# TREATMENT OF PTSD AND SUBSTANCE ABUSE COMORBIDITY

*Theresa Souza & C. Richard Spates*

The best evidence suggests that PTSD is often comorbid with other Axis I and Axis II disorders. National estimates hypothesize that over 85% of individuals with PTSD will meet diagnostic criteria for at least one additional disorder. Inpatient substance abuse centers report that up to 50% of their clients also meet criteria for PTSD. The comorbidity of PTSD and substance abuse (PTSD-SA) is unlike that of other comorbid disorders due to the complex relationship between PTSD symptom presentation, the effects of the substance of choice, and the effects of withdrawal symptoms when attempts are made to eliminate the substance from the body. This paper seeks to explore three prominent theories regarding the etiology of PTSD-SA comorbidity as well as the unique relationship between the symptom presentations of these disorders. Also addressed is the controversy between clinical lore and recent empirical evidence regarding efficacious treatments for individuals with this combination of disorders.
Keywords: PTSD, substance use.

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Approximately 50% of individuals in inpatient substance abuse treatment centers will also meet criteria for comorbid PTSD (Brown et al., 1999). This combination of disorders has severe consequences for the individual in terms of course, symptom severity, and effectiveness of treatment. When working with a PTSD-SA population, there are several forms of substances which are more likely to be abused when compared to substance users that do not meet criteria for PTSD. Furthermore, these substances appear to be related to the specific symptoms pattern exhibited by the individual (Stewart, Conrod, Pihl, & Dongier, 1999). Research also indicates that some symptoms of PTSD are more likely than others to elicit substance use in general (Sharkansky, Brief, Peirce, Meehan, & Mannix, 1999). Additionally, the negative impact that substance use relapse risk situations have on an individual may further interfere with the individual's ability to cope effectively with the symptoms of PTSD, which would lead to an increase in both PTSD and SA symptoms/behaviors (Sharkansky et al., 1999). The combination of PTSD-SA also poses several barriers to effective treatment. Some of these barriers are based on clinical lore, and have not undergone the rigorous empirical testing pivotal in the field of psychology. Other barriers have been supported in the empirical field, and these must be addressed in order for treatment to be effective.

## Post-traumatic Stress Disorder

Post-traumatic Stress Disorder (PTSD) was first recognized by the American Psychiatric Association as a diagnosable condition in 1980 when it was introduced into the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DMS-III) (American Psychiatric Association, 1980). Since that time, PTSD etiology, symptomology, and treatment have been extensively studied.

PTSD is defined as the development of three categories of symptoms following exposure to a traumatic event in which the individual both (1) came into contact with an event that involved actual or threatened death or serious injury to self or others, and (2) responded to this event with intense fear, helplessness, or horror (American Psychiatric Association, 2000). In essence, exposure to a traumatic event is not sufficient to warrant a diagnosis of PTSD. The subjective, emotional experience of the individual in the aftermath of the trauma must also be taken into account (APA, 2000). The three clusters of symptoms that classify PTSD are reexperiencing, avoidance and numbing, and hyperarousal. Each of these symptom clusters is distinct and affects different areas of psychological functioning. Additionally, disturbances in each category can give rise to comorbid diagnoses associated with that cluster of symptoms that will further disrupt the individual's level of functioning (Taylor, 2006). Lastly, the DSM-IV-TR (2000) states that the symptoms must occur for a minimum of one month and cause clinically significant distress and impairment in several areas of functioning.

The first cluster of symptoms, reexperiencing, refers to the persistent emergence of thoughts and feelings associated with the traumatic event. This can occur in several modalities. These include intrusive images, distressing nightmares, acting and feeling as if the event were occurring again, and psychological distress and/or physiological reactivity when confronted with reminders of the traumatic event (American Psychiatric Association, 2000).

The second cluster of symptoms, avoidance and numbing, involves both the persistent avoidance of stimuli associated with the trauma and the numbing of general responsiveness that was not characteristic of the individual prior to the trauma (American Psychiatric Association, 2000). Examples of avoidance include all efforts to keep oneself from coming into contact with thoughts, feelings, conversations, activities, places, or people that remind the individual of the trauma. Symptoms of numbing include the inability to remember aspects of the event, decreased interest in pleasurable activities, feelings of detachment from others, restricted range of affect, and a sense of shortened future. Recent research indicates that avoidance and numbing may be separate clusters of symptoms as they differ in both their clinical correlates and in responsiveness to treatment (Taylor, 2006).

The final cluster of symptoms is hyperarousal. This category refers to persistent symptoms of increased physiological arousal that were not present prior to exposure to the traumatic event. Examples include sleep difficulties, irritability, anger, difficulties concentrating, hypervigilance, and an exaggerated startle response (American Psychiatric Association, 2000).

PTSD Comorbidity

The best evidence suggests that PTSD is often comorbid with other Axis I and Axis II disorders (Barlow, 2002). However, most clinical trials targeting symptoms of the disorder utilize individuals with PTSD as a sole diagnosis, or at least they are silent with respect to other comorbid conditions. Unfortunately, cases of uncomplicated PTSD are likely the exception, not the rule, for individuals exposed to a traumatic event. While PTSD itself is estimated to occur in 8% of the population, the prevalence rates increase dramatically in persons suffering from other mental disorders (Riggs, Volpicelli, Kalmanson, & Foa, 2003). The National Comorbidity Survey (1995) estimates that over 88% of men and 79% of women who meet the criteria for chronic PTSD also meet the criteria for one or more additional psychiatric diagnoses (Kessler, Sonnega, Hughes, & Nelson, 1995). The most frequent diagnoses comorbid with PTSD include Major Depressive Disorder, substance abuse disorders, Borderline Personality Disorder, Paranoid Personality Disorder, and Bipolar Disorder.

