A MODEL OF POST-TRAUMATIC SYMPTOMS, NEGATIVE AFFECT, COPING USE MOTIVATIONS, SUBSTANCE USE, AND SUBSTANCE USE CONSEQUENCES IN INCARCERATED INMATES

by

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Thesis directed by Assistant Professor Andrew Lac

ABSTRACT

This study applies frameworks of the self-medication hypothesis and social cognitive theory to investigate pathways from post-traumatic stress disorder (PTSD) symptoms to substance use consequences (as mediated by negative affect, coping use motivations, and substance use) in incarcerated males. Data from 397 incarcerated male inmates were drawn from a recently collected dataset. Participants were administered the Post-Traumatic Stress Disorder Checklist and Adult Substance Use Survey Revised to assess symptoms of PTSD, negative affect, coping use motivations, substance use, and psychophysiological and behavioral consequences of use. Confirmatory factor analysis described interfactor correlations and was utilized to eliminate one poor fitting item determining that all other items significantly loaded onto their corresponding factors. A hypothesized structural equation model tested pathways starting from PTSD symptoms and ending at each psychophysiological and behavioral consequences. In the final model containing only significant pathways, PTSD symptoms directly explained higher negative affect ($\beta = .66, p < .001$) and coping use motivations ($\beta = .44, p < .001$). Negative affect explained higher psychophysiological consequences ($\beta = .37, p < .001$), whereas coping use motivations contributed to higher substance use ($\beta = .30, p < .001$) as well as higher behavioral ($\beta = .21, p < .001$) consequences. Substance use explained higher psychophysiological consequences ($\beta = .35, p < .001$) and higher behavioral
consequences ($\beta = .39, p < .001$). Tests of indirect effects supported that pathways from PTSD symptoms to substance use outcomes were mediated through negative affect, coping motivations, and substance use. Findings highlight negative affect and coping use motivations as fully mediating pathways from PTSD to substance use outcomes providing useful treatment targets for incarcerated male inmates.
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CHAPTER I

INTRODUCTION

Both post-traumatic stress disorder (PTSD) and substance use contribute to the United States maintaining the highest incarceration rate in the world (Al-Rousan, Rubenstei, Sieleni, Deol, & Wallace, 2017) with approximately 2,255,000 inmates residing in United States jails or prisons at any given time (Kaeble, Glaze, Tsoutis, & Minton, 2016) and each incarcerated inmate costing taxpayers an average of $28,983.40 per year (Davis, 2014). Approximately 46% of all inmates are incarcerated for drug related offenses, more than any other crime (Federal Bureau of Prisons, 2018). PTSD has also been linked to criminality (Sadeh, & McNiel, 2015) and is overrepresented in incarcerated populations (Gosein, Stiffler, Frascoia, & Ford, 2016). Approximately 48% of inmates satisfy PTSD diagnostic criteria compared to 4% in the general population (Briere, Agee, & Dietrich, 2016). Furthermore, six or more traumatic event exposures anticipate a 64% likelihood of developing PTSD in inmates compared to 12% in the general population (Briere et al., 2016). Male inmates are likely to experience multiple traumas prior to incarceration (Sindicich et al., 2014) and additional traumas during incarceration (Schneider, et al., 2011). PTSD symptoms are implicated in the development of detrimental outcomes including substance use (Bennett, Peer, & Gjonbalaj-Marovic, 2014), criminal behavior (Reid, 2015), and incarceration (Fazel, Bains, & Doll, 2006).
Consequences of PTSD symptoms frequently overlap with consequences of substance use (Alsawy, Wood, Taylor, & Morrison, 2015; Conner, Bossarte, Arora, & Katz, 2014; Freeman et al., 2013; Novaco & Chemtob, 2015; Wrocklage et al., 2016) including behavioral consequences and consequences that produce both psychological and physical outcomes (Diercks, et al., 2008, Grant et al., 2016; White, Loeber, Stouthamer-Loeber, & Farrington, 1999). A financial burden estimated at $520.5 billion per year results from consequences of psychoactive substance use in the United States (National Institute on Drug Abuse, 2017). The current research was designed to test pathways and processes from PTSD symptoms to psychophysiological and behavioral consequences as multivariately mediated through negative affect, coping motives, and substance use in imprisoned criminal offenders.

**Post-Traumatic Stress Disorder**

PTSD is an anxiety problem resulting from exposure to traumatic events and characterized by symptom clusters including intrusive recollections, avoidance, cognitive and mood symptoms, and arousal and reactive symptoms (Sanchez, & Brownlee-Duffeck, 2018). PTSD symptoms can develop from direct or indirect exposure to traumatic events including physical and sexual assault, combat, exposure to a natural disaster, or witnessing death (Boccia, D’Amico, Bianchini, Marano, Giannini, & Picardi, 2016). Inmates tend to be over exposed to especially impactful forms of interpersonal trauma (Badour, Resnick, & Kilpatrick, 2017), exemplified in incarcerated men who report direct exposure to violence, seeing someone else injured or killed, and the death of someone close as the most frequently experienced traumas (Wolff, Huening, Shi, & Frueh, 2014). Development of PTSD is marked by systematic neurobiological
changes including reduction in hippocampal (Bremmer et al., 1995) and ventromedial
prefrontal cortex (Kasai et al., 2008) volumes. These changes are implicated in cognitive
difficulties related to accurate threat assessment, generalization of trauma cues, and
extinguishing of fear learning (Boccia, D’Amico, Bianchini, Marano, Giannini, &
Piccardi, 2016).

**Negative Affect**

Compared to those without PTSD, individuals with PTSD are more likely to use
psychoactive substances to cope with negative emotions (Leeies, Pagura, Sareen &
Bolton, 2010). Negative affect is an emotional construct (Watson & Clark, 1999)
capturing general subjective distress via emotions including anger, anxiety, depression
(Watson, 1988), nervousness, and worry (Waller, Tellegen, McDonald, & Lykken, 1996).
Individuals high in negative affectivity are discontented with their environmental
experiences (DeLisi & Vaughn, 2014) and demonstrate more externalizing problem
behaviors (Volkow et al., 2014) including aggressive and violent acts (Ousey, Wilcox, &
Schreck, 2015). Negative affectivity is also a risk for criminal behavior (Garfalo, &
Velotti, 2017) and leads to higher rates of criminal recidivism (Wolf & Baglivio, 2017).
Negative emotional experiences as assessed through mood disorders are highly prevalent
in prison inmates (Chiles, Von Cleve, Jemelka, & Trupin, 1990; Gunter et al., 2008).

**Coping Use Motivations**

Coping use motivations represent a cognitive link between symptoms of PTSD
and substance use outcomes. Substance use motivations are considered a primary
cognitive predictor of substance use behaviors because individuals are motivated to use
psychoactive substances to fulfill different underlying needs (Cooper, Kuntche, Levitt,
Barber & Wolf, 2016). Coping use motivations represent “internally generated negative reinforcement motives” to use substances in an effort to “relieve distressing internal experiences” (Cooper, 1994, p.118). Coping motivations have been linked to greater substance use quantity and frequency (Anderson, Sitney, & White, 2015; Holahan et al., 2003; Windle, 2000) and higher levels of substance use consequences (Blevins, Abrantes, & Stephens, 2016).

**Substance Use Consequences**

Substance use is related to many harmful consequences including compromised physical health, infectious diseases, loss of productivity, compromised quality of life, crime and violence, abuse and neglect, health care costs, and motor vehicle accidents (World Health Organization, & World Health Organization Management of Substance Abuse Unit, 2014). Substance use outcomes can be broadly classified into psychophysiological and behavioral consequences. Psychophysiological consequences include intoxication or withdrawal symptoms such as mental confusion, physical shakes, and tachycardia whereas behavioral consequences include symptoms that harm self or others including passing out, stumbling, violent behaviors, and blackouts (Wanberg, 2015). Consequences are more likely to occur from polysubstance use (Bennett & Holloway, 2005) and many psychoactive substances including those consumed legally and illicitly (Boden, Fergusson, & Horwood, 2013; Bradford & Payne, 2012; Gizzi & Gerkin, 2010) are independently related to psychophysiological (Mirijello, et al., 2015; Moeller, Huttner, Struffert, & Muller, 2016; Swift & Stout, 1992) and behavioral (Boles & Miotto, 2003) consequences.
Interrelations

**PTSD and negative affect.** Brain regions such as the hippocampus, dorsal anterior cingulate cortex, rostral anterior cingulate cortex, amygdala, and insula are involved in regulating emotional processes and implicated in the development of PTSD (Garfinkel & Liberzon, 2009). These brain regions are aroused through exposure to emotionally aversive stimuli (Shin et al., 1997) and specifically associated with negative emotionality (Etkin, Egner, & Kalisch, 2011; Hamann, Ely, Hoffman, & Kits, 2002). Such regions are consistently hyper-aroused in individuals diagnosed with PTSD (VanElzakker, Staples-Bradley, & Shin, 2018; Vujanovic, Bonn-Miller, & Petry, 2016), suggesting a neurological connection between PTSD and negative emotionality (Badour, Resnick, & Kilpatrick, 2017). Common PTSD-related emotions including anger (Olatunji, Ciesilski, & Tolin, 2010), shame and guilt (Wilson, Drozdek, & Turkovic, 2006), and self-disgust (Badour & Adams, 2015) often facilitate negative beliefs about the self and the world (Foa, Ehlers, Clark, Tolin, & Orsillo, 1999). Decreased ability to tolerate such emotions predicts both increased PTSD symptoms and coping through substance use (Banducci, Bujarski, Bonn-Miller, Patel, & Connolly, 2016). The intimate relation between PTSD and negative emotionality has prompted researchers to include negative affect as a Diagnostic and Statistical Manual of Mental Health Disorders - Fifth Edition (DSM-5) PTSD symptom by including additional items such as “having strong negative beliefs about yourself, other people, or the world” (Badour, Resnick, & Kilpatrick, 2017).

In a previous structural equation investigation conducted by the National Center for PTSD Miller, Vogt, Mozley, Kaloupek, and Keane (2006) recruited a sample of 1,002
Vietnam veterans to demonstrate the mediating self-medicative role of negative emotionality. Negative emotions including anger, worry, anxiety, and regret were captured using the PSY-5 scale and represented as a latent factor. The negative emotionality construct was found to mediate the effects of PTSD symptoms on alcohol problems. In a second study, Badour, Resnick, and Kilpatrick (2017) using a nationally representative sample of 1522 interpersonal trauma survivors found that 83% of individuals with a history of assault related PTSD (AR-PTSD) endorsed persistent negative affect compared to 8% without AR-PTSD. AR-PTSD is characterized by interpersonal trauma exposures frequently endorsed by male inmates including physical assault or witnessing violence (Wolff, Huening, Shi, & Frueh, 2014). Taken together, results support the self-medicative role of negative affect by demonstrating its mediational function between PTSD symptomology and substance use outcomes and emphasizing how unique trauma exposures and expressions of PTSD common to inmates can increase negative emotional states.

**PTSD and coping use motivations.** Individuals with PTSD symptoms may be at greater risk for using psychoactive substances because of increased motivation to manage undesirable internal experiences (Back, Brady, Jaanimägi, & Jackson, 2006; Najavits, 2004) that yield increased avoidance strategies (Plumb, Orsillo, & Luterek, 2004) as postulated in the self-medication hypothesis (Khantzian, 1997). The self-medication hypothesis proposes that the anxiety-reducing properties of psychoactive substances are utilized to manage the anxiety provoking symptoms of PTSD (Conrod & Stewart, 2003). The assessment of coping use motivations is a common approach to determine the extent that individuals consume psychoactive substances to reduce and regulate stressful
experiences (Windle & Windle, 2015) and coping use motivations are significantly predicted by PTSD symptoms (Potter, Vujanovic, Marshall-Berenz, Bernstein, & Bonn-Miller, 2011). Coping use motivations have contributed to explaining the pathway from PTSD symptoms to substance use through moderation and mediational analyses (Bonn-Miller, Vujanovic, Feldner, Bernstein, & Zvolensky, 2007; O’Hare & Sherrer, 2011; Potter, Vujanovic, Marshall-Berenz, Bernstein, & Bonn-Miller, 2011; Simpson, Stappenbeck, Luterek, Lehavot, & Kaysen, 2014).

Several studies have highlighted the strength of the PTSD coping use motives connection. Potter, Vujanovic, Marshall-Berenz, Bernstein, & Bonn-Miller (2011) in a sample of 142 adult men and women discovered a significant positive incremental association between PTSD symptoms and marijuana coping use motives. Simpson, Stappenbeck, Luterek, Lehavot, & Kaysen (2014) using a sample of 86 male and female participants with co-morbid PTSD and alcohol use disorder determined that as PTSD symptoms increased, drinking also increased for those with higher coping motives. Other types of use motivations including social motives and conformity motives failed to interact with PTSD to increase drinking in the same study. Finally, O’Hare and Sherrer (2011) in a study of 116 adult men and women with severe mental illness discovered that drinking to cope fully mediated the pathway from PTSD symptoms to alcohol quantity and frequency measures.

PTSD and substance use. PTSD is related to substance use (Cougle, Bonn-Miller, Vujanovic, Zvolensky, & Hawkins, 2011; Jacobson, Southwick, & Kosten, 2001; Perkonigg, Kessler, Storz, & Wittchen, 2000), such that an established diagnosis of PTSD precipitates a four times greater chance of developing a substance use disorder.
PTSD symptoms and substance use tend to be comorbid (Perkonigg, Kessler, Storz, Wittchen, 2000; Roberts, Roberts, Jones, & Bisson, 2015) with both contributing to lower general functioning and poorer treatment outcomes (Schäfer & Najavits, 2007). In inmate samples, PTSD and substance abuse are the most common Axis I conditions (Butler, Andrews, Allnut, Sakashita, Smith, & Basson, 2006; Simpson, et al., 2007; Smith & Trimboli, 2010) and those diagnosed with both conditions usually report more extensive psychopathology (Sindicich et al., 2014).

The literature asserts that PTSD symptoms should increase the propensity for substance use. After the September 11th terrorist attack, residents of Manhattan were found to have increased consumption of substances including alcohol and marijuana (Vlahov, et al., 2002). Post-traumatic symptoms are independently linked to greater alcohol and cannabis consumption (Hien, et al., 2015; Kevorkian et al., 2015; Simpson, Stappenbeck, Luterek, Lehavot, & Kaysen, 2014), but have also been found to decrease alcohol consumption following PTSD diagnosis (McFarlane, 1998). Higher levels of PTSD symptoms also predict increased opioid (Hassan, Le Foll, Imtiaz, & Rehm, 2017), benzodiazepine (Vasiliadis, Lamoureux-Lamarche, & Previle 2016) and stimulant use (Back, Brady, Jaanimagi, & Jackson, 2006). Furthermore, treatment interventions designed to decrease PTSD symptom severity also managed to subsequently decrease illegal substance consumption (Rugless, Hien, Hu, & Campbell, 2014).

**PTSD and substance use consequences.** Inmates with comorbid PTSD symptoms and substance use demonstrate a complicated medley of symptoms including impulsivity, aggression, hypervigilance, and anger that can increase the likelihood of violent behavior (Barrett, Mills, & Teeson, 2011). Such individuals experience more
psychophysiological consequences manifest through cardiovascular issues and bodily pain (Ouimette, Goodwin, & Brown, 2006) and behavioral consequences including increased impulsivity (Cohen & Hien, 2006), self-injurious behavior (Evren & Evren, 2005) and illegal acts (McCauley et al., 2012). PTSD symptoms are also independently related to psychophysiological and behavioral consequences attributed to substance use including violence (Novaco & Chemtob, 2015), psychosis (Alsawy, Wood, Taylor, & Morrison, 2015), paranoia (Freeman et al., 2013), sleep disturbances (Lamarche & De Koninck, 2007), and seizures (Myers, Perrine, Lancman, Flemming & Lancman, 2013).

**Negative affect and substance use.** Negative affect is related to increased substance consumption (Elkins et al., 2006; Sher et al., 2005; Wills, Sandy, Shinar, & Yaeger, 1999), an association understood neurobiologically through lower levels of extracellular dopamine that increase the risk for usage (Volkow et al., 2014). Longitudinally, negative affect is associated with subsequently higher substance use, whereas positive affect is associated with lower use (Wills, Sandy, Shinar, & Yaeger, 1999). Negative emotionality is a risk factor for the use of multiple types of psychoactive substances (Hicks et al., 2012; Sherman, Zinser, Sideroff, & Baker, 1989; Sher et al., 2005; Wheeler, Twining, Jones, Slater, Grigson, & Carelli, 2008) as measured by negative emotionality constructs including depression (Rounsaville, 2004) and anger (Goldstein et al., 2005).

**Negative affect and substance use consequences.** Negative affectivity and substance use foster similar consequences (Curhan et al., 2014; Shorey, McNulty, Moore, & Stuart, 2015; Sanchez-Gonzalez, May, Koutnik, & Fincham, 2015). Furthermore, negative affectivity is indirectly (through increased consumption; Martens, et al., 2008)
and directly (Park & Grant, 2005) related to greater substance use consequences. Miller, Vogt, Mozley, Kaloupek, and Keane (2006) determined that the pathway from PTSD symptoms to substance use consequences was mediated by negative emotionality in a structural equation model.

**Coping use motivations and consequences.** Substance use motivation is a cognitive factor that contributes to substance use and substance use consequences (Maisto, Carey, & Bradizza, 1999). In one investigation comparing polysubstance users to groups who used only alcohol or an illicit drug, polysubstance users had higher tension reduction motives and used substances to cope more than single substance using groups (Ullman, Townsend, Starzynski, & Long, 2006). In other research, negative motives for drinking were associated with heavier drinking and more drinking related complications (Jessor et al., 1968) and only coping motivations (as analyzed alongside enhancement, social, and conformity motives) directly and indirectly predicted usage problems (Cooper, 1994). This significant direct pathway from coping motives to substance use consequences, while controlling for both alcohol and cannabis, has been corroborated across several investigations (Carey & Correia 1997; Merrill, Wardell, & Read, 2014; Simons, Coreia, Carey, & Borsari 1998).

**Theoretical Frameworks**

**Self-medication hypothesis.** The self-medication hypothesis supports the view that those who experience PTSD symptoms are more likely to use substances in response to negative emotions (Waldrop, Back, Verduin & Brady, 2007), because the effects of psychoactive substances help to temporarily relieve or change negative affect (Khantzian, 1997; Suh, Ruