The Comorbidity of Post-Traumatic-Stress Disorder (PTSD) and Substance Use Disorders

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Abstract

Posttraumatic stress disorder (PTSD) and substance use disorders (SUDs) frequently co-occur. Among individuals seeking treatment for SUDs, approximately 30 % to 50 % meet criteria for lifetime PTSD. Epidemiologic surveys demonstrate that individuals with PTSD have 4-5 times more likely to have a SUD at some point in their lives compared to individuals who do not have PTSD. Self-medication and susceptibility are two hypotheses that have been proposed to help explain the etiological relationship between PTSD and SUDs. It is also possible that common factors, such as genetic, neurobiological, or environmental factors, contribute to the high rate of PTSD-SUD co-occurrence. Integrated psychotherapy approaches for the treatment of patients with both disorders show promise. There are also a number of pharmacotherapeutic agents that have demonstrated preliminary efficacy in the treatment of co-occurring PTSD/SUD, but further investigation is needed. This chapter reviews these and other advances in the study of comorbid PTSD and SUDs, and suggests areas for future work.

120.1 Introduction

Posttraumatic stress disorder (PTSD) is a psychiatric disorder that occurs after exposure to an event (experienced or witnessed) involving actual or threatened death, serious injury, or threat to the physical integrity of oneself or others. The traumatic event must be followed by at least 1 month of symptoms, such as intrusive recollection, avoidance or emotional numbing, and hyperarousal, that interfere with the individual's ability to function. Substance use disorders (SUDs) commonly co-occur with PTSD. Moreover, comorbid PTSD/SUD is associated with a more complex and costly clinical course when compared with either disorder alone, so identification and treatment of both illnesses in individuals with comorbidity is essential to optimize clinical care.

Discussions of international issues in co-occurring PTSD and substance use are complicated by a number of factors. Definitions and experiences of trauma are culturally bound and in many countries can be connected with issues of politics and social justice. There is also debate about the cross-cultural application of the DSM-defined PTSD criteria. Modifications of the DSM criteria and textual modification have been suggested to improve cross-cultural applicability (Hinton and Lewis-Fernandez 2011). Similarly, patterns of substance use and definitions of SUDs also occur in cultural contexts that can tremendously alter the perspectives of acceptable use and willingness to honestly report use. In addition, we do not have accurate estimates of prevalence of either SUDs or PTSD alone or the comorbidity in many areas of the world. So, in the sections that follow, much of the data that is presented is based on studies conducted in a few countries. However, the diagnostic, phenomenologic, and neurobiologic underpinnings of the relationships and treatment options for PTSD and SUDs discussed are likely to apply broadly.

120.2 Epidemiology of PTSD/SUD Comorbidity

120.2.1 United States

Prevalence estimates for PTSD, SUD, and comorbid PTSD/SUD among US adults are primarily garnered from three sources: national epidemiological surveys and Veteran and treatment-seeking populations. Early estimates were provided by the National Comorbidity Survey (NCS; $N = 8{,}098$), conducted from 1990 to 1992, indicated a 7.8 % lifetime prevalence for PTSD and a 26.6 % lifetime prevalence for SUD among the general population (aged 15–54), (Kessler et al. 1994, 1995). Individuals with PTSD were between two and four times more likely to meet criteria for an SUD than those without PTSD. A decade later, the National Comorbidity Survey – Replication (NCS-R; $N = 5{,}692$) indicated a 6.8 % lifetime prevalence of PTSD and 35.3 % lifetime prevalence of any SUD (Kessler et al. 2005; Harvard School of Medicine 2007). More recently, the 2010 National Epidemiologic Survey on Alcohol and Related Conditions (NESARC; $N = 34{,}653$) estimated that 6.4 % of the population met lifetime criteria for PTSD, more than one in five (22.3 %) of those with PTSD met criteria for drug abuse or dependence, and nearly half (46.4 %) met criteria for any SUD (Pietrzak et al. 2011).

