

SUBSTANCE USE DISORDER AND TRAUMA

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Substance use disorder (SUD) is characterized by an intense, enduring, and often irresistible desire for subjective effects of substances (*cravings*); impaired insight; poor judgment and risky decision-making about substance-seeking behavior; markedly reduced desire for naturally rewarding social relationships and activities; reduced sensitivity to euphoric effects of substances over time (*tolerance*); uncomfortable and sometimes life-threatening withdrawal symptoms that develop when stopping substance use; negative emotions when unable to obtain access to substances (e.g., dysphoria, anxiety, irritability); compulsive substance seeking that persists despite repeated damaging consequences to self, family, and society; and multiple relapses (American Psychiatric Association, 2013; Crean, Crane, & Mason, 2011; Goldstein et al., 2009; Kalivas & O'Brien, 2008; Koob & Volkow, 2010; National Institute on Drug Abuse [NIDA], 2011; Schoenbaum, Roesch, & Stalnaker, 2006; Tiffany & Wray, 2012; Volkow & Li, 2004). The structural brain abnormalities and associated cognitive and behavioral disruptions seen in individuals with SUD are so striking that many experts have come to refer to the disorder as a disease of the brain (Volkow & Li, 2004).

Different patterns of disease expression can be seen for the various substances of abuse depending on factors, such as dosing, duration of use, route of administration (e.g., intravenous versus inhalation), and even gender and culture (Back & Hyman, 2014; Koob, 2008). Although there is currently debate as to whether certain substances, such as marijuana,

are of less concern than others, there is no doubt that, overall, SUD is a tremendous public health concern that contributes to numerous preventable social and health problems and financial costs to society (Child Welfare Information Gateway, 2014; National Institute on Alcohol Abuse and Alcoholism [NIAAA], 2005; NIDA, 2005, 2007, 2011). SUD is one of the most common psychiatric disorders in the United States with a 14.8% lifetime rate in the community (Kessler et al., 2005), and new substances of abuse are emerging all the time based on novel chemical formulations.

CO-OCCURRENCE WITH TRAUMA AND POSTTRAUMATIC STRESS DISORDER

Decades of research, as well as repeated clinical observation, show important linkages between SUD and trauma as well as trauma-related disorders such as posttraumatic stress disorder (PTSD; Najavits, 2015). In general, the linkages are strongest between the disorders of SUD and PTSD and thus much of this chapter focuses on that comorbidity. For example, a number of prospective studies indicate that PTSD renders one vulnerable to SUD and vice versa (e.g., Chilcoat & Breslau, 1998; Shipherd, Stafford, & Tanner, 2005; Wolitzky-Taylor, Bobova, Zinbarg, Mineka, & Craske, 2012). In a longitudinal study of 1,007 adults who were assessed and followed up 3–5 years later, Chilcoat and Breslau (1998) found that a diagnosis of PTSD on initial assessment increased the risk of later development of drug abuse or dependence. Likewise,

Wolitsky-Taylor and colleagues (2012) conducted a prospective study of 627 adolescents who were assessed and then reassessed over a 4-year follow-up period. They found that anxiety disorders (including PTSD) at baseline were significant predictors of subsequent onset of an alcohol use disorder (AUD). In addition to the strong association between the two disorders, both SUD and trauma also are associated with various other psychiatric conditions, including mood disorders, anxiety disorders, and personality disorders.

Rates of PTSD-SUD comorbidity vary depending on the population and sampling methodology. Some of the highest rates of co-occurrence have been found for combat and sexual assault in terms of trauma types (Bailey & Stewart, 2014). Moreover, PTSD-SUD co-occurrence has been found across adolescent, adult, and older-adult developmental stages.

Pathways

Several etiological pathways have been proposed to account for the high rates of co-occurring SUD and PTSD. Predominant models include (a) the self-medication hypothesis, in which PTSD increases the risk for development of SUD; (b) the substance-induced hypothesis, in which SUD increases the risk for development of PTSD; and (c) the shared vulnerability model, in which a third common factor contributes to the development of both SUD and PTSD (Meyer, 1986; Smith & Randall, 2012). What follows is a brief review of the self-medication hypothesis, which is contrasted with the substance-induced hypothesis. Shared cognitive and neurobiological vulnerabilities implicated in the co-occurrence of SUD and PTSD then are reviewed.

Self-medication and substance-induced hypotheses.

The *self-medication hypothesis* proposes that individuals with PTSD use substances to alleviate painful emotional states (Khantzian, 1997). Symptom relief negatively reinforces drug use, leading to a pathological reliance on substance use to manage enduring negative affective states, and heightening the risk for later development of SUD (Buckner, Heimberg, Ecker, & Vinci, 2013; Marmorstein, 2012). High rates of individuals

with anxiety disorders—the category for PTSD in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1994), although the fifth edition (American Psychiatric Association, 2013) now situates PTSD in its own category—endorse self-medication as a goal of their substance use, and this subpopulation was four times more likely to develop a new SUD 3 years after diagnosis (Crum et al., 2013).

