSD-specific treatment would allow individuals with PTSD to maintain gains in substance use outcomes and to continue to show depression symptom improvements. We found that individuals both with and without PTSD maintained the small improvements in depression at the one year follow-up, and that individuals with PTSD actually had better substance use outcomes than did those without PTSD. As such, it appears that providing additional individually-delivered treatment following group ICBT may have added benefits for depression and substance use.

Overall, results suggest that trauma-exposed participants both with and without PTSD benefit similarly from ICBT and CPT-M treatment in terms of both PTSD and depression symptoms. Results also suggest that there may be a slight advantage for CPT-M over ICBT with respect to heavy drinking outcomes for individuals with PTSD. Therefore, the only outcome in which CPT-M was superior to ICBT was heavy drinking for those with PTSD. It may be that CPT-M has the greatest advantage for those with more severe co-occurring problems, whereas ICBT may be sufficient for those with less severe comorbidities. This study advances current research by supporting the efficacy of both CPT and ICBT for trauma-exposed individuals with comorbid SUD and depression. Most studies on integrated PTSD/SUD treatments that address past traumatic events within session have focused on exposure-based (see Roberts et al., 2015) rather than cognitive-based interventions (note that present-focused PTSD/SUD treatments also exist, namely Seeking Safety). We know of only one non-randomized study (Kaysen et al., 2014) and one case report (McCarthy & Petrakis, 2011) that specifically examined CPT for comorbid PTSD/SUD; both had promising results. We selected CPT-M rather than exposure-based PTSD treatment for the present study, in part, because challenging maladaptive thoughts can be helpful for all three disorders (PTSD, SUD, and depression), and thus skills learned to manage trauma symptoms translate well to depression and SUD. However, the fact that ICBT and CPT-M are both primarily cognitive interventions may have resulted in limited differences between treatment conditions. Future studies comparing exposure-based interventions to cognitive-based interventions could reveal a different pattern of findings.

Despite the common cognitive component of CPT-M and ICBT, there are two key differences. First, ICBT includes a substantial behavioral component (increasing healthy activities, building social network), with only one-third of the intervention focused on cognitive skills. Second, CPT-M is specifically trauma-focused and encourages challenging trauma-related thoughts. In ICBT, focus is on thoughts associated with negative mood with all examples related to depression or substance use.

It is possible that our approach of providing participants with group (Phase 1) ICBT treatment prior to randomization into Phase 2 ICBT or CPT-M may have limited differences between treatment conditions in Phase 2. Further, improvements during and after Phase 2 could be due to the continuing effects of the Phase 1 intervention rather than the effects of the Phase 2 interventions. Our two-phased approach was intended to allow participants to experience improvements in depression, establish abstinence from substance use, and increase coping ability prior to addressing trauma-related symptoms. However, the fact that participants had already experienced some improvements in symptoms prior to randomization may have made it difficult to detect post-randomization differences. A future study that immediately randomizes to CPT-M or ICBT may reveal more differences between treatment conditions.

Other study limitations should be noted. First, our sample was comprised of mostly male veterans; results may not generalize to female and nonveteran samples. Second, the majority (~82%) of our sample met current criteria for PTSD. Future studies with larger samples and more even distributions of participants with and without PTSD would increase confidence in conclusions about symptom trajectories for trauma-exposed individuals who do not meet criteria for PTSD. Third, our study used a self-report measure of PTSD symptoms; an interview-based measure could be more reliable. Fourth, it should be noted that PTSD and depression have many overlapping symptoms, which makes interpretation of results difficult. Finally, some symptom improvements may be attributable to prescribed antidepressant and addiction medications, although this seems unlikely given that medication usage did not differ across treatment conditions. These limitations are countered by several strengths, including one year of follow-up, large sample size, randomization into two active treatment conditions, and utilization of treatments that are easily disseminated and learned by clinicians.