The prevalence rates of comorbid disorders vary considerably. Studies indicate that PTSD and depression co-occur in rates between 15% and 53% (Holtzheimer, Russo, Zatzick, Bundy, & Roy-Byrne, 2005). Higher rates are reported in refugee and war veteran samples than in other forms of trauma. Inpatient substance abuse treatment centers report that up to 50% of their clients also meet criteria for PTSD (Brown, Stout, & Mueller, 1999). An average of 17.8% of individuals with PTSD will also suffer from a personality disorders. Of these, 29% will meet criteria for paranoid personality disorder, 26% for borderline personality disorder, and the remaining personality disorder range from 10-14% respectively (Golier, Yehuda, Bierer, & Mitropoulou, 2003). Lastly, approximately 16% of individuals suffering from PTSD will also meet criteria for Bipolar Disorder (Otto, Perlman, Wernicke, Reese, Bauer, & Pollack, 2004).

The effects of comorbid diagnoses negatively affect both treatment course and outcome (Holtzheimer et al., 2005). The previously described symptoms of PTSD become intensified with the existence of further disruptions in psychological functioning introduced by the additional disorders. The specific presentation of symptoms will vary depending on the nature of the comorbid disorder. However, some commonalities to all the comorbid diagnoses are increased distress for the individual, worsened

PTSD symptoms, increased rates of premature therapy termination, and decreased rates of successful treatment (Najavits, Runkel, Neuner, Frank, Thase, Crits-Christoph, & Blaine, 2003).

## Substance Abuse

Problems with substance abuse and dependence are among the most prevalent disorders diagnosed in the United States. Prior to determining the comorbid effects of substance use and PTSD, it is important to understand the distinction between abuse and dependence. The DSM-IV-TR defines substance abuse as a maladaptive pattern of substance use leading to impairment and/or distress within a 12 month period in one or more of four different areas of functioning. These areas include (1) failure to fulfill major obligations in work, school, or home; (2) recurrent substance use in hazardous situations such as driving while impaired; (3) recurrent legal problems associated with substances including use, possession, and dealing, and; (4) continued use despite social or interpersonal problems caused or worsened by the substance use (American Psychiatric Association, 2000). In substance abuse, the individual can continue to function in daily life. However, a continuous risk of developing substance dependence is present.

Substance dependence is characterized as a more pervasive pattern of substance use. The time spent engaged in activities associated with obtaining, using, and recovering from the substance creates severe impairment in the individual's daily functioning. The degree of impairment far exceeds that which is seen in substance abuse. The DSM-IV-TR defines substance dependence as a maladaptive pattern of substance use leading to impairment and/or distress within a 12 month period in three or more different areas of functioning. These areas include (1) tolerance (either an increase in the amount of a substance to achieve the desired effect or a decrease in effects associated with use of the same amount of the substance); (2) withdrawal, characteristic symptoms associated with cessation of a substance that vary based on the substance of use; (3) use of the substance in greater amounts over a longer period of time than was originally intended; (4) persistent desire or unsuccessful attempts to control the use of the substance; (5) increasing amounts of time spent obtaining, using, and recovering from the substance; (6) reduction or elimination of social, occupational, and recreational activities not associated with substance use, and ; (7) continued substance use despite recurrent physical or psychological problems caused or worsened by use of the substance (American Psychiatric Association, 2000). For the purposes of this paper, substance use will refer to substance dependence as described by the DSM-IV-TR.

### Substance Types

The DSM-IV-TR (2000) refers to a substance as a drug of abuse, a medication, or a toxin. These substances are further grouped into 11 different classes based on their physiological and psychological effects. The classes utilized are alcohol, amphetamines, caffeine, cannabis (marijuana), cocaine, hallucinogens, inhalants, nicotine, opiates, phencyclidine, sedatives, hypnotics, and anxiolytics. Each class was formed based upon the substance's effects on nervous system functioning. Several classes in the DSM-IV-TR can be combined into larger groups based upon these effects, and were separately identified for reasons to be presented shortly.

Scientifically, there are five general categories of substances. The first is depressants. This category of substance results in behavioral sedation. Their purpose is to induce relaxation in the individual. The two DSM-IV-TR classes of substances that can be described as depressants are alcohol, and the sedative, hypnotic, and anxiolytic group (Durand & Barlow, 2006).

The second category of substances is stimulants. These substances result in mood elevations. They cause the individual to become more alert and active. The DSM-IV-TR classes included in this category are amphetamines, cocaine, nicotine, and caffeine (American Psychiatric Association, 2000).

The third category of substances is opioids. These substances produce analgesic effects, as well as euphoria. This class of substance is not combined with any other class in the DSM-IV-TR.

The fourth category is the hallucinogens. These substances alter the sensory perceptions of the user. They can result in delusions, paranoia, and hallucinations. Substances that are included in this class from the DSM-IV-TR are cannabis and other hallucinogens (Durand & Barlow, 2006).

The final category of substances includes all other drugs of abuse. These are substances that do not fit neatly into one of the previous categories. They include inhalants, steroids, and some over-the-counter medications. They produce a wide variety of effects on the brain and body that may span more than one of the previously discussed classes (American Psychiatric Association, 2000).