Despite evidence for the self-medication hypothesis, several challenges remain. First, not all substances lead to reductions in physiological arousal or negative affect. For example, although cannabis may reduce physiological arousal, there is less support for alcohol's tension reduction properties (Buckner et al., 2013), highlighting possible dissociations between the objective and subjective benefits of alcohol use. Second, support is strong for the *substance-induced hypothesis*, which shows that substance use actually may cause or exacerbate symptoms of PTSD directly through its impact on the central nervous system (via reduced or increased activity of certain neurotransmitters and neurobiological systems) or indirectly through negative psychosocial consequences, which contribute to increased levels of stress and anxiety (Smith & Randall, 2012).

Shared vulnerability models. Several cognitive mechanisms have been identified as shared vulnerability factors that underlie the strong association between SUD and PTSD (Buckner et al., 2013; Naifeh, Tull, & Gratz, 2012). *Attentional bias* has been implicated in the onset and maintenance of both SUD and PTSD (Bacon & Ham, 2010). Individuals with SUD and PTSD have a tendency to preferentially attend to threat-, or substance-related cues in the environment (Bardeen & Orcutt, 2011; Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & Van IJzendoorn, 2007; Field, Marhe, & Franken, 2014), which may contribute to increased anxiety and drug craving. A recent study revealed that cocaine-dependent individuals with PTSD displayed a greater attentional bias toward cocaine cues after exposure to a trauma-related script, highlighting the role of cognitive processes

in the link between these two disorders (Tull, McDermott, Gratz, Coffey, & Lejuez, 2011). *Anxiety sensitivity*—the fear of anxiety-related symptoms (e.g., increased heart rate, sweating, muscle tension) secondary to beliefs that these sensations may be physically harmful or may lead to negative social consequences—also has been implicated (Reiss & McNally, 1985). Cross-sectional and prospective studies indicate that anxiety sensitivity is associated strongly with both PTSD symptom severity and alcohol or drug consumption and can predict the development of a new AUD and SUD years later (Chandley, Luebke, Messman-Moore, & Ward, 2014; DeMartini & Carey, 2011; Elwood, Hahn, Olatunji, & Williams, 2009; Gillihan, Farris, & Foa, 2011). *Outcome expectancies*—positive or negative beliefs about the consequences of substance use—also have been examined as factors in the relationships between SUD and PTSD (Carmody et al., 2012). Expectancies are presumed to influence levels of craving, substance-seeking behaviors, and frequency and quantity of substance use. Studies consistently find these positive associations among individuals with SUD alone and in those with co-occurring SUD and PTSD (Carmody et al., 2012; Goldsmith, Thompson, Black, Tran, & Smith, 2012).

Overall, these cognitive processes may be moderated by sex, social context, and other cognitive variables, such as drinking refusal, self-efficacy, and coping skills (Pasche, 2012). Given that some of these studies were cross-sectional and used homogenous samples (e.g., predominantly White college students or treatment-seeking populations) with single substance use, these findings may not generalize to a more racially or ethnically diverse sample with co-occurring SUD.

Evidence from basic science points to several neurobiological abnormalities that are common to both sets of disorders. Studies have found disruptions in several neurotransmitter levels, including dopamine, norepinephrine, and serotonin in the development of SUD or PTSD (Norman et al., 2012). These neurotransmitters play a role in both regulating the stress response system as well as influencing the incentive salience of drugs of abuse (Norman et al., 2012). Likewise, the hypothalamic-pituitary-adrenal (HPA) axis, a part of the neuroendocrine system

responsible for the management of stress, also has been implicated in both the development of SUD and PTSD (Koob & Zorrilla, 2010; Norman et al., 2012). Overall, evidence supports multiple causal pathways in the development of SUD-PTSD comorbidities, which may vary as a function of the specific types of SUD and anxiety disorders involved and the onset and course of these disorders. Nevertheless, once both sets of disorders have developed, a bidirectional process occurs whereby alcohol or drug use's short-term effects on anxiety or PTSD symptoms and the exacerbation of symptoms that are caused by chronic alcohol or drug intoxication or withdrawal effects are mutually reinforced (Smith & Randall, 2012).

Childhood Trauma and SUD

Childhood trauma, and more broadly childhood maltreatment (physical, sexual and emotional abuse, and neglect), shows important associations with SUD. Trauma can influence the initiation of substance use behavior and the development and course of SUD, including relapse following treatment. Although trauma can lead to dysfunction at any stage of development, childhood trauma is especially important in *setting the stage* for SUD that is often chronic and severe (Bailey & Stewart, 2014). Stress, especially chronic, uncontrollable, or high-intensity traumatic stress that occurs early in life, may interact with social, personality, and genetic or biological variables to influence an individual's vulnerability to develop SUD (Brady, Killeen, Brewerton, & Lucerini, 2000; Cleck & Blendy, 2008; De Bellis, 2002; Enoch, 2011; Enoch et al., 2010; Goeders, 2004; Hyman & Sinha, 2009; Sinha, 2008, 2009). Yet, many children and adolescents who experience even the most severe forms of early life trauma do not go on to develop SUD, indicating that factors such as gene-environment interactions, extent of exposure to substances, and receipt of high-quality social support following trauma may influence resilience (Cicchetti, 2013; Enoch, 2011; Ozbay et al., 2007). Nevertheless, for vulnerable individuals, such as those raised in impoverished and challenging social environments with parents and peers who serve as poor models of adaptive coping, the development of SUD may have an etiological base in early life traumatic stress.