The current study has important clinical implications for how to best treat complex patients experiencing co-occurring SUD, depression, and PTSD. Both in community (see McCauley et al., 2012) and VA settings, dual-diagnosis SUD treatment is typically provided in group formats. Although the group format is a cost-effective means of addressing SUD and co-occurring symptoms, not everyone benefits from group treatment or maintains gains over time (as seen in Norman et al., 2010, for those participants with comorbid PTSD in addition to SUD and depression). Although our study did not specifically compare a two-phased approach to a single group phase approach, the fact that improvements (which were substantial for substance use but small for PTSD and depression) were generally maintained one year after individual treatment suggests utility of providing additional individual treatment for complex individuals with multiple diagnoses. We recommend clinicians discuss the advantages and disadvantages of engaging in further individual treatment upon finishing group treatment. Given our finding that treatment gains were largely maintained over time in both CPT-M and ICBT conditions, ICBT may be a viable alternative to trauma-focused treatment for individuals who are not willing to engage in trauma-focused treatment (e.g., CPT or Prolonged Exposure). Further, ICBT may be a useful intervention for treatment programs lacking providers trained in the delivery of PTSD-specific treatments. Finally, given that specific trauma-related thoughts are not discussed within ICBT, it may be helpful for treating a wide range of trauma types.

Acknowledgements

This research was supported by a Department of Veterans Affairs, Veterans Health Administration, Office of Research and Development, Clinical Sciences Research & Development Merit Review Award to

Susan R. Tate. We also appreciate the support of the VA Office of Academic Affiliations Interprofessional Advanced Fellowship in Addictions Treatment. These entities had no role in the study design, data collection, writing, or submission of this paper for publication. No actual or potential conflicts of interest exist for any authors on this paper.

References

Blake, D. D., Weathers, F. W., Nagy, L. M., Kaloupek, D. G., Gusman, F. D., Charney, D. S., & Keane, T. M. (1995). The development of a clinician-administered PTSD scale. *Journal of Traumatic Stress*, *8*, 75–90. http://dx.doi.org/10.1002/jts.2490080106.

Bonin, M. F., Norton, G. R., Asmundson, G. J., Dicurzio, S., & Pidlubney, S. (2000). Drinking away the hurt: the nature and prevalence of PTSD in substance abuse patients attending a community-based treatment program. *Journal of Behavior Therapy and Experimental Psychiatry*, *31*, 55–66. http://dx.doi.org/10.1016/S0005-7916(00)00008-2.

Brady, K. T., Killeen, T. K., Brewerton, T., & Lucerini, S. (2000). Comorbidity of psychiatric disorders and posttraumatic stress disorder. *Journal of Clinical Psychiatry*, *61*, 22–32.

Bright, J. W., & Dawkins, G. S. (1965). Communication: Some aspects of curve fitting using orthogonal polynomials. *Industrial & Engineering Chemistry Fundamentals*, *4*, 93–97. http://dx.doi.org/10.1021/i160013a017.

Carey, K., Carey, M. P., Maisto, S., & Henson, J. (2004). Temporal stability of the timeline followback interview for alcohol and drug use with psychiatric outpatients*65*, 774–781. http://dx.doi.org/10.15288/jsa.2004.65.774.

Carlier, I. V., Voerman, B. E., & Gersons, B. P. (2000). Intrusive traumatic recollections and comorbid posttraumatic stress disorder in depressed patients. *Psychosomatic Medicine*, *62*, 26–32. http://dx.doi.org/10.1097/00006842-200001000-00004.

Currie, S. R., Patten, S. B., Williams, J. V. A., Wang, J. L., Beck, C. A., El-Guebaly, N., & Maxwell, C. (2005). Comorbidity of major depression with substance use disorders. *The Canadian Journal of Psychiatry / La Revue Canadienne De Psychiatrie*, *50*, 660–666.

Darves-Bornoz, J. M., Alonso, J., de Girolamo, G., Graaf, R. D., Haro, J. M., Kovess-Masfety, V., ... Gasquet, I. (2008). Main traumatic events in Europe: PTSD in the European study of the epidemiology of mental disorders survey. *Journal of Traumatic Stress*, *21*, 455–462. http://dx.doi.org/10.1002/jts.20357.