The prevalence rates of substance dependence are difficult to determine due to the secrecy and illegality of the behaviors associated with the disorder. Alcohol has historically been considered to be the most prevalent drug of choice. It is estimated that over 15 million Americans are dependent on alcohol alone. Twenty-three percent of adults report engaging in binge drinking. This can be defined as consuming five or more alcoholic drinks in one sitting. In regards to college aged adults, the numbers rise to over 40% (Durand & Barlow, 2006). The prevalence rates of other substances vary widely.

## PTSD-SA Comorbidity

As previously stated, approximately 50% of individuals in inpatient substance abuse treatment centers will also meet criteria for comorbid PTSD (Brown et al., 1999). This combination of disorders has severe consequences for the individual in terms of course, symptom severity, and effectiveness of treatment.

When working with a PTSD-SA population, there are several forms of substances which are more likely to be abused when compared to substance users that do not meet criteria for PTSD. Furthermore, these substances appear to be related to the specific symptoms pattern exhibited by the individual (Stewart, Conrod, Pihl, & Dongier, 1999). This can occur in two different forms. The first refers to the PTSD symptom clusters of intrusion, arousal, numbing, and avoidance. Self-medication through substances in order to relieve symptoms of PTSD can lead to abuse and dependence for the substance class whose function is associated with this type of symptom. Second, classes of substances that serve to exacerbate the symptoms of PTSD can be negatively reinforced by substance-intoxication-induced or withdrawal-induced intensification (Stewart et al., 1999).

Many classes of abused substances are correlated with PTSD. These include alcohol, opiates, anxiolytics, analgesic, and cocaine. (Stewart et al., 1999). Although it is difficult to obtain accurate data regarding alcohol use, this class of substance is believed to be the most commonly abused substance among individuals with PTSD. Studies indicate that alcohol consumption is correlated with an increase in PTSD symptoms associated with the arousal symptoms.

Through urinalysis, it is possible to determine with increased accuracy the prevalence of illicit substance use in PTSD individuals. Studies indicate that opiate abuse occurs in approximately 23% of PTSD cases. This is followed by marijuana (20%), Benzodiazepines (11%), and cocaine (8%). Other categories of substances, when present, were found in under 5% of the cases (Calhoun, Bosworth, Hertzberg, Sampson, Feldman, Kibry et al., 2000).

Substance dependence on anxiolytics is correlated with the symptoms clusters of arousal and numbing, while analgesic dependence is correlated with the symptom clusters of arousal, intrusion, and numbing (Stewart et al., 1999).

Estimates indicate that the prevalence rates of PTSD and cocaine dependence are high. Studies suggest that approximately 45% of cocaine-dependent individuals will meet criteria of PTSD (8% PTSD individuals (Calhoun et al., 2000)) at some point during their lifetime. Furthermore, 24% of cocaine-dependent individuals will meet criteria for current PTSD (Back, Dansky, Carroll, Foa, & Brady, 2001). In general, cocaine and opiate users report higher rates of exposure to traumatic events when compared to abusers of other groups of substances. Additionally, cocaine users appear more vulnerable to developing PTSD after exposure to a trauma, as well as experience more severe symptoms of PTSD and increased levels of social impairment (Back et al., 2001). PTSD and cocaine dependence appear to be related through the second pathway (Stewart et al., 1999). The effects of cocaine serve to intensify PTSD symptoms, especially while one is withdrawing from cocaine. As a result, any attempts to reduce cocaine use serve to induce both symptoms of cocaine withdrawal and a concurrent increase in distress associated with trauma symptoms (Back et al., 2001).

Research also indicates that some symptoms of PTSD are more likely than others to elicit substance use in general (Sharkansky, Brief, Peirce, Meehan, & Mannix, 1999). Within the substance dependence literature, a taxonomy of situations that lead to relapse has been proposed. This taxonomy includes the following intrapersonal risk situations: negative emotional states, negative physical states, positive emotional states, testing personal control, and giving in to temptations or urges. Additionally, several interpersonal risk situations are also proposed. These include social pressure to use substances and positive interpersonal interactions (Marlatt & Gordon, 1985). Several of these risk situations are associated with the course and symptomolgy of PTSD-SA.

First, consistent with the self-medication hypothesis, PTSD-SA individuals experience an increased likelihood for exposure to high-risk, trauma inducing environmental conditions than the general population (Kellogg & Triffleman, 1998). The relationship between substance use and violent personal histories has been well-documented. Threats to one's self may be presented through gang activity, accidents while under the influence of substances, illegal activity to obtain substances, and violent acts committed while under the influence of substances (Kellogg & Triffleman, 1998). Exposure to these types of situations can result in the risk factors of negative emotional states; and in many instances, negative physical states. As previously stated, the primary requirement for PTSD diagnosis is the existence of an event that involved actual or threatened death or serious injury (American Psychiatric Association, 2000). A cycle of trauma and substance use can be created through these combined diagnoses.

Second, both the intrapersonal risk factors of negative emotional states and negative physical states are characteristic symptoms of PTSD (Sharkansky et al., 1999). It is not essential for the individual to experience repeated traumas in order to be affected by the combined symptoms of PTSD, or to be at increased risk for relapse as a result of them. Several of Marlott's (1985) relapse risk factors exist within the diagnosis of PTSD. Negative emotional states are elicited by the intrusive and hyperarousal symptoms characteristic of the diagnosis (American Psychiatric Association, 2000). Negative physical states are induced by the physiological reactions to the fear and panic associated with stimuli that remind the individual of the traumatic event.

Third, some evidence exists to suggest that PTSD symptomology itself is not solely a result of interpersonal conflict, negative emotional states, and physical illness, but that once PTSD has been established, it can be elicited by these states as well (Sharkansky et al., 1999).

