



Demographic and clinical characteristics of treatment seeking women with full and subthreshold PTSD and concurrent cannabis and cocaine use disorders

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ABSTRACT

While the detrimental effects of concurrent substance use disorders (SUDs) are now being well documented, very few studies have examined this comorbidity among women with posttraumatic stress disorder (PTSD). Data for these analyses were derived from the “Women and Trauma” study conducted within the National Drug Abuse Treatment Clinical Trials Network. Women with full or subthreshold PTSD and co-occurring cannabis use disorder (CUD) and cocaine use disorder (COD; $N = 99$) were compared to their counterparts with co-occurring CUD only ($N = 26$) and co-occurring COD only ($N = 161$) on rates of trauma exposure, psychiatric disorders, psychosocial problems, and other substance use utilizing a set of multivariate logistic regressions. In models adjusted for age and race/ethnicity, women with PTSD and COD only were significantly older than their counterparts with CUD only and concurrent CUD + COD. Relative to those with CUD only, women with concurrent CUD + COD had higher odds of adult sexual assault. Relative to those with COD only, women with concurrent CUD + COD had higher odds of alcohol use disorder in the past 12 months. Finally, relative to those with CUD only, women with COD only had higher odds of ever being arrested/convicted and adult sexual assault. The higher rates of adult sexual assault and alcohol use disorder among those with concurrent CUD + COD suggest the need for trauma-informed approaches that can respond to the needs of this dually-diagnosed population. Moreover, the causal link between repeated traumatic stress exposure and polysubstance use requires further examination.

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1. Introduction

The co-occurrence of posttraumatic stress disorder (PTSD) and substance use disorders (SUDs) is well established and associated with a host of complex clinical and public health challenges. The estimated prevalence rates of SUDs in persons with PTSD range from 34%–52% (Mills, Teesson, Ross, & Peters, 2006a; Vujanovic, Bonn-Miller, & Petry, 2016) while the rates of current PTSD in persons with SUDs range from 15%–42% (Pietrzak, Goldstein, Southwick, & Grant, 2011; Vujanovic et al., 2016), depending on the clinical population studied, with pregnant women and adolescents in substance abuse treatment

having the highest rates. These rates far surpass the rates of either PTSD or SUD alone among the general populations (Vujanovic et al., 2016).

A number of etiological models have been proposed to explain the co-occurrence of PTSD and SUDs including the “high risk hypothesis,” “self-medication hypothesis,” and “shared vulnerability” models (McCauley, Killeen, Gros, Brady, & Back, 2012; Ruglass, Lopez-Castro, Cheref, Papini, & Hien, 2014; van Dam, Ehrling, Vedel, & Emmelkamp, 2013). The self-medication hypothesis has received the most clinical and research attention and posits that individuals use substances as a way to manage painful affect states. Indeed, individuals with anxiety and/or PTSD frequently endorse affect regulation as a reason for their substance use (McCauley et al., 2012; van Dam et al., 2013). Nevertheless, other theoretical models are just as likely. For example, the high risk hypothesis posits that a substance-using lifestyle places the individual at high-risk for exposure to traumatic events and subsequent development of PTSD (McCauley et al., 2012; van Dam et al., 2013). Shared vulnerability models implicate common cognitive, affective,

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and neurobiological factors (e.g., attentional bias, emotion regulation difficulties, and dysfunction in the HPA axis) in the development of both PTSD and SUD and their associations. For example, research shows that adults with either PTSD or SUDs may exhibit deficits in emotion processing and self-regulation, thus there is growing appreciation for this perspective (Koenen, 2006; Sloboda, Glantz, & Tarter, 2012).

Given the complexity of the relationship between PTSD and substance use disorders due to differences in the psychoactive properties of various substances; the various meanings of substance use in the context of PTSD; and the significant number of bio-psycho-social factors that likely contribute to the use of particular substances by individuals with PTSD, a multifactorial model of understanding the association is necessary (McCauley et al., 2012; Ruglass, Hien, Hu, & Campbell, 2014a; van Dam et al., 2013).

Previous research examining the association between PTSD and SUDs has often focused on the global category of SUD, generally obscuring the differential relationship between PTSD and specific substances (Hien et al., 2009; Zlotnick, Johnson, & Najavits, 2009). Other studies have examined the relationship between PTSD and specific SUDs such as cannabis use disorder (CUD) (Bonn-Miller, Boden, Vujanovic, & Drescher, 2013; Kevorkian et al., 2015) or cocaine use disorder (COD), suggesting differential mechanisms of association. In the context of PTSD, cannabis use is often associated with reductions in traumatic memories and improvements in sleep disturbances (Boden, Babson, Vujanovic, Short, & Bonn-Miller, 2013; Cogle, Bonn-Miller, Vujanovic, Zvolensky, & Hawkins, 2011), whereas, cocaine is often linked with PTSD for its ability, in the short-term, to reduce trauma-related avoidance and numbing symptoms (S. Back et al., 2000; Back, Brady, Jaanimägi, & Jackson, 2006; Najavits et al., 2003). In both cases, however, and in the long-run, chronic substance use is often associated with a worsening of PTSD (Ruglass, Hien, Hu, & Campbell, 2014b). A growing body of literature suggests that a large proportion of those with PTSD are polysubstance users or have dual drug use disorders (Salgado, Quinlan, & Zlotnick, 2007; Schäfer & Najavits, 2007). Yet, very few studies have examined the differential impact of concurrent SUDs (i.e., more than one SUD simultaneously) in the context of PTSD, which may have important implications for treatment and recovery. The additive or interactive effect of concurrent substance use may have detrimental health and psychosocial effects. Existing studies that have examined concurrent substance use or use disorders either alone (Leri, Stewart, Tremblay, & Bruneau, 2004; Peters, Schwartz, Wang, O'Grady, & Blanco, 2014) or in the context of other psychiatric disorders indicate greater psychological and social problems, and poorer treatment outcomes among co-occurring substance users (Peters et al., 2014; Salgado et al., 2007).