Driessen, M., Schulte, S., Luedecke, C., Schaefer, I., Sutmann, F., Ohlmeier, M., ... Havemann-Reinicke, U. (2008). Trauma and PTSD in patients with alcohol, drug, or dual dependence: A multi-center study. *Alcoholism: Clinical and Experimental Research*, *32*, 481–488. http://dx.doi.org/10.1111/j.1530-0277.2007.00591.x.

Felker, B. L., Kirchner, J. E., Chan, D., & Rubenstein, L. V. (2007). Prevalence of depression–PTSD comorbidity: Implications for clinical practice guidelines and primary care-based interventions. *Journal of General Internal Medicine*, *22*, 711–718. http://dx.doi.org/10.1007/s11606-006-0101-4.

Foa, E. B., Keane, T. M., Friedman, M. J., & Cohen, J. A. (2009). *Effective treatments for PTSD: Practice guidelines from the International Society for Traumatic Stress Studies* (2nd ed.). New York, NY: Guilford Press.

Ford, J. D., Russo, E. M., & Mallon, S. D. (2007). Integrating treatment of posttraumatic stress disorder and substance use disorder. *Journal of Counseling & Development*, *85*, 475–489. http://dx.doi.org/10.1002/j.1556-6678.2007.tb00616.x.

Frees, E. W. (2004). *Longitudinal and panel data: Analysis and applications in the social sciences.* New York: Cambridge University Press. http://dx.doi.org/10.1017/CBO9780511790928.

Grant, B. F., Stinson, F. S., Dawson, D. A., Chou, S. P., Dufour, M. C., Compton, W., ... Kaplan, K. (2004). Prevalence and co-occurrence of substance use disorders and independent mood and anxiety disorders: results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Archives of General Psychiatry*, *61*, 807. http://dx.doi.org/10.1001/archpsyc.61.8.807.

Green, B. L., Krupnick, J. L., Chung, J., Siddique, J., Krause, E. D., Revicki, D., ... Miranda, J. (2006). Impact of PTSD comorbidity on one-year outcomes in a depression trial. *Journal of Clinical Psychology*, *62*, 815–835. http://dx.doi.org/10.1002/jclp.20279.

Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery & Psychiatry*, *23*, 56–62. http://dx.doi.org/10.1136/jnnp.23.1.56.

Harrell, F. E. (2001). *Regression Modeling Strategies: With Application to Linear Models, Logistic Regression, and Survival Analysis.* New York: Springer-Verlag. http://dx.doi.org/10.1007/978-1-4757-3462-1.

Hegel, M. T., Unützer, J., Tang, L., Areán, P. A., Katon, W., Noël, P. H., ... Lin, E. H. (2005). Impact of comorbid panic and posttraumatic stress disorder on outcomes of collaborative care for late-life depression in primary care. *American Journal of Geriatric Psychiatry*, *13*, 48–58. http://dx.doi.org/10.1097/00019442-200501000-00008.

Hemmy Asamsama, O., Dickstein, B. D., & Chard, K. M. (2015). Do scores on the Beck Depression Inventory-II predict outcome in Cognitive Processing Therapy? *Psychological Trauma: Theory, Research, Practice, and Policy*. http://dx.doi.org/10.1037/tra0000034 (Advance online publication).

Holtzheimer, Paul E., III, Russo, J., Zatzick, D., Bundy, C., & Roy-Byrne, P. (2005). The impact of comorbid posttraumatic stress disorder on short-term clinical outcome in hospitalized patients with depression. *The American Journal of Psychiatry*, *162*, 970–976. http://dx.doi.org/10.1176/appi.ajp.162.5.970.

Kadden, K., Carroll, K., Donovan, D., Cooney, N., Monti, P., Abrams, D., LItt, M., & Hester, R. (1992). *Cognitive-Behavioral Coping Skills Therapy Manual.* Project MATCH, NIAAA, NIH, 94–3724.

Kay-Lambkin, F. J., Baker, A. L., Lewin, T. J., & Carr, V. J. (2009). Computer-based psychological treatment for comorbid depression and problematic alcohol and/or cannabis use: a randomized controlled trial of clinical efficacy. *Addiction*, *104*, 378–388. http://dx.doi.org/10.1111/j.1360-0443.2008.02444.x.

Kaysen, D., Schumm, J., Pedersen, E. R., Seim, R. W., Bedard-Gilligan, M., & Chard, K. (2014). Cognitive Processing Therapy for veterans with comorbid PTSD and alcohol use disorders. *Addictive Behaviors*, *39*, 420–427. http://dx.doi.org/10.1016/j.addbeh.2013.08.016.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K. R., ... Wang, P. S. (2003). The epidemiology of major depressive disorder: Results from the national comorbidity survey replication (NCS-R). *JAMA: Journal of the American Medical Association*, *289*, 3095–3105. http://dx.doi.org/10.1001/jama.289.23.3095.

Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*, 617–627. http://dx.doi.org/10.1001/archpsyc.62.6.617.

Lydecker, K. P., Tate, S. R., Cummins, K. M., McQuaid, J., Granholm, E., & Brown, S. A. (2010). Clinical outcomes of an integrated treatment for depression and substance use disorders. *Psychology of Addictive Behaviors*, *24*, 453–465. http://dx.doi.org/10.1037/a0019943.

McCarthy, E., & Petrakis, I. (2011). Case report on the use of cognitive processing therapy–cognitive, enhanced to address heavy alcohol use. *Journal of Traumatic Stress*, *24*, 474–478. http://dx.doi.org/10.1002/jts.20660.

McCauley, J. L., Killeen, T., Gros, D. F., Brady, K. T., & Back, S. E. (2012). Posttraumatic stress disorder and co-occurring substance use disorders: Advances in assessment and treatment. *Clinical Psychology: Science and Practice*, *19*, 283–304. http://dx.doi.org/10.1111/cpsp.12006.

McGovern, M. P., Lambert-Harris, C., Alterman, A. I., Xie, H., & Meier, A. (2011). A randomized controlled trial comparing integrated cognitive behavioral therapy versus individual addiction counseling for co-occurring substance use and posttraumatic stress disorders. *Journal of Dual Diagnosis*, *7*, 207–227. http://dx.doi.org/10.1080/15504263.2011.620425.

Monson, C. M., Schnurr, P. P., Resick, P. A., Friedman, M. J., Young-Xu, Y., & Stevens, S. P. (2006). Cognitive processing therapy for veterans with military-related posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, *74*, 898–907. http://dx.doi.org/10.1037/0022-006X.74.5.898.

Muñoz, R. F., & Miranda, J. (1996). *Group therapy manual for cognitive-behavioral treatment of depression.* Santa Monica, CA: RAND.

Narula, S. U. (1978). Orthogonal polynomial regression for unequal spacing and frequencies. *Journal of Quality Technology*, *9*, 170–179.

National Center for PTSD (2014). Using the PTSD Checklist for DSM-IV (PCL). Retrieved from www.ptsd.va.gov/professional/pages/assessments/assessment-pdf/PCL-handout.pdf

National Institute for Clinical Excellence (2004). *Depression: Management of depression in primary and secondary care.* Clinical Practice Guideline No 23. London: National Institute for Clinical Excellence, 1–670.

Norman, S. B., Stein, M. B., & Davidson, J. R. (2007). Profiling posttraumatic functional impairment. *The Journal of Nervous and Mental Disease*, *195*, 48–53. http://dx.doi.org/10.1097/01.nmd.0000252135.25114.02.

Norman, S. B., Tate, S. R., Wilkins, K. C., Cummins, K., & Brown, S. A. (2010). Posttraumatic stress disorder's role in integrated substance dependence and depression treatment outcomes. *Journal of Substance Abuse Treatment*, *38*, 346–355. http://dx.doi.org/10.1016/j.jsat.2010.01.013.