Indeed, rates of childhood trauma are high in clinical and community samples of individuals with SUD (e.g., Medrano, Hatch, Zule, & Desmond, 2002; Hyman, Paliwal, & Sinha, 2007). Individuals with SUD also report greater childhood victimization than those without SUD (e.g., Enoch et al., 2010). Stress-coping theories of SUD (Wills & Hirky, 1996) posit that aversive life circumstances that significantly elevate subjective distress can increase the likelihood that a child or adolescent will be motivated to turn to substance use to avoid or escape from stress, enhance positive affect, and reduce negative affect. This is not to say that traumatized youth will not seek out substances for other reasons, such as for social or recreational goals, but that the motivation for stress-relief and numbing or avoidance of aversive emotional states may be more salient for children and adolescents who are subjected to traumatic victimization. This may be most evident in children who are subjected to multiple types of victimization (*poly-victimization*) that occur for long periods of time.

Early life stress, including childhood traumatic stress, may influence the development of SUD through the derailment of normal neurological development, disruption of the brain's stress-circuitry, alterations in gene expression within the dopamine reward pathway, and the deterioration of brain regions involved in adaptive coping and self-regulation (Brady & Back, 2012; De Bellis, 2002; Enoch, 2011; Hien, Cohen, & Campbell, 2005; Sinha, 2008). In effect, childhood trauma may increase survivors' sensitivity to future life stressors, undermine their ability to cope with stress, and intensify the rewarding effects of substances (Brady & Back, 2012). Indeed, in samples of adults with SUD, greater childhood trauma severity is associated with greater subjective distress and greater self-reported use of avoidance as a coping strategy (Hyman, Paliwal, & Sinha, 2007; Medrano et al., 2002).

In addition to the neurobiological insults related to the high stress of traumatic victimization, the poor social learning environments common to traumatized children may lead to the restricted development of reasoning and decision-making capacities and overall critical-thinking skills necessary to solve

problems and regulate affect (Kostolitz, Hyman, & Gold, 2014). Research indicates that not only stress but also the individual's ability to effectively cope with stress (through effective problem-solving, conflict resolution, and behavioral and cognitive control), contributes to the likelihood of substance use (Hyman & Sinha, 2009; Wills & Hirky, 1996). Specifically, individuals who engage in greater use of problem-focused coping and who rely less on emotion-focused and avoidant strategies typically have better outcomes, including a lower likelihood of substance use initiation and, for treatment-seekers, greater protection against substance use relapse (Wills & Hirky, 1996). As these capacities are negatively affected by childhood trauma, heightened stress reactivity concomitant with diminished coping ability can place traumatized children at high risk for SUD throughout the life span.

Moreover, the additive impact of the cognitive and behavioral deficits associated with SUD (e.g., reliance on substance use as a coping strategy, impulsivity, poor judgment and decision-making) and PTSD (e.g., dissociation, explosive anger) can result in increased risk of additional stress through greater affiliation with deviant peers, violence perpetration, financial problems, academic problems, interpersonal problems, occupational problems, criminal behavior, and possible retraumatization.

Even though SUD is more prevalent in men (Compton, Thomas, Stinson, & Grant, 2007) the association between childhood trauma and the development of SUD appears to be stronger in women (Hyman, Garcia, & Sinha, 2006; MacMillan et al., 2001; Simpson & Miller, 2002; Widom & Hiller-Sturmhöfel, 2001). Moreover, two recent studies have demonstrated effects of childhood trauma on substance use relapse in women but not in men (Heffner, Blom, & Anthenelli, 2011; Hyman et al., 2008). In these relapse studies, associations between childhood trauma severity and relapse outcomes were present in the absence of or after controlling for PTSD, indicating the importance of addressing childhood trauma in treatment, particularly among women, regardless of whether or not they developed PTSD.

Effects of SUD on Brain Stress and Reward Pathways

A caveat often overlooked when conceptualizing the stress–SUD relationship is the effect that chronic substance use behavior has on brain stress and reward circuitry. According to Koob (2008), “Addiction comprises three stages: preoccupation/anticipation, binge/intoxication, and withdrawal/negative affect, in which impulsivity often dominates at the early stages, and compulsivity dominates at terminal stages” (p. 11). As the individual becomes more deeply affected by SUD, greater withdrawal symptoms create a physiological stressed state of negative emotionality, which takes on a greater role in driving the SUD (Koob, 2008). It is not uncommon at this point to hear individuals with SUD state that they no longer feel much of a high from using substances (because of tolerance), but are just using to feel *normal*. For people with SUD who also suffer from the day-to-day difficulties associated with their traumas and other problems in living, withdrawal symptoms associated with chronic substance use may serve to increase stress-sensitivity and intensify trauma-related symptoms. Thus, in addition to being a response to traumatic-stress related brain changes and social learning deficits, chronic substance use itself may compound detriments to brain stress systems, further impair cognitive functioning, and further increase the stress response in traumatized individuals.