In general, cannabis use has been found to be associated with a significant increase in use of a number of other substances, including cocaine, supporting the “gateway drug theory,” which posits that cannabis use typically precedes use of other illicit substances with greater addiction potential such as cocaine (Fergusson et al., 2006; Secades-Villa et al., 2015). Indeed, cocaine and cannabis co-use is a common type of polydrug use. Studies suggest up to 90% of individuals with COD co-use cannabis and up to 53% will have a concurrent CUD (Lindsay, Stotts, Green, Herin, & Schmitz, 2009; Miller, Klahr, Gold, Sweeney, & Cocores, 1990). Cannabis and cocaine may be co-used to increase the positive subjective effects of cocaine (Lukas, Sholar, Kouri, Fukuzako, & Mendelson, 1994) or they may be functionally independent in the same person (Budney, Higgins, & Wong, 1996). Chronic cannabis use has been shown to negatively affect vascular function, leading to higher anxiety and exacerbated stress-induced cravings for alcohol and cocaine (Fox, Tuit, & Sinha, 2013). Researchers have posited other variables that might contribute to the concurrent use of substances such as cannabis and cocaine including underlying genetic/personality vulnerabilities or environmental factors such as similar drug markets contributing to ease of access and availability (Agrawal, Budney, & Lynskey, 2012; Tzilos, Reddy, Caviness, Anderson, & Stein, 2014).

Regardless of the underlying mechanisms, concurrent cannabis and cocaine use is associated with more severe psychopathology compared with use of either drug alone (Lindsay et al., 2009). Studies indicate the interactive effects of combining both drugs can lead to more detrimental health consequences, including increased absorption of cocaine, increased heart rate and blood pressure, and heavier or more frequent drug use (Lindsay et al., 2009). Moreover, concurrent cannabis use has been associated with increased cocaine dependence, frequently leading to greater psychopathology and functional impairment, and poorer treatment outcomes (Lindsay et al., 2009).

Cannabis and cocaine use have been linked to a number of factors that may contribute to their common association with PTSD, including high rates of childhood and adult trauma, re-victimization, higher levels of posttraumatic symptom severity, social marginalization, poor mental and physical health, and criminal justice involvement (Saddichha, Werker, Schuetz, & Krausz, 2015). In one study examining a highly traumatized population, childhood sexual abuse was more strongly associated with adult use of cannabis and cocaine, as compared to heroin, alcohol, and tobacco, suggesting type of abuse may influence type of substance use (Khouri, Tang, Bradley, Cubells, & Ressler, 2010). This same study found that severity of cocaine use disorder was also strongly correlated with both severity of current PTSD symptoms and number of traumatic childhood exposures (Khouri et al., 2010). Crack cocaine use, in particular, has been associated with more severe psychopathology and PTSD symptom severity in addition to more significant substance abuse, including cannabis (Narvaez et al., 2014). The association between severity of cocaine use and PTSD symptom severity is thought to be driven by cocaine's exacerbation of hyperarousal symptoms of PTSD, while the use of cannabis in the context of severe PTSD symptoms is thought to be driven by perceived alleviation of negative affect to improve sleep or reduce hyperarousal symptoms (Bonn-Miller, Babson, & Vandrey, 2014; Bonn-Miller, Boden, Bucossi, & Babson, 2014). Individuals suffering from posttraumatic stress have endorsed using cannabis as a short-term emotion regulation strategy and those with high PTSD symptom scores were more likely to use cannabis for negative affect reduction (Bonn-Miller, Boden, et al., 2014; Bonn-Miller, Vujanovic, Feldner, Bernstein, & Zvolensky, 2007). Several studies of individuals with co-occurring PTSD and CUD further highlight the complexity of the relationship between these disorders. Specifically, the rate of PTSD was found to be higher among patients with a CUD diagnosis than those with other SUDs (Bonn-Miller, Harris, & Trafton, 2012), yet greater frequency of cannabis use was associated with PTSD symptom reduction, with one study finding a 75% reduction in PTSD symptoms associated with cannabis use (Bonn-Miller, Vujanovic, Boden, & Gross, 2011; Greer, Grob, & Halberstadt, 2014). Overall, findings give a mixed picture of the impact of co-occurring CUD/COD, suggesting in some cases an amelioration of PTSD symptoms, and in others, an escalation. And few studies have explored concurrent CUD + COD and associated consequences despite high rates of polysubstance use and use disorders among those with PTSD ((Salgado et al., 2007).

Thus, more research is needed to better understand the factors associated with concurrent CUD/COD among individuals with PTSD. The present study characterizes the psychiatric and psychosocial correlates of concurrent CUD + COD at baseline in a large, multi-site, randomized clinical trial (“The Women and Trauma Study”) examining treatments for women with co-occurring PTSD and SUD (Hien et al., 2009). We hypothesized that trauma exposure, psychiatric disorders, psychosocial problems and other substance use would be more prevalent or severe in women with PTSD (full or subthreshold) and concurrent CUD + COD relative to their counterparts with CUD only, and relative to their counterparts with COD only. This hypothesis is based on previous research showing more psychosocial problems and psychiatric symptoms among those who co-use substances or have poly-drug use disorders compared to those with only one SUD (Ullman, Townsend, Starzynski, & Long, 2006).

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