Veterans constitute a population of particular interest due to their increased risk for developing both PTSD and SUDs in comparison to the general population (Kang et al. 2003; Hoge et al. 2004; Sabella 2012). Post-deployment prevalence rates have been estimated at approximately 21 % for SUDs and between 15 % and 20 % for PTSD among Veterans of Operation Enduring Freedom/Operation Iraqi Freedom (OEF/OIF) (Hoge et al. 2004; Bray and Hourani 2007; Seal et al. 2007; Thomas et al. 2010). Severity of combat exposure has been directly linked to risk for development and chronicity of PTSD symptoms (Kang et al. 2003) and misuse of substances (Santiago et al. 2010). Administrative data from the Department of Veterans Affairs indicate that among Veterans serving in the Vietnam era or later, almost half (41.4 %) with an SUD were also diagnosed with PTSD (Petrakis et al. 2011). Conversely, a recent study followed a large cohort of Veterans diagnosed with PTSD ($n = 272{,}509$) over a 3-year timeframe and found that nearly one in five (19.33 %) were diagnosed with a comorbid SUD and presence of a comorbid SUD was positively associated with mortality at follow-up (hazards ratio = 1.70; Bohnert et al. 2013).

Treatment-seeking individuals have rates of comorbid PTSD and SUD that are consistently higher than the general population. Patients seeking treatment for PTSD are up to 14 times more likely than patients without PTSD to have an SUD (Jacobsen et al. 2001; Chilcoat and Menard 2003; Ford et al. 2007). Conversely, among patients seeking treatment for SUDs, lifetime PTSD rates range from approximately 30 % to over 60 % (Dansky et al. 1994; Triffleman et al. 1995; Back et al. 2000; Clark et al. 2001; Brady et al. 2004). Finally, a multimodal assessment of comorbidity among patients at a level I trauma surgery center indicated that 79 % had one or more SUD and/or PTSD comorbidity; three in

four (74 %) met criteria for an SUD, whereas one in four patients endorsed symptoms consistent with PTSD (Zatzick et al. 2012). Variations in prevalence estimates among treatment-seeking populations are most likely attributable to methodological differences, including differences in patient populations sampled and measurement techniques.

120.2.2 International Prevalence Estimates

As mentioned above, we do not have accurate prevalence estimates of the rates of either PTSD or SUDs in much of the world. While the World Health Organization (WHO) has sponsored a series of international studies of mental health disorders, the complexity of the trauma questions used, cultural differences in the definition of trauma, and reluctance to discuss traumatic events likely led to underestimates of PTSD prevalence (Kessler and Greenberg 2002). In general, the estimates for lifetime PTSD prevalence range from a low of 0.3 % in China to 6.1 % in New Zealand (Demyttenaere et al. 2004). As might be expected, some evidence suggests that the risk of PTSD is higher among people from less developed countries who have been exposed to prolonged traumatic experiences associated with wars and political and ethnic violence. For example, 65 % of Bosnian refugees resettled in the United States have PTSD (Weine et al. 1995) and 73 % of Palestinian children exposed to war trauma experienced PTSD (Thabet and Vostanis 1999). In a recent WHO survey, disability from common mental and physical disorders was assessed in nationally representative samples from 26 countries (Bruffaerts et al. 2012). While physical disorders were considerably more common than mental disorders, there was more disability associated with mental disorders as compared to physical disorders at an individual level. Of all physical and mental disorders, PTSD was associated with the highest level of disability.

While little is known about the prevalence of PTSD in many countries, even less is known about the prevalence of co-occurring PTSD and SUDs. An epidemiologic survey conducted in Australia in 2007 found that the 12-month prevalence of PTSD was 6.4 %, higher than the prevalence of any other anxiety disorder (National Survey of Mental Health and Wellbeing 2007). SUDs were much more common in individuals with mental illness as compared to those without mental illness, and nearly 40 % of those with anxiety disorders reported daily drug misuse. Data for PTSD specifically was not presented. This suggests that the common co-occurrence of PTSD and SUDs which has been found in the United States may exist in other countries. However, in an investigation of the prevalence of psychiatric disorders in South Africa (Stein et al. 2008), the authors note that local factors such as poverty and lack of access to substances may change the occurrence of certain psychiatric disorders and comorbidities.

120.3 Etiologic Relationship Between PTSD and SUD

120.3.1 Self-Medication Hypothesis

A number of theories have been posited to explain the etiology and functional associations between PTSD and co-occurring SUDs. The most prominent theory is the *self-medication hypothesis* (Khantzian 1985, 1990, 1997; Reed et al. 2007; Menary et al. 2011). According to the self-medication theory, substance use is negatively reinforced when it alleviates PTSD symptoms, such as sleep impairment, intrusive memories, nightmares, hyperarousal, and feelings of estrangement. In support of this theory, Saladin and colleagues (1995) compared individuals with PTSD only vs. PTSD/SUD and found that hyperarousal and avoidance symptoms were more severe among the comorbid PTSD/SUD group. Laboratory-based findings also provide support for the self-medication model. One study examined responsivity to trauma cues (i.e., presentation of personalized trauma narrative) and found that individuals with comorbid PTSD/SUD demonstrate increased craving for substances in response to the trauma cues (Coffey et al. 2002). Moreover, research has shown that trauma cue-elicited craving is significantly reduced following exposure therapy for PTSD (Coffey et al. 2006). Finally, increases in craving have been shown to be positively correlated with severity of PTSD symptoms (Saladin et al. 2003).

Among patients with PTSD/SUDs, the drug of choice (e.g., central nervous system depressant or stimulant) may reflect an attempt to alleviate a particular cluster of symptoms. For example, Saladin et al. (1995) found that PTSD/SUD individuals with more severe hyperarousal symptoms (Criterion D) were more likely to be dependent on alcohol than cocaine. Likewise, PTSD/SUD individuals with more severe avoidance (Criterion C) and flashback symptoms (Criterion B) were more likely to be dependent on cocaine. More recently, Tull and colleagues (2010) observed a significant relationship among PTSD hyperarousal symptoms and dependence on heroin, as opposed to crack/cocaine and alcohol dependence. In addition to self-medication of PTSD symptoms, individuals with PTSD/SUDs may also use substances to self-medication withdrawal symptoms, which may mimic symptoms of PTSD. For example, withdrawal from alcohol or drugs may result in sleep disturbances, difficulty concentrating, irritability and anger, and feeling "on edge." Thus, withdrawal symptoms may contribute to a reinforcing cycle of self-medication among individuals with PTSD/SUD.

Research examining the temporal order of onset of development of PTSD and SUDs also provides some insight with regard to etiology (Najt et al. 2011). In the majority of cases, the development of PTSD precedes the development of the SUD (Chilcoat and Breslau 1998; Compton et al. 2000; Jacobsen et al. 2001; Stewart and Conrod 2003; Back et al. 2005, 2006). Furthermore, PTSD and SUD symptoms have been shown to covary over time. For example, Ouimette and colleagues (2010) tracked weekly fluctuations in PTSD and SUD symptoms among 35 PTSD/SUD outpatients over a 26-week period. The findings provided support

for the self-medication hypothesis and showed that increases in PTSD symptoms were associated with increases in SUD severity. More recently, Simpson and colleagues (2012) used daily interactive voice response (IVR) to examine the relationship between PTSD symptoms and same-day as well as next-day alcohol craving among 29 outpatients entering SUD treatment (26/29 had PTSD). The findings showed that greater PTSD severity was associated with greater alcohol craving and greater hyperarousal symptoms were particularly associated with craving. Next-day craving was predicted by nightmares the previous night, emotional numbing, and hypervigilance. Finally, several studies investigating civilian and Veteran patients' perceptions of the interrelationship of PTSD and SUD symptoms demonstrate support for the self-medication hypothesis (Brown et al. 1998; Back et al. 2006).

The *high-risk hypothesis* (Chilcoat and Breslau 1998; Acierno et al. 1999) posits that the lifestyle of an individual with an SUD increases the likelihood of being exposed to a traumatic event and subsequently developing PTSD. For example, individuals with SUDs often spend time in dangerous environments and engage in high-risk behaviors associated with obtaining or using substances (e.g., prostitution, theft) that may put them at risk for experiencing a Criterion A event. The *susceptibility hypothesis* posits a biological vulnerability to developing PTSD among individuals with SUDs. Individuals who engage in chronic substance use often experience anxiety and arousal and exhibit poor coping skills (e.g., more avoidant or emotion-focused coping vs. problem-focused coping) (Sharkansky et al. 1999; Stewart et al. 2000; Jacobsen et al. 2001; Staiger et al. 2009). Lastly, there is some evidence that other common factors, such as genetics, common neurophysiologic systems, described below, and prior exposure to traumatic events, may play a role in the etiology of comorbid PTSD/SUD (Stewart and Conrod 2008; Kingston and Raghavan 2009; Khoury et al. 2010; Norman et al. 2012).

120.3.2 Neurobiology

A growing body of evidence from basic science and translational studies implicates common neurobiologic pathways and abnormalities involved in anxiety disorders and SUDs. One of the bridging neurobiologic constructs between anxiety disorders and SUDs involves the role of stress. Corticotrophin-releasing factor (CRF), one of the key hormones involved in the stress response, has been implicated in the pathophysiology of anxiety, affective, and addictive disorders. Stress stimuli that activate CRF circuits are also known to potentiate mesolimbic dopaminergic reward pathways in laboratory animals. Similarly, human laboratory studies have shown that emotional stress and negative affect states increase drug craving in drug- and alcohol-dependent individuals. Animal models indicate that early-life stress and chronic stress result in long-term changes in stress responses which can alter the sensitivity of the dopamine system to stress and increase susceptibility to self-administration of substances of abuse. This may provide the neurobiologic underpinnings of the well-established relationship between early-life adversity, PTSD, and SUDs in adolescents and adults (Brady and Sinha 2005).

120.4 Assessment

Symptom assessment is critical to the effective treatment of PTSD/SUD and should ideally encompass detection of trauma exposure and substance misuse, evaluation of diagnostic criteria for PTSD and SUD, and monitoring of symptom severity (Steenkamp et al. 2011; Tucker et al. 2011). Historically, instruments assessing PTSD and SUD were predominantly developed for use with in English-speaking, westernized cultures. However, in 1990, recognizing the need for cross-cultural assessment of mental illness, the World Health Organization (WHO) developed a tool that addressed criteria for both the American Psychiatric Association's Diagnostic and Statistical Manual and the International Classification of Disease (ICD). The resulting Composite International Diagnostic Interview (CIDI; Robins et al. 1989) was a comprehensive, structured, modular interview designed to assess mental disorders – including but not limited to PTSD and SUD. As of 2011, versions of the CIDI have been translated into approximately 25 languages for use in at least 20 countries (WHO 2004). Recognition of the important roles that culture and language play in the conceptualization, experience, and expression of PTSD and SUD is increasing (Westermeyer 1995; Hollifield et al. 2002). As a result, increased efforts have been made to translate and adapt a range of instruments previously validated with English-speaking populations (e.g., Mollica et al. 1992; Westermeyer 1995; Ertl et al. 2010; Ali et al. 2012) as well as to develop culturally specific instruments, to screen, diagnose, and monitor symptoms of PTSD and SUD in a variety of international populations (e.g., Dao et al. 2012; Jayawickreme et al. 2012; Kok et al. 2013).

Regardless of cultural context, there are several general constructs relevant to the assessment of PTSD and SUD. These constructs include determination of the presence, order of onset, frequency, duration, and severity of symptoms, as well as the degree to which symptoms interfere with or impair daily functioning, employment, and interpersonal relationships (for review, see Rodriguez et al. 2012). Several reviews of PTSD assessment (e.g., Hollifield et al. 2002; Elhai et al. 2005; Rodriguez et al. 2012; Wisco et al. 2012), SUD assessment (Westermeyer 1995; Fitch et al. 2004), and PTSD/SUD assessment (e.g., Jacobsen et al. 2001; Jane-Llopis and Matytsina 2006; Najt et al. 2011) are available in the extant literature. When assessing comorbidity, special consideration should be given to the relationships between symptoms, including PTSD symptoms as potential motivators for substance misuse. Validated self-report, semi-structured, and fully structured interview instruments are available to assist with (1) screening for trauma exposure, (2) screening for PTSD/SUD symptoms, (3) determining diagnosis, and (4) monitoring symptom change over time. Table 120.1 presents a sampling of validated measures frequently used in the assessment of PTSD and SUD.

In addition to self-report and interview, biological testing is recommended in assessing SUD and may overcome some of the culture-bound limitations of self-report and clinician-administered assessments. Urine drug screening (UDS) is the most common and preferred method for detecting illicit drug use given that it is cost effective and minimally invasive and provides a quantitative means for measuring the use of substance (Preston et al. 1997; Wolff et al. 1999; Richter and Johnson 2001).

Table 120.1 Assessment and screening instruments

Screening		
Measure	Source reference	Language/country of origin
Trauma life events questionnaire	Kubany et al. 2000	English
Short posttraumatic stress disorder rating interview (SPRINT)	Connor and Davidson 2001	English
PTSD checklist – civilian version, short form	Lang and Stein 2005	English
Trauma Screening Questionnaire (TSQ)	Brewin et al. 2002	English
Primary Care PTSD Screen (PC-PTSD)	Prins et al. 2003	English
Alcohol Use Disorders Identification Test (AUDIT)	Saunders et al. 1993	English
Alcohol, Smoking, and Substance Involvement Screening Test (ASSIST)	Humeniuk et al. 2008	English
CAGE	Cooney et al. 1995	English
Drug Abuse Screening Test (DAST)	Gavin et al. 1989	English
Davidson Trauma Scale (DTS)	Ali et al. 2012; Davidson et al. 1997	English, Urdu/Uganda
HADStress	Gulden et al. 2010	Ethiopia
Diagnosis		
Clinician-Administered PTSD Scale (CAPS)	Blake et al. 1995	English
Alcohol use disorders and associated disabilities interview schedule	Grant and Hasin 1990	English
Anxiety Disorder Interview Schedule for DSM-IV (ADIS)	DiNardo et al. 1994	English
Composite International Diagnostic Interview, version 3.0	Robins et al. 1989	English, German, French, Dutch, Chinese, others
Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I)	First et al. 1996	English
Mini-International Neuropsychiatric Interview (MINI)	Sheehan et al. 1998	English
Self-Report Inventory for PTSD (SRIP)	Kok et al. 2013	Dutch
Posttraumatic stress disorder interview for Vietnamese refugees	Dao et al. 2012	Vietnamese
Symptom monitoring		
Impact of events scale	Weiss and Marmar 1996	English
PTSD Checklist (PCL)	Blanchard et al. 1996	English
PTSD Symptom Scale (PDS)	Foa et al. 1993; Ertl et al. 2010	English; Bantu
Addiction Severity Index (ASI)	McLellan et al. 1992	English
Timeline Follow-Back (TLFB)	Sobell and Sobell 1995	Universal

(*continued*)

Table 120.1 (continued)

Screening		
Measure	Source reference	Language/country of origin
Symptom interaction		
Inventory of drinking situations	Annis et al. 1997	English
Drinking motives questionnaire	Cooper 1994	English
Inventory of drug-taking situations	Annis and Martin 1985	

Additional biological assessment options exist for use as either adjunctive or alternative assessments of SUD: testing of bodily fluids, such as blood and saliva, breathalyzer analysis for recent alcohol use, hair analysis techniques, and a blood-based testing method known as percent carbohydrate-deficient transferrin (Aithal et al. 1998; Wolff et al. 1999; Arndt 2001). These methods are less frequently used due to higher cost, increased invasiveness, false positives, and/or narrow detection windows (Widdop and Caldwell 1991; Jaffe 1998).

120.4.1 Psychotherapeutic Treatment

Historically, psychosocial treatment approaches for individuals with PTSD and SUDs have adhered to the *sequential treatment model*, in which the SUD is treated first and trauma work is deferred until a period of sustained abstinence (e.g., 3–6 months) has been achieved (Schnitt and Nocks 1984; Nace 1988). This generally entails two separate providers (i.e., one provider addresses SUD and another addresses PTSD) in two separate clinics with little cross-communication. Proponents of the sequential model state that (a) continued substance use will impede therapeutic efforts and/or (b) trauma-focused work may increase risk for relapse (Nace 1988; Pitman et al. 1991). However, there is little empirical data to support these concerns. Given the high co-occurrence of PTSD and SUDs, the covarying interrelationship between PTSD symptoms and substance use severity, as well patients' preferences for treatment (e.g., less than 30 % of patients prefer sequential treatments; Back et al. 2006), recent advances in psychosocial treatments have focused on the development and testing of *integrated treatment models*. In contrast to sequential treatments, integrated treatments are provided by the same clinician and address both the SUD and PTSD concurrently. The integrated model posits that addressing the PTSD symptoms early in treatment will likely improve recovery from SUDs, particularly if substances are being used to self-medicate trauma-related symptoms (Brady et al. 2001; Back 2010; Hien et al. 2010; Mills et al. 2012).

Compelling support for the integrated model is provided by recent investigations examining the temporal course of symptom improvement among PTSD/SUD patients. Among 94 outpatients with alcohol dependence and PTSD, improvements in PTSD symptoms had an impact on improvements in alcohol-dependence symptoms, but decreases in drinking did not impact PTSD symptoms (Back et al. 2006).

Hein and colleagues (2010) replicated these findings using data from a larger sample ($N = 353$) from a National Institute on Drug Abuse (NIDA) Clinical Trials Network (CTN) study. For every unit of PTSD improvement, the odds of being a heavy substance user at follow-up decreased more than fourfold (Hien et al. 2010). A growing body of literature examining the tolerability and efficacy of addressing PTSD among SUD patients demonstrates that substance use typically decreases significantly and does not increase with the addition of trauma-focused interventions (Triffleman 2000; Brady et al. 2001; Najavits et al. 2005; McGovern et al. 2009; Hien et al. 2010; Back et al. 2012; Mills et al. 2012).

Prolonged exposure (PE; Foa et al. 2007) therapy has been deemed one of the most effective treatments available for PTSD (IOM 2008), but there is limited research exploring its efficacy in substance-abusing populations. PE involves having the patient revisit the traumatic memories (i.e., imaginal exposures) and approach safe but anxiety-producing situations in real life that are avoided by the patient (e.g., in vivo exposures). A recent meta-analysis demonstrated large effect sizes for PE in comparison to control conditions (Powers et al. 2010). Furthermore, a longitudinal study conducted among 65 patients 5–10 years after receiving PE demonstrated maintenance of effects with only 17.5 % of patients meeting diagnostic criteria for PTSD (Resick et al. 2012). PE has demonstrated effectiveness in addressing PTSD among a variety of traumatic stress populations, including victims of rape, physical assault, refugees, motor vehicle accidents, combat, terrorism, childhood abuse, and mixed trauma types (Foa et al. 2005; McDonagh et al. 2005; van Minnen and Foa 2006; Bryant et al. 2008; Nacasch et al. 2011; Resick et al. 2012). Despite that fact that PE is one of the most effective treatments for PTSD, the majority of integrated treatment interventions developed to date generally do not include PE components. Rather, treatment tends to focus on psychoeducation, exploring the relationship between PTSD symptoms and substance use, self-management of symptoms and negative emotions, and development of cognitive behavioral coping skills (Miller and Guidry 2001; Ford and Russo 2006; McGovern et al. 2009).

One of the most widely used and investigated integrated treatments to date is *Seeking Safety* (SS), a non-exposure-based 24-session manualized therapy that prioritizes establishing and maintaining safety (Najavits et al. 1998; Hien et al. 2004, 2008). Other key concepts include anticipating dangerous situations, setting boundaries, anger management, and affect regulation. In a study of 107 women comparing SS to relapse prevention (Hien et al. 2004), both treatments resulted in improved substance use and PTSD severity; however, no significant between-group differences in PTSD or SUD symptoms were observed. In a larger national multisite community study, SS was compared to a women's health education (WHE) group (Hien et al. 2009) in 353 women. Both SS and WHE resulted in significantly improved PTSD symptoms; however, neither group resulted in a significant reduction in abstinence rates over time.

More recently, Back and colleagues developed an exposure-based, manualized cognitive behavioral therapy for PTSD/SUDs (Back et al. 2001, 2012) (in press). The treatment, *COPE* (*Co*ncurrent Treatment of PTSD and

Substance Use Disorders Using *P*rolonged *E*xposure), combines evidence-based cognitive behavioral therapy for SUDs (Carroll 1998) with the key components of prolonged exposure for PTSD (Foa et al. 2007), which includes both in vivo and imaginal exposure techniques. COPE was initially trialed as a 16-session, individual intervention and tested in an uncontrolled psychotherapy development study among patients ($N = 39$) presenting with comorbid PTSD and cocaine dependence (Brady et al. 2001). In this study, no signs of increased substance were observed with the inclusion of PE. Treatment completers demonstrated significant improvements in all PTSD symptom clusters and a significant reduction in cocaine use from baseline to end of treatment (Brady et al. 2001). Reductions in PTSD and SUD symptoms were maintained at 6-month follow-up. Mills and colleagues (2012) recently completed a randomized control trial of COPE plus treatment as usual (TAU) vs. TAU alone. Participants were 103 patients (62.1 % female) with civilian PTSD and SUDs in Sydney, Australia. For this trial, COPE consisted of 13, individual sessions. From baseline to 9-month follow-up, significant reductions in PTSD symptom severity were found for both groups; however, the COPE group demonstrated a significantly greater reduction in PTSD symptom severity (mean difference –16.09) and lower rates of PTSD diagnosis as compared to the control group (56.4 % vs. 79.2 %). No significant between-group differences in rates of abstinence, number of SUD dependence criteria met, or retention were found. The findings suggest that integrated PTSD/SUD treatments employing PE techniques for PTSD can be used safely without an increase in substance use, can lead to sustained improvements across various domains (e.g., depression), and produce greater improvements in PTSD than TAU. Currently, COPE is being evaluated as a 12-session intervention in a randomized controlled trial among Veterans, and the preliminary findings are positive (Back et al. 2012). Recently, prolonged exposure has been incorporated into existing residential SUD treatment with promising preliminary results supporting its safety, feasibility, and efficacy (Henslee and Coffey 2010; Berenz et al. 2012).

120.4.2 Pharmacological Treatment

There are relatively few studies of medication treatments for co-occurring PTSD and SUDs. Sertraline, a serotonin reuptake inhibitor with FDA approval for the treatment of PTSD, was investigated in a double-blind, placebo-controlled, 12-week trial (Brady et al. 2005). Individuals with early onset PTSD and less severe alcohol dependence demonstrated improvements in alcohol use severity with sertraline treatment, while individuals with later onset PTSD and more severe alcohol dependence evidenced more favorable alcohol use outcomes when treated with placebo. Petrakis and colleagues (2005) investigated the use of agents targeting alcohol consumption, disulfiram and naltrexone, alone or in combination, in outpatients with alcohol dependence (AD) and a variety of comorbid psychiatric disorders (42.9 % PTSD). Individuals treated with either drug evidenced fewer drinking days

as compared to those on placebo, and those treated with disulfiram reported less craving. Individuals receiving active medication demonstrated greater symptom improvement (e.g., less anxiety) pre- to posttreatment as measured by the Brief Symptom Inventory. No advantage of combining disulfiram and naltrexone was reported. In a more recent study (Petrakis et al. 2011), paroxetine (serotonin reuptake inhibitor) was compared to desipramine (norepinephrine uptake inhibitor) in men with both AD and PTSD. Desipramine was superior to paroxetine with respect to study retention and alcohol use outcomes. Although the serotonin uptake inhibitors are the only FDA-approved medications for the treatment of PTSD, the current study suggests that norepinephrine uptake inhibitors may present clinical advantages. Further investigation of the use of medications as an adjunct to psychotherapeutic treatment in the treatment of co-occurring PTSD and SUDs is needed.

120.5 Conclusion

In summary, while we do not have information about the international prevalence estimates of co-occurring PTSD and SUDs, many studies suggest that these two disorders commonly co-occur. A number of theories have been posited to explain the functional relationship between these disorders, and there are clear neurobiologic connections. Medication treatments have shown some promise, but more investigation is needed. In terms of psychotherapeutic approaches, integrated treatment has been accepted as a safe and effective model of treatment. Although non-trauma-focused treatments offer some PTSD symptom reduction, data suggests that trauma-focused, exposure-based treatment offers greater symptom reduction than non-exposure-based treatment in SU treatment programs. Recent evidence demonstrates that improvement in PTSD positively impacts substance use outcomes, clearly supporting a more rigorous approach to the assessment and treatment of PTSD among patients with SUDs.

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