Ouimette, P. C., Brown, P. J., & Najavits, L. M. (1998). Course and treatment of patients with both substance use and posttraumatic stress disorder. *Addictive Behaviors*, *23*, 785–795. http://dx.doi.org/10.1016/S0306-4603(98)00064-1.

Resick, P. A., Monson, C. M., & Chard, K. M. (2008). *Cognitive processing therapy: Veteran/military version.* Washington DC: Department of Veterans' Affairs.

Resick, P. A., & Schnicke, M. K. (1992). Cognitive processing therapy for sexual assault victims. *Journal of Consulting and Clinical Psychology*, *60*, 748–756. http://dx.doi.org/10.1037/0022-006X.60.5.748.

Roberts, N. P., Roberts, P. A., Jones, N., & Bisson, J. I. (2015). Psychological interventions for post-traumatic stress disorder and comorbid substance use disorder: A systematic review and meta-analysis. *Clinical Psychology Review*, *38*, 25–38. http://dx.doi.org/10.1016/j.cpr.2015.02.007.

Robins, L. N., Wing, J., Wittchen, H. U., Helzer, J. E., Babor, T. F., Burke, J., ... Towle, L. H. (1988). The Composite International Diagnostic Interview: An epidemiologic instrument suitable for use in conjunction with different diagnostic systems and in different cultures. *Archives of General Psychiatry*, *45*, 1069–1077. http://dx.doi.org/10.1001/archpsyc.1988.01800360017003.

Singer, J., & Willett, J. (2003). *Applied longitudinal data analysis: Modelingchange and event occurrence.* New York: Oxford University Press. http://dx.doi.org/10.1093/acprof:oso/9780195152968.001.0001.

Sobell, L. C., & Sobell, M. B. (1992). Timeline Follow-Back: A technique for assessing self-reported alcohol consumption. In R. L. Litten, & J. P. Allen (Eds.), *Measuring Alcohol Consumptions: Psychosocial and Biochemical Methods*. Totowa, New Jersey: Human Press. http://dx.doi.org/10.1007/978-1-4612-0357-5_3.

Souza, T., & Spates, C. R. (2008). Treatment of PTSD and substance abuse comorbidity. *Behavior Analyst Today*, *9*, 11–26. http://dx.doi.org/10.1037/h0100643.

Stewart, S., Conrod, P. J., Samoluk, S. B., Pihl, R. O., & Dongier, M. (2000). Posttraumatic stress disorder symptoms and situation-specific drinking in women substance abusers. *Alcoholism Treatment Quarterly*, *18*, 31–47. http://dx.doi.org/10.1300/J020v18n03_04.

van Dam, D., Vedel, E., Ehring, T., & Emmelkamp, P. M. (2012). Psychological treatments for concurrent posttraumatic stress disorder and substance use disorder: A systematic review. *Clinical Psychology Review*, *32*, 202–214. http://dx.doi.org/10.1016/j.cpr.2012.01.004.

Weathers, F. W., Keane, T. M., & Davidson, J. R. T. (2001). Clinician-administered PTSD Scale: A review of the first ten years of research. *Depression and Anxiety*, *13*, 132–156. http://dx.doi.org/10.1002/da.1029.

Weathers, F. W., Litz, B. T., Huska, J. A., & Keane, T. M. (1994). *PTSD checklist.* Boston: National Center for PTSD.

Weis, M. (2010). Integrated and holistic treatment approach to PTSD and SUD: A synergy. *Journal of Addictions & Offender Counseling*, *31*, 25–37. http://dx.doi.org/10.1002/j.2161-1874.2010.tb00064.x.

West, B. T., Welch, K. B., & Galecki, A. T. (2007). *Linear mixed models: A practical guide to using statistical software.* Boca Raton, FL: Chapman & Hall.

Willenbring, M. L. (1986). Measurement of depression in alcoholics. *Journal of Studies on Alcohol*, *47*, 367–372. http://dx.doi.org/10.15288/jsa.1986.47.367.