Last, the negative impact that substance use relapse risk situations have on an individual may further interfere with the individual's ability to cope effectively with the symptoms of PTSD, which would lead to an increase in both PTSD and SA symptoms/behaviors (Sharkansky et al., 1999). The intrusive symptoms of PTSD have demonstrated increases in both levels of distress and negative

emotional states. This led to subsequent increases in drug and alcohol cravings for the individuals. Additionally, analog studies indicate increased craving for substances following the presentation of trauma-related stimuli as compared to neutral stimuli (Coffey, Schumacher, Brimo, & Brady, 2005).

To summarize, of the various relapse risk factors proposed in the Marlatt taxonomy of substance use; unpleasant emotions, physical discomfort, and interpersonal conflict appear to elicit higher rates of relapse in PTSD-SA populations than in substance dependent only populations.

PTSD-SA Treatment Considerations

The combination of PTSD and SA not only intensifies the symptoms of both disorders, but also poses several barriers to effective treatment. First, PTSD-SA individuals rarely present for treatment with anxiety concerns. While these individuals present for treatment at a higher rate than SA only individuals, their presenting problem to both hospitals and outpatient treatment centers is substance abuse (Brown, Stout, & Mueller, 1999). As a result, the comorbid PTSD frequently remains undiagnosed. Second, individuals with PTSD-SA demonstrate markedly less improvement during treatment than SA only individuals in several areas. These include the acquisition of more adaptive coping skills, belief in positive cognitions regarding their abilities, and general ability to effective manage psychological distress. The PTSD-SA groups demonstrated greater levels of avoidance coping mechanisms than the SA only groups (Ouimette, Finney & Moos, 1999). Third, PTSD-SA individuals demonstrate increased levels of interpersonal conflict. This leads to a greater level of difficulty developing rapport with the therapist and compliance with the treatment protocol (Sharkensky et al., 1999). The combination of misdiagnosis, decreased therapeutic rapport, increased psychological distress, and decreased ability to learn more adaptive coping skills leads to an increased likelihood of relapse for substance abuse and increased PTSD symptom presentation. This also leads to decreased confidence in one's ability to successfully complete treatment and increase one's quality of life. It is hypothesized that these are all factors in the elevated rates of early treatment termination in this population.

Pathways to PTSD-SA Diagnoses

While it has become apparent that various forms of comorbidity exist when dealing with PTSD and SA separately, simply assessing for and identifying these conditions are not the only problems faced by researchers and clinicians. The pathway through which the disorders emerged jointly in the individual should be determined in order to identify the most appropriate treatment modality for the individual (Jocobsen & Kosten, 2001). Although this area of study is recent, early stages of research indicate that knowledge of the etiological pathway to PTSD and its comorbid disorders may influence the method of therapeutic intervention likely to elicit the greatest degree of changes in psychological functioning. This relationship is present in both successful treatment, and in the exasperation and intensification of existing symptoms (Hien et al., 2004).

At present, there are three proposed pathways through which comorbidity can occur. The first theory views PTSD as a secondary disorder (Otto et al., 2004). This pathway occurs in individuals who suffer from a history of psychiatric illness. The presence of a psychiatric condition with these individuals places them at increased risk of being exposed to a traumatic event, and they also have an increased risk of developing PTSD following exposure to the traumatic event. One such example involves individuals with a diagnosis of schizophrenia. Epidemiological studies conducted with this population found rates of comorbid PTSD ranging as high as 42% (Muenzenmaier, Castille, Shelly, Jamison, Battaglia, Opler, & Alexander, 2005). The theory posits that the symptoms and lifestyles associated with schizophrenia predispose an individual to victimization. Such factors include psychosis, paranoia, homelessness, and physical and psychological abuse.

This pathway can relate directly to substance use. Individuals with substance abuse disorders frequently engage in high-risk behaviors in order to obtain their substance of choice. These behaviors include drug dealing, theft, assault, prostitution, gang activity, and more (Kellogg & Triffleman, 1998). These behaviors have the potential of placing the individual in a variety of traumatic situations that could subsequently lead to the development of PTSD. Once established, the symptoms of PTSD can increase the individual's use of substances in order to cope with the additional stressors that tax their psychological functioning.

The second pathway involves PTSD as the primary disorder. These individuals displayed no signs of severe psychological distress prior to their exposure to the traumatic event and the subsequent emergence of PTSD. It is hypothesized that the intrusive thoughts, nightmares, avoidance behaviors, hyperarousal, dissociation, and depersonalization symptoms characteristic of PTSD serve as catalysts for further deterioration in psychological functioning (Brown, Stout, & Mueller, 1999). For example, the increased physiological arousal in public settings associated with PTSD frequently gives rise to the avoidance of an increasing number of situations. The decreased anxiety associated with this avoidance negatively reinforces such behavior. As a result, the individual becomes more and more secluded from previously enjoyable activities and the social support that has demonstrated positive effects on psychological functioning. Given time, these actions may develop into Major Depressive Disorder, which is the most frequent comorbid diagnosis to PTSD (Otto et al., 2004).

Similarly, the individual may turn to alcohol or other substances in order to reduce the negative impact of the intrusive symptoms of PTSD, or to calm themselves from the hyperarousal symptoms (Riggs, Rukstalis, Volpicelli, Kalmanson, & Foa, 2003). Unfortunately for the individual, the use of substances will not permanently eliminate the symptoms. As the drug leaves the system, the symptoms return and or intensify. A negative reinforcement cycle develops. The individual continues to utilize substances in order to reduce or avoid the symptoms of PTSD. As with most substances, physical tolerance to the drug develops. The individual's body will require increasing amounts of the substance in order to achieve the desired effects. In time, physical dependence on the substance develops as well (Coffey et al., 2005). This is most likely in instances where the primary disorder has gone untreated or not treated successfully. Research on alcoholism has demonstrated that individuals, who suffered from intense anxiety and panic symptoms prior to their use of alcohol, were more likely to consume alcohol in 8 out of 12 situations compared to alcoholic without a history of anxiety (Sharkansky et al., 1999).