PTSD-SUD: Information Processing Models

Although it is well established that cognitive factors play a prominent role in the etiology of both PTSD and SUD (Brewin, 2001; Ehlers & Clark, 2000; Field & Cox, 2008; Goldman, 1999; Tiffany, 1990), there has been surprisingly little examination of cognitive mechanisms that may shed light on the co-occurrence of the two disorders. Emotion and cognition are linked inextricably, and as such, the way in which emotionally relevant information is processed in the brain may be one mechanism by which PTSD and SUD are related to each another. Accordingly, we discuss the role of information processing, as it is relevant to PTSD, to SUD, and to how the two disorders may present together.

Information processing and PTSD. Nearly four decades ago, P. J. Lang (1979) addressed the importance of cognitive processes in traumatic stress by introducing the concept of *fear networks*. In this conceptualization, fear-related information (e.g., trauma cues) would lead to the activation of associated cognitions and then to behavioral responses. P. J. Lang (1979) and others (Chemtob et al., 1988; Foa, Steketee, & Rothbaum, 1989) later built on the fear network concept by positing that trauma-relevant stimuli activate information networks containing information about the emotional meaning of that event, guiding behavioral responding.

Information processing and substance misuse.

P. J. Lang’s (1979) model of information processing in PTSD is in many ways similar to theories of information activation and information processing that can be found in the substance cognition literature (Dunn & Goldman, 2000; Field & Cox, 2008; Steele & Josephs, 1990; Tiffany, 1990). Although the specific cognitive mechanisms that are posited may vary from model to model, all cognitive models of substance use and SUD share the premise that relevant stimuli initiate changes in cognitive processes. These changes then in turn have implications for SUD outcomes.

Cognitive processes linking PTSD and SUD. To date, the literature has examined cognitive processes that are specific to one set of symptoms or the other (PTSD or SUD). Yet an intriguing possibility is that cognitive processes may link PTSD to SUD. That is, as cognitive models of PTSD and SUD both point to the critical role of information processing in determining behavioral symptom outcomes, and suggest that these processes may be put into action in the context of motivationally relevant cues, it is reasonable to look to these differences in information processes as a possible mechanism of effect underlying this comorbidity.

One possible mechanism of association pertains to the allocation of cognitive resources, and one way that this has been examined in both the PTSD and SUD literatures is using the modified Stroop task (e.g., Constans, McCloskey, Vasterling, Brailey, & Mathews, 2004; Cox, Fadardi, & Pothos, 2006). In the modified Stroop paradigm, participants

are asked to correctly identify color for target words from psychologically relevant word groups. Response time to color naming of a target word is interpreted as an index of emotional-cognitive processing. Although the literature is mixed (cf. Kimble, Frueh, & Marks, 2009), at least some evidence from the PTSD literature suggests that information processing is slowed in the context of threat cues. A number of studies have found general patterns of slowed responding in those with PTSD (Ashley, Honzel, Larsen, Justus, & Swick, 2013; El Khoury-Malhame et al., 2011; Kaspi, McNally, & Amir, 1995; McNally, Kaspi, Riemann, & Zeitlin, 1990; McNally, Amir, & Lipke, 1996), with more interference for trauma words. Litz and Keane (1989) posited a mechanism for this slowing: as the individual with PTSD biases attention to threat cues, cognitive resources are drawn away from concurrent tasks such as rapid responding. This slowing may represent either an attentional bias, a depletion of cognitive resources, or both.

This conceptualization is consistent with cognitive-motivational conceptualizations of alcohol and other drug information processing that assert that slowing to alcohol and drug words may reflect underlying cognitive processes that shed light on a person's affective or motivational state (cf. Field & Cox, 2008; Sayette, 1999). Indeed, in a number of studies using cognitive paradigms with substance use stimuli (e.g., Cox, Blount, & Rozak, 2000; Cox, Brown, & Rowlands, 2003; Read & Curtin, 2007; Stewart, Hall, Wilkie, & Birch, 2002), reaction time slowing is observed in response to alcohol and other drug stimuli. An interpretation of these findings is that working memory resources become occupied by motivationally relevant stimuli, resulting in slower behavioral responding.

How might this slowing connect PTSD and SUD? Numerous possibilities exist. For example, in their review of theories of emotion in alcohol research, A. R. Lang, Patrick, and Stritzke (1999) posited that emotion motivates drinking behavior by serving as an "action disposition" (p. 334) that points an organism toward a certain course of behavior. One possibility is that when confronted with trauma cues, self-regulation capacities controlled by executive functioning may become compromised,

thus rendering vulnerability to substance misuse (Bugg, 2012). Another possibility is that, as trauma fear networks are activated, so too may be self-medication cognitions relevant to substance use. These and other conceptualizations are intriguing possibilities and represent empirical explorations that have the potential to add substantially to the current PTSD-SUD knowledge base. Yet to date, little or no research has examined implicit substance cognitions as they may relate to PTSD-SUD interplay. As the field continues to refine our understanding of the complex mechanisms that may undergird PTSD-SUD co-occurrence, this will be an important area of future investigation.

The Role of Traits in PTSD-SUD Associations

A growing literature also has sought to understand the co-occurrence of PTSD-SUD, as well as other psychiatric comorbidity, by considering higher order traits that may account for two sets of symptoms presenting at the same time (e.g., Clark, 2005; Kramer, Polusny, Arbisi, & Krueger, 2013; Krueger, 1999; Krueger & Markon, 2006). From this *shared vulnerability* perspective, certain personality traits represent temperamental endophenotypes that predispose individuals to both PTSD and SUD (Grekin, Sher, & Wood, 2006; Miller, 2003; Read, Griffin, Wardell, & Ouimette, 2014; Tull, Barrett, McMillan, & Roemer, 2007). Two traits suggested to be of fundamental importance to the expression of psychopathology broadly (Clark, 2005) and PTSD and SUD in particular (Elkins, McGue, Malone, & Iacono, 2004; Grekin et al., 2006; McGue, Slutske, & Iacono, 1999; Miller, Greif, & Smith, 2003; Tull et al., 2007) are negative emotionality and disinhibition. Negative emotionality, or neuroticism, is the propensity to experience negative affect (e.g., sadness or depression, anger, anxiety). Disinhibition is a tendency toward risk-taking, impulsive action, and attitudes not bound by traditional rules or social expectations. These two trait facets have been characterized as internalizing and externalizing predispositions (Krueger, 1999; Miller, Fogler, Wolf, Kaloupek, & Keane, 2008; Wolf et al., 2010), dispositions that may represent vulnerabilities to the specific expression of PTSD and

SUD. That is, PTSD and SUD may emerge following trauma exposure, as a vulnerable system is stressed by exposure to a traumatic event (Kramer et al., 2013). A number of empirical studies have offered support for this conceptualization (e.g., Miller et al., 2008; Read, Merrill, Griffin, Bachrach, & Khan, 2014; Vaidyanathan, Patrick, & Iacono, 2011).

A variant of the trait vulnerability perspective is the *scar hypothesis* whereby psychopathology leaves its mark (scar) on the individual, thus increasing vulnerability for other psychological disorders (Lewinsohn, Steinmetz, Larson, & Franklin, 1981). In this case, it is not PTSD *per se*, but the effect of PTSD on traits that confers risk for substance abuse. The identification of these *scars* can facilitate understanding of the development of or relapse to comorbid psychopathology (Beevers, Rohde, Stice, & Nolen-Hoeksema, 2007). Following the scar hypothesis, both negative emotionality and disconstraint may worsen acutely following traumatic stress, compromising self-regulatory processes and increasing risk for substance abuse (Miller & Litz, 2004). In contrast to a self-medication conceptualization, which posits direct associations, this trait vulnerability pathway is indirect.

In 2006, Miller, Vogt, Mozley, Kaloupek, and Keane explicitly tested self-medication and trait vulnerability models of PTSD-AUD. In a large sample of Vietnam veterans, these investigators tested cross-sectional relations between PTSD and AUD through negative emotionality and disconstraint, and found support for a mediated pathway through both traits. The authors concluded that associations between PTSD and AUD could best be characterized as indirect, occurring through temperamental traits and presumably exacerbated by PTSD.

Beyond the influence of internalizing and externalizing vulnerabilities that may link PTSD to substance use outcomes, other mechanistic variables also have been posited. As with trait characteristics, such as negative emotionality and disconstraint, these other individual-level factors can act as pre-existing vulnerabilities to both PTSD and SUD, increasing the likelihood of developing the two disorders, or they can serve as mediators through which one symptom presentation (PTSD or SUD) influences the other. Some of the most important

of these are characteristics that pertain to an individual's ability to regulate or to manage negative emotions. Among the most prominently implicated of these are emotion regulation and coping. Emotion regulation is the capacity to perceive, understand, regulate, and respond appropriately to strong emotions (Gratz & Roemer, 2004; Rottenberg & Gross, 2007). Deficits in emotion regulation have been observed in individuals with PTSD (Ehring & Quack, 2010; Tull et al., 2007), SUD (Berking et al., 2011; Fox, Axelrod, Paliwal, Sleeper, & Sinha, 2007), and those with co-occurring PTSD-SUD (McDermott, Tull, Gratz, Daughters, & Lejuez, 2009; Radomski & Read, 2016; Weiss, Tull, Anestis, & Gratz, 2013). Coping can best be described as the way in which an individual approaches challenges (Menaghan, 1983). Coping is thought to be an important etiological contributor to both PTSD and SUD, as they present both uniquely and together (Corbin, Farmer, & Nolen-Hoeksema, 2013; Hruska & Delahanty, 2012; Moos & Moos, 2006; Read, Griffin, et al., 2014; Ullman, Filipas, Townsend, & Starzynski, 2006). As with internalizing and externalizing traits, both emotion regulation and coping have been suggested as potential vulnerability factors, preceding the onset of PTSD-SUD, as well as mechanistic variables, as part of a mediated pathway from one to the other.

Self-Regulatory Deficits

Findings from a body of developmental literature on self-regulatory systems from a neurobehavioral and clinical perspective provide one potential lens through which to consider the mechanisms by which trauma and PTSD may be linked to SUD. The potential serious short- and long-term consequences of childhood exposure to trauma have come into sharper focus because of the recent work of developmental researchers. Intriguing developmental findings of relevance to the SUD field suggest that childhood victimization and associated traumatic stress may influence maturing self-regulatory systems. These systems may mediate or moderate the relationship between early traumatic stress and later SUD in women.

The lack or loss of self-regulatory abilities is considered by some experts to be the most far-reaching

effect of psychological trauma in both children and adults (Cloitre et al., 2009). Self-regulatory processes are internal organizing functions that filter, coordinate, and temporally organize experience (Posner & Rothbart, 2000). These functions are essential for a range of subsequent capacities, including development of cognitive problem-solving; information processing; and emotional, interpersonal, and communication skills. Given the central role of self-regulation in managing, controlling, and coordinating emotion, cognition, and behavior, problems in self-regulation cut across multiple diagnostic domains and reflect broad classes of maladaptive psychosocial functioning. Specific higher order cognitive abilities subsumed under the construct of self-regulation include attentional control, strategic planning, initiation and regulation of goal-directed behavior, self- and social monitoring, abstract reasoning, cognitive flexibility, and the ability to organize and adaptively utilize information contained in working memory (Raffalli, Crockett, & Shen, 2005; Rothbart, Posner, & Kieras, 2006).

Findings demonstrate that childhood trauma exposure disrupts many of these functions and is associated with cognitive and behavioral dysregulation (Cicchetti & Toth, 2005), hypervigilance (Lieberman, Chu, Van Horn, & Harris, 2011), and biased information processing and cognitive problem-solving (Hadwin, Garner, & Perez-Olivas, 2006). Emotion regulation also includes the ability to modulate and tolerate emotional experience with deficits evidenced by affect lability, poor affect tolerance and expression, and maladaptive emotion-focused coping. Childhood traumatic stress exposure challenges maturing emotional regulation mechanisms at multiple developmental points by interfering with the preschool tasks of differentiating affective states, with the school-age child's developing capacity to elaborate on affective expression, and with the adolescent's task of achieving an understanding of the origin and consequences of emotions (Cicchetti & Toth, 2005). The developmental achievement of self-regulation rests on successful acquisition of these skills and has consequences for self and social development. Appropriate self-regulation is critical to successful family,

peer, and social functioning (McClelland, Cameron, Wanless, & Murray, 2007).

The ability to control and modulate emotional stimulation is thought to provide the foundation for capacity to form social relationships (Kim & Cicchetti, 2010; Lopes, Salovey, Côté, Beers, & Petty, 2005). For example, Cole and Putnam (1992) have proposed that core self-concepts in large part are defined by the capacity to regulate internal states and by behavioral responses to stress. Some have argued that the problems of emotion regulation and interpersonal functioning are a relatively distinct feature of childhood trauma and derive from the trauma's disruptive impact on the achievement of the developmental goals of affect regulation and interpersonal relatedness (Cicchetti & Toth, 2005).

Recent studies indicate that problems with emotion regulation in trauma-exposed children are apparent in pre- and early teen years, notably a critical time period for the development of SUD. For example, a study comparing the emotion management skills of abused girls with nonabused healthy controls, ages 6 to 12 years old, found that the abused girls were more likely to hide their feelings and to have extreme emotional reactions (Shipman, Zeman, Penza, & Champion, 2000). The abused girls in this study also had fewer adaptive coping strategies, and they expected less support and more conflict in situations in which anger was expressed. Thus, even at an early age, childhood abuse is associated with insufficient coping skills, problems in handling strong emotions (particularly anger), and limited expectations of others as resources in emotionally difficult situations. Studies examining the specific role of emotional dysregulation in reactive aggression among children provide further support for the link between a child's inability to self-soothe and to modulate negative feelings during social interactions and aggressive behavior (Lee & Hoaken, 2007).

Recent findings from the PTSD literature provide evidence confirming that trauma has the most profound impact when onset occurs during early childhood or adolescence, whereas the effects are less pervasive in individuals who are older at first traumatic exposure (Banyard, Williams, & Siegel, 2001). In contrast, the younger the victims are at the time of the trauma exposure and the longer the

duration of the trauma, the more likely they are to have problems in adulthood in a variety of areas, in addition to PTSD symptoms such as behavioral impulsivity, affective lability, and aggression (van der Kolk, Roth, Pelcovitz, Sunday, & Spinazzola, 2005). Clearly, adult revictimization also plays a role. Studies comparing individuals with childhood trauma only and individuals with childhood trauma and subsequent victimization in adulthood show that the revictimized individuals are consistently more troubled, particularly in the domains of affect modulation, anger management, and interpersonal relationships (Classen, Palesh, & Aggarwal, 2005).

These and other similar findings resulted in the identification of a syndrome of psychological problems in adulthood shown to be frequently associated with histories of prolonged and severe interpersonal abuse (Herman, 1992; van der Kolk et al., 2005). This cluster of symptoms, called complex PTSD or disorders of extreme stress not otherwise specified, includes alterations in self-regulatory systems described earlier: regulation of affective impulses (e.g., difficulty modulating anger), cognitive processes (e.g., disruptions in attention, memory and consciousness), and relationship to others (e.g., problems with intimacy and trust).

Neurobiological Functioning

Although a thorough review of the neurobiological literature is beyond the scope of this chapter, relevant empirical evidence has demonstrated that self-regulatory deficits may have neurobiological correlates. Neurobiological research on trauma and the stress response, largely derived from studies of war veterans with PTSD, has documented that adults with PTSD demonstrate neurobiological changes to the volume and activity levels of major structures in the limbic system, including the amygdala and hippocampus (Sapolsky, 2000; Teicher, Andersen, Polcari, Anderson, & Navalta, 2002), hypersensitivity of the HPA axis to cortisol (Yehuda, 2006), and release of neurotransmitters leading to dysregulation of arousal systems and the endogenous opioid systems (Heim & Nemeroff, 2009). The main cluster symptoms of PTSD correspond to these documented neurobiological changes; all involve self-regulatory functions. Thus, a possible

vulnerability to SUD use could be viewed on a neurobiological level as an effort to address related self-regulatory deficits. For example, individuals may use substances to dampen the biological effects of dysregulated stress response systems (De Bellis, 2002) increasing the probability of SUD.

Self-Medication Model in Relation to Self-Regulation Processes

The symptoms of complex trauma—encompassing self-regulatory affective and cognitive systems—represent developmental domains, which also have been implicated as vulnerability factors for SUD. The self-medication hypothesis described earlier is consistent with the developmental findings reviewed earlier that suggested that exposure to childhood trauma disrupts the self-regulatory processes, in turn leading to long-term difficulties in these areas. Self-regulation deficits have been implicated in the initiation and maintenance of SUD (de Wit, 2009; Khantzian & Schneider, 1986; Volkow, Koob, & McLellan, 2016) and thus may play an important mediating role between trauma exposure and subsequent substance use. For example, findings from the SUD literature show that difficulty in emotional regulation and tolerance of painful feelings, inability to self-soothe, and instability of behavioral control is typical of adolescents and adults with SUD (Aguilar de Arcos et al., 2008; Cheetham, Allen, Yücel, & Lubman, 2010). Self-medicating with substances can lessen the effects of hyperarousal and numbing symptoms in individuals with PTSD. The hyperarousal symptoms would be diminished or masked by alcohol or other depressants, thereby providing temporary relief from the dysregulated feeling states that go along with PTSD.

TREATMENT

The presence of SUD also affects how PTSD needs to be addressed in treatment. Addressing PTSD-SUD is not about just applying treatments for each, but requires conceptualization of how each disorder affects the other and how to engage in strategies to address each without worsening the other. It requires a careful balancing act. Too often, however, clinicians do not address either the SUD or the PTSD.

Although comprehensive coverage of the PTSD-SUD treatment literature is not possible, several general points can be made following Najavits (2015) and drawing on Benish, Imel, and Wampold (2008); Imel, Wampold, Miller, and Fleming (2008); Najavits and Anderson (2015); Najavits and Hien (2013); and Powers, Halpern, Ferenschak, Gillihan, and Foa (2010).

PTSD-SUD Studies Consistently Show Positive Outcomes

In more than 40 outcome studies thus far, results consistently have been positive across numerous domains. Earlier concerns that addressing PTSD and SUD together would worsen the patient's state have not been borne out. But it is important to remember that all studies used new models specifically designed for PTSD-SUD or made major changes to classic PTSD therapies to make them feasible and tolerable for SUD samples.

No Greater Benefit Is Gained From Past-Focused Models

All studies using a PTSD exposure (past-focused) approach combined it with an SUD coping skills (present-focused) approach, but none outperformed models that were present-focused alone at end-of-treatment. Also notable is that all studies that included a past-focused component were delivered in an individual modality rather than in a group and almost always were restricted to less complex samples than the present-focused studies, in keeping with the PTSD-alone literature. *Less complex* means that patients typically were excluded if they had drug use disorders (rather than alcohol only); current domestic violence, homelessness, suicidality, violence, cognitive impairment, or serious mental illness; or criminal justice involvement. In contrast, present-focused models primarily were delivered in a group modality and accepted a much broader range of patients.

Overall, with PTSD-SUD patients, greater emotional intensity in sessions does not equal better outcomes. Both present- and past-focused models may be helpful to patients, based on readiness of the patient and clinician, training, setting, and other contextual factors. Such findings are consistent with

psychotherapy research broadly, which shows that when compared to each other manual-based models perform equally well, including those developed for PTSD and those developed for SUD (Benish et al., 2008; Imel et al., 2008; Powers et al., 2010). Clinicians have a lot of choice in which models to use, and the evidence thus far does not indicate any greater benefit from past-focused models for this population than the easier-to-tolerate, typically less expensive, present-focused models.

The Seeking Safety Model Has the Strongest Evidence Base

The most evidence-based model for co-occurring PTSD-SUD at this point is Seeking Safety (SS). SS has been the subject of the majority of PTSD-SUD studies (more than 20), including the most randomized controlled studies. It is also the model with the most number of studies by independent investigators, which are less subject to positive bias than studies by the treatment developer (Chambless & Hollon, 1998). SS has had consistently positive outcomes and is the only model thus far to outperform a control on both PTSD and SUD (Hien et al., 2015; Najavits & Hien, 2013). SS is also the most widely implemented PTSD-SUD model in treatment programs.

PTSD May Be Easier to Treat Than SUD

In the literature thus far, when there were differences between conditions, they were more often on PTSD or other mental health variables, and less often on SUD. This may indicate that in patients with PTSD-SUD, PTSD and mental health issues may be easier to treat than SUD. That remains a question for future research but does fit clinician perceptions (Back, Waldrop, & Brady, 2009). This pattern also fits the current view of PTSD as amenable to time-limited treatment, whereas SUD (severe SUD in particular) is conceptualized as a chronic relapsing disorder needing ongoing care (Arria & McLellan, 2012).

RECOMMENDATIONS FOR PRACTICE

The following clinical suggestions from Najavits (2015) can help improve care for patients with PTSD-SUD:

- *Attend to both PTSD and SUD if the patient has both.* This may seem simple but all too often is not done in practice. There are many reasons for it, including lack of sufficient training on PTSD or SUD in professional degree programs. The disorders also are known to evoke strong emotional reactions in clinicians and, for SUD in particular, stigma and negative attitudes (Imhof, 1991; Pearlman & Saakvitne, 1995).
- *Ensure accurate assessment.* Accurately identify both PTSD and SUD, along with other diagnoses and problems that may be present. Use validated instruments rather than home-grown instruments or ad hoc questions.
- *Work together with the patient to explore treatment options.* Collaboration is crucial. Ultimatums often drive the patient away and reinforce distrust of professionals.
- *Be compassionate.* Listen closely and convey empathy. PTSD-SUD patients have typically lived lives of extraordinary pain. They are often highly sensitive and feel enormous self-hatred. They are used to being misunderstood by their own families and communities and, unfortunately, sometimes by clinicians.
- *Recognize differences among PTSD-SUD patients.* These patients vary in many ways, including the presence or absence of co-occurring personality disorders, physical health problems, financial concerns, and legal issues. They also differ in strengths.
- *Understand that severity of PTSD and SUD is key, not order of onset.* Some clinicians erroneously believe that if the PTSD occurred first (which it does in most cases), then addressing PTSD is primary. Yet it is not the order of onset of PTSD and SUD, but rather the *severity* of each, that determines the treatment plan. For example, if a patient has a severe SUD and PTSD, it is widely understood by those knowledgeable about SUD that the patient needs to address the SUD immediately and in a serious way (e.g., some combination of detoxification; residential care; 12-step groups, SMART Recovery, or other self-help; counseling; medication). Major studies of patients with PTSD-SUD that included numerous patients with substance dependency consistently have situated their studies in settings in which patients currently are receiving substantial SUD care (e.g., Boden et al., 2012; Coffey, Stasiewicz, Hughes, & Brimo, 2006; Foa et al., 2013; Hien et al., 2009; McGovern, Lambert-Harris, Alterman, Xie, & Meier, 2011)—even though the typical order of onset for such patients is PTSD preceding SUD.
- *Directly monitor substance use.* Good SUD care requires the clinician to actively inquire about substance use at every visit. Ideally, this use will be verified by urinalysis, breathalyzer, or other biological methods. Even if those are not possible, which may be the case in private practice settings, it is crucial to use a valid self-report instrument and to have a clear written contract on substance use.
- *Do not push exposure-based (past-focused) treatments.* Patients sometimes are pushed too strongly into past-focused models with statements such as, “You’re avoiding if you don’t do it,” “This is the only way to really recover,” and “If you do this work, it will get to the root of your problems and you won’t need substances anymore.” Even if well intentioned, these are not accurate for most patients with PTSD-SUD, especially those with severe SUD. For a notable clinical example of how exposure can worsen substance use see Morris (2015).
- *Attend to behavioral addictions as well as SUD.* Focus is increasing on behavioral addictions, such as excessive gambling, work, exercise, Internet, pornography, and sex (Najavits, Lung, Froias, Paull, & Bailey, 2014).
- *Choose PTSD-SUD models based on realistic factors.* Both the clinician and patient need models that fit for them. Factors such as preference for individual versus group work, past treatment experiences, appeal of various treatments, insurance coverage, and other factors, will play a role.

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