The third and final pathway is referred to as reciprocal increased vulnerability. This theory recognizes that individuals with histories of trauma, particularly in childhood, are at heightened risk for several psychological disorders as adults. Each of these disorders increases the likelihood of further trauma and decreases levels of healthy psychological functioning. As a result, proponents of this theory posit that the primary diagnosis is inconsequential for the individual. Only the presentation of symptoms should be considered when treating the individual, and this treatment should be tailored towards that person's specific needs (Meenzenmaier et al., 2005).

Treatment Target Rules: Clinical Lore

In regards to efficacious treatment for these comorbid disorders, current practices may be inconsistent with empirical studies due to commonly held beliefs and clinical lore on how to best treat substance dependence. Two main theories exist in the therapeutic community which may be limiting the amount of research available as well as the dissemination of alternate forms of treatment. These are the Pandora's Box hypotheses and the self-medication hypothesis.

The Pandora's Box hypothesis states that any attempts to address trauma related material in the incipient stages of substance dependence treatment would severely interfere with treatment effectiveness.

Encouraging the individual to recall trauma material is believed to result in an influx of negative thoughts and emotions with which the individual is not equipped to cope at this stage in therapy. In essence, "Pandora's box" would be opened and neither the client nor the therapist would be capable of dealing with the resultant effects. As a result, proponents of this theory believe that the individual will be incapable of maintaining abstinence from substances and would either resume or intensify their drug behaviors (Hien, Cohen, Miele, Litt, & Capstick, 2004).

The Pandora's Box hypothesis is sustained by a number of beliefs. The first is that most PTSD-SA individuals initially seek treatment for substance dependence. Advocates state that it would be inappropriate to then treat the clients for a problem for which they did not seek treatment. Secondly, substance abuse is associated with a decreased tolerance for negative affect. It is believed that addressing trauma-associated material early in treatment will increase their level of negative affect (Triffleman, Carroll, Kellogg, 1999). Third, many proponents of exposure therapy for PTSD state that this type of treatment requires the ability to cope with powerful affective and cognitive material. They believe that substance users do not meet these requisite criteria (Brady, Dansky, Back, Foa, & Carroll, 2001). Fourth, anecdotal evidence is presented that suggests PTSD or combined PTSD-SA treatment will be ineffective unless abstinence is first achieved. Last, clinical lore suggests that PTSD symptoms will reduce or remit with the cessation of substance use (Triffleman et al., 1999).

The second theory, self-medication, posits a differing view of PTSD-SA comorbidity and occurs in two parts. Proponents of this view suggest the substance dependence as a secondary diagnosis. In the first part, it is believed that the individual began to abuse substances in order to cope with the symptoms associated with PTSD. In essence, the substance abused served, in part, as a self-induced medical intervention. The effects of the substance on the brain and body reduced the anxiety and/or depression present when the symptoms were at full intensity (Hien et al., 2004).

The second aspect of the self-medication hypothesis concerns withdrawal symptoms. By this point in the substance use, the individual has developed physical dependence on the substance. Any attempt on the part of the addict to reduce this substance use results in a vicious cycle of withdrawal symptoms, increased PTSD symptoms, and increased cravings for the substance associated with each. The individual has previously been conditioned to escape the PTSD symptoms by use of a substance, so this pattern is repeated. Any further attempt to halt substance use only serves to reinforce the existing pattern (Jacobsen et al., 2001).

One common theme in both of the theories found in clinical lore is the idea that the substance dependence diagnosis should be the primary focus of treatment. The main reason for this stems from a multitude of ideas, few of which were formulated based on empirical data. Prior to addressing the validity of therapeutic focus in this population, it is essential to understand the method by which PTSD is treated. This is especially important because much of the clinical lore cited above is advanced by professionals who may have no background in empirically supported treatments for PTSD, even if they are trained substance abuse counselors. Their perspective is therefore based on their experience but not informed by evidence based practice for treating this complex disorder.

## PTSD Treatment

Although various forms of treatment exist for PTSD, Exposure Therapy has shown the highest levels of effectiveness (Taylor, Thordarson, Federoff, Maxfield, Lovell, & Ogrodniczuk, 2003). Exposure therapy can be administered in three forms; imaginal, interoceptive, and in vivo. Each of these involves the client coming into contact with some form of the pivotal traumatic memory or event. In imaginal exposure, the client may be asked to recount the traumatic memory to the therapist multiple times in order to process the events. In time, the intense emotional and physiological arousal states

associated with the memory loose their potency. Interoceptive exposure involves exposing the client to the physiological reactions, sensations, and subjective perceptions of distress associated with the traumatic reaction. The client learns how to monitor and down-regulate their bodily reactions to trauma-relevant imaginal or interoceptive sources of stimulation. In vivo exposure involves the client physically confronting some physical or environmental context of the trauma and remaining in that situation until their arousal level declines (Taylor, 2006).

Other treatments utilized for PTSD include cognitive therapy, relaxation training, and EMDR. However, an element of exposure is present in each of these therapeutic modalities, and research suggests that the exposure component alone is responsible for the positive effects of treatment (Spates & Koch, 2004; Davidson, 2001, Van Etten & Taylor, 1998). Over the past 30 years, several meta-analytic reviews have been conducted on the various therapies utilized in the treatment of PTSD (Van Etten & Taylor, 1998; Waller, Mulick, & Spates, 2000). All of these have concluded that Exposure Therapy and Eye Movement Desensitization and Reprocessing (EMD/R) demonstrate the most consistent empirical evidence regarding effectiveness in the treatment of PTSD. While EMD/R contains strong connections to Exposure Therapy, there is evidence to suggest that clients find this type of therapy to be less aversive than other forms of exposure (Waller et al., 2000). This information is essential when considering treatment options, as the drop-out rates for PTSD treatment are high. When combining the PTSD drop-out rate to the substance abuse relapse rates, the need to identify intervention strategies that are both effective and palatable for the clients is increased exponentially.

Exposure Therapy, like any therapy, does not come without side-effects to the client. While the procedure is quite safe, and the client is never placed in a dangerous situation, Exposure Therapy may increase levels of arousal and the frequency of nightmares associated with the traumatic event during the course of treatment. These effects dissipate quickly during treatment, but many clients report discomfort associated with them (Taylor, 2006). Clinical lore suggests that deliberately subjecting substance dependent individuals with PTSD to a treatment that is known to increase arousal symptoms is analogous to encouraging them to abuse substances in increasing amounts (Hien et al., 2004). However, as previously stated, this theory appears to stem from "educated guesses" about the responses of substance abusing individuals and does not stem from empirical data. One guess is that it arises in the context of substance treatment where therapy is delivered by individuals who are trained only to treat the SA disorder and not trained in treating the trauma reaction and thus only witness increased arousal with no available effective intervention at their disposal.

Substance Use Treatment

Several types of therapies have been proposed for the treatment of substance abuse. The most frequently utilized treatment strategy involves a 12-Step program, which typically falls under the caveat of Alcoholics Anonymous (AA) or Narcotics Anonymous (NA). There exists no clear definition or single explanation as to the treatment modality and techniques of 12-Step programs. While there are certain core concepts, assumptions, hypotheses, and ideas that can be used to identify the approach in a broad sense, there are no clear guidelines as to the process for conducting the meetings (Rotgers, Morgenstern, & Walters, 2006). The wide varieties of substance abuse programs that follow a 12-Step model are heterogeneous. The program itself can best be described as a social movement or fellowship. Within the program itself, there is no formal assessment process, diagnosis, case conceptualization, dispensation of medication, treatment plan, case management, individual therapy, or group therapy (Rotgers et al., 2006).

The practice of 12-Step meetings began in 1953 and views substance abuse from the disease model. The belief inherent in the program is that total abstinence from the substance is the only method through which control can be taken over one's life. The meetings are generally conducted by community members who have achieved abstinence. Stories of the trials and tribulations of other abusers are related

to show that no individual is alone in their battle against substance dependence. Individuals that have maintained abstinence for some length of time serve as sponsors for newer members, and offer support during the beginning stages of abstinence. Each member is taught a series of 12 steps designed to admit powerlessness over the substance, make amends for past wrongs, and grow closer to a spiritual entity (Rotgers et al.; 2006).

Another treatment strategy for substance abuse involves the use of pharmacological agents to decrease cravings for substances, decrease the impact of withdrawal symptoms on the client (Vocci, Acrid, Elkashef (2005), decrease the physiological effects that substances have on the body and/or create unpleasant side effects for the individual who utilizes substances while taking an alternate form of medication (Kleber, 2004).

The first pharmacological intervention is known as agonist substitution. This involves providing the substance abuser with a drug that has a similar chemical make-up as their drug of abuse. The new drug is less harmful on the body of the user. The goal of this form of therapy is to eliminate the body's dependence on the drug of abuse without causing the body to simultaneously cope with the symptoms of withdrawal. The individual can then be titrated off of the "safe" drug until complete abstinence is reached (Miller & Carroll, 2006). Unfortunately, this method has limitations. While the first stage of the treatment has shown effectiveness in substance abuse treatment, the second has demonstrated multiple problems. The clients frequently become dependent on the "safe" drug. There is also a high relapse rate for the original drug once the "safe" drug is eliminated (Miller & Carroll, 2006). One example of this type of pharmacological intervention is methadone maintenance therapy for opiate use. Methadone is a long-lasting synthetic opiate that has shown effectiveness in achieving abstinence, as well as decreasing the craving for, drugs such as heroin and hydromorphone. This has subsequently resulted in reductions in relapse, decreases in criminal activity, and reduced mortality rates as compared to those actively taking the original substance (Vocci, Acri, Elkashef (2005).

Antagonist drug therapies are designed to counteract, or block, the euphoric effects of substances on the user. In essence, if an individual were to use the addictive substance while taking the medication, they would not achieve the "high" that they are accustomed to experiencing when taking the drug (Miller & Carroll, 2006). The goal is to break the stimulus-response pairing between ingestion of the substance and the resultant feelings of euphoria and thus extinguish the drug taking behavior. This method does not result in dependence on the new drug. However, the disadvantage of this type of pharmacological therapy is in the side effects. Ingestion of a drug antagonist will elicit immediate withdrawal symptoms from the drug of abuse. This experience is highly aversive for the client, which decreases the probability that they will continue to take the antagonist when the doctor or therapist is not present (Kleber, 2004). One example of this form of pharmacological therapy is Naltrexone. Naltrexone is a long-acting opioid antagonist that will block the effectiveness of opioids for up to 72 hours (Kleber, 2004).

The final form of pharmacological therapy for substance abuse is aversive treatment. These drugs both block the euphoric effects of the substance of abuse (antagonistic properties) and make the ingestion of the abused substance highly unpleasant for the individual (Miller & Carroll, 2006). One example of this type of therapy is disulfram, or Antabuse, for alcohol addiction. If an individual were to drink alcohol while taking disulfram, the result would be nausea, vomiting, elevated heart rate, and respiration. Other subjects have reported feelings of anxiety and paranoia as well. As a result, treatment compliance to this form of pharmacotherapy is poor (Vocci et al., 2005). O'Brian (1996) conducted an analysis of pharmacological treatments for substance abuse. The analysis concluded that abstinence from drugs is generally not achieved with initial courses of treatment, regardless of treatment modality. However, the outcomes for substance abuse treatment utilizing pharmacological medication was comparable to those of other chronic conditions such as diabetes and hypothyroidism (O'Brian, 1996).

PTSD-SA Treatment Implications

To summarize thus far, relapse is common in substance abuse treatment, and the rates of relapse only increase when PTSD occurs in conjunction with substance use. Current treatments for each of these disorders separately are vastly different. The main component of PTSD treatment is exposure therapy, while the treatment of choice for substance use involves a 12-Step program targeting abstinence from the substance. Clinical lore suggests that prior to treatment for PTSD, the substance use must be addressed or exposure therapy would at best fail, and at worse incite increased substance use (Jocobsen & Kosten, 2001). However, this belief is based on "hunches" and not on empirical data. Few studies exist to date testing the validity of these strongly-held beliefs, and those that do exist suggest an alternate view of PTSD-SA comorbidity.

An emerging view states that PTSD treatment should be introduced earlier in the therapy. These individuals note that purposefully ignoring the symptoms of PTSD while targeting substance use leaves the clients vulnerable to increasing levels of distress with no appropriate coping mechanisms. By addressing PTSD symptoms early in treatment, much suffering could be alleviated. This could serve as a motivating factor for continuing treatment and increase confidence that abstinence can be both achieved and maintained (Triffleman et al., 1999).

Empirical Evidence Regarding PTSD-SA Treatment

Early studies have shown that joint PTSD-SA treatment, regardless of treatment modality, is essential in achieving and maintaining abstinence from substances. Those individuals who receive PTSD treatment in addition to SA treatment in the first three months of intervention are 3.7 times more likely to be free of substances at a 5-year follow-up than individuals who receive only SA treatment (Ouimette, Moos, Finney, 2003). The same study tracked the clients yearly for 5 years. Those clients who continued to experience problems with substances in the 5$^{th}$ year of the study received PTSD treatment at that time. Those clients were 4.6 times more likely to be free of substances at follow-up than those who did not receive the treatment (Ouimette et al., 2003). This indicates that SA treatment alone was not sufficient to maintain abstinence from substances. However, it is important to note that of the total sample, only 27% were in remission from substances at follow-up. There remains much work to be done in the area of PTSD-SA treatment.

One recent area of research addressed the question of treatment tolerance. Can individuals with substance dependence tolerate exposure therapy prior to the complete cessation of substance use, or will exposure therapy lead to increased drop-out rates and/or increased substance use?

Preliminary studies indicate that exposure therapy is tolerated well in the substance abusing population. Brady et al., (2000) studied 39 cocaine dependent individuals. Of those who prematurely terminated treatment, 75% did so prior to the onset of the exposure component, which was in session 6 of the treatment protocol. Of those available for a follow-up survey, none of the participants reported exposure therapy as the reason for termination. Reasons included admittance to an inpatient facility, transportation and scheduling difficulties, and relocation (Brady et al., 2000).

Hien et al., (2004) studied 96 women with PTSD-SA comparing cognitive behavioral therapy, including an exposure component, relapse prevention, and community care. The researchers found no differences between the active treatment groups in client drop-out rate. Furthermore, both active treatment groups demonstrated significant decreases in symptom severity. The results continued after treatment was completed and follow-up data indicated sustained improvement in functioning (Hien et al., 2004). Furthermore, meta-analyses report treatment completion rates for exposure based PTSD-SA treatments as 39%, which is higher than the 28% completion rate reported be the National Institute on Drug Abuse for substance abuse treatment alone (Coffey et al., 2005).

The aforementioned meta-analysis by O'Brian (1996) included the effectiveness of pharmacological interventions in relation to comorbid disorders. While the analysis concluded that abstinence from drugs is generally not achieved for SA only treatment, when comorbid conditions were considered, research was very poor. Pharmacological treatment interventions have demonstrated efficacy with comorbid depression and opiate addiction, but effectiveness studies with other substances were lacking. No treatment outcome studies on pharmacological interventions for comorbid substance dependence and anxiety disorders were found at that time (O'Brian, 1996).

Subsequent to the O'Brian (1996) review, there have been several studies conducted on pharmacological interventions, few of which included substance abusers with comorbid PTSD. Several studies support the use of pharmacological interventions for opiate dependence (Sigmon, Wong, Chausmer, Liebson, & Bigelow, 2004; Sigmon, 2007; Trafton, Minkel, & Humphreys, 2006).

When including comorbid PTSD participants, opioid substitution therapy demonstrated positive results. Subjects with and without PTSD treated for SA with Methadone showed comparable, significant reductions in use of heroin (Trafton, Minkel, & Humphreys, 2006). The number of days of heroin use decreased by 91% for individuals with comorbid PTSD, and these results were maintained at a 12 month follow-up. Use of cocaine, marijuana, and prescription opioids also decreased in both groups (Trafton et al., 2006). However, in order to achieve these SA symptom reductions in the comorbid PTSD group, higher doses of opiate medication and additional psychological treatment sessions were required. Furthermore, the PTSD-SA patients demonstrated increased levels of psychiatric symptoms both at intake as well as throughout SA treatment. These symptoms did not diminish regardless of the patients' responsiveness to SA treatment (Trafton et al., 2006).

The second question that has received much needed attention in the literature is the effectiveness of PTSD only or concurrent PTSD-SA treatment on comorbid populations. Two exposure-based treatments have been developed for PTSD-SA comorbidity. The first is concurrent treatment of PTSD and cocaine dependence (CTPCD), and the other is substance dependence PTSD therapy (SDPT) (Coffey et al., 2005).

CTPCD is an 8 week PTSD-SA protocol consisting of twice weekly sessions, each of 90 minute duration. The program was initially developed for cocaine-dependent individuals, but can be adapted for use with other substances (Back et al., 2001). The treatment package contains components of the cognitive skills training, relapse prevention, cognitive restructuring, in vivo, and imaginal exposure therapies. The primary goals of the program are to (1) educate clients about the relationship between substance abuse and PTSD, (2) achieve and maintain abstinence from substances, (3) decrease severity of PTSD symptoms and (4) reduce high-risk behaviors that could lead to future traumatization and substance abuse (Back et al., 2001). The first 5 sessions focus on Psychoeducation regarding exposure therapy, PTSD, and SA. Additionally, core coping skills for the cessation of substance use are presented. These include sessions dealing with cravings for substances, motivation and commitment to cease SA, assertiveness skills, and decision making (Coffey et al., 2005). Exposure therapy is begun in session 6 and continues throughout the remainder of treatment.

SDPT is a manual-based individual therapy for PTSD-SA. It is a compilation of a variety of treatments for both PTSD and SA. The goals of SDPT are threefold. They include abstinence from substance use, maintenance of abstinence during the course of trauma work, and overall reduction of PTSD severity (Triffleman, Carroll, Kellogg, 1999). SDPT consists of twice weekly individual therapy sessions over the course of approximately 20 weeks. Treatment is presented in two phases. The first phase, lasting 12 weeks, focuses on psychoeducation for PTSD, SA, and the effects of comorbidity as well as SA treatment. Specific skills taught during this phase of treatment include examination of craving and dysphoria associated cognitions, generalization of alternate cognitions, and increasing management of

emotional and physical states (Triffleman et al., 1999). The final 8 weeks, phase two, focuses on PTSD treatment. This phase is a combination of Stress Inoculation Training and Exposure Therapy. Both phases of treatment include in-session tasks and out-of-session homework assignments.

## Future Directions

On the basis of this review, several changes are needed in the arena of PTSD-SA treatment. First, accurate assessment of psychological functioning prior to the onset of treatment is a necessity. Studies indicate that the majority of PTSD-SA individuals that seek treatment, will do so for their SA problem only. These individuals may then receive SA treatment on multiple occasions before the PTSD is diagnosed. Research supports the notion that PTSD-SA individuals utilize hospital and outpatient services at a much higher rate than SA only individuals, yet they do not receive differing forms of treatment, primarily because the PTSD remains undiagnosed (Brown, Stout, & Meuller, 1999). This is disturbing as approximately 50% of individuals with PTSD will also meet criteria for a substance use disorder (Brown et al., 1999).

Once a PTSD-SA individual is accurately diagnosed, additional barriers exist to effective treatment. The long-held opinion that psychopathology of any type cannot be addressed until the substance use problem has been addressed is currently under debate in the psychological community, especially in relation to comorbid PTSD. Recent empirical studies surrounding PTSD and Depression (O'Donnell et al., 2004), PTSD and OCD (Gershuny et al., 2002), as well as PTSD and SA (Coffey et al., 2005) have indicated that treating the disorder that emerged first (primary pathway diagnosis) can result in remission, or improvement, of the secondary diagnosis without specifically targeting this diagnosis in treatment. The psychological field is in need of additional research to replicate these results and to determine if altering current SA treatments to address the primary pathway diagnosis first will increase treatment success. Currently, the relapse rate for substance abuse is high, and this rate increases dramatically as co-occurring psychological stressors and diagnoses are identified. PTSD appears to be one of the most difficult comorbid disorders to treat with SA due to the intensification of PTSD symptoms while withdrawing from substances and learning alternate coping skills to address the PTSD symptoms (Sharkansky et al., 1999).

Within the past decade, several studies have addressed the untested belief that PTSD-SA individuals cannot tolerate exposure therapy, the preferred treatment for PTSD, until they have achieved abstinence from their substance of abuse (Brady et al., 2001; Triffleman et al., 1998; Coffey et al., 2005). The results of these studies have indicated that exposure therapy is well-tolerated. The drop-out rates are comparable to PTSD only individuals. Furthermore, the treatment outcomes for those individuals that complete PTSD-SA treatment are better than for those who receive SA only treatment (Brady et al., 2001).

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Author Contact Information:
Theresa M. Souza, MS
Western Michigan University
230 Morhouse St
Galesburg, MI 49053
269-665-9915

Responsible for major organization of the manuscript, major writing and final editing of the manuscript

C. Richard Spates, Ph.D.
Professor & Director Anxiety Disorders Laboratory
Department of Psychology
Western Michigan University
Kalamazoo, MI 49008
Tel: 269 387-4329
Fax: 269 387-4550

Assisted with the organization, direction to selected resources, and assisting with editing of the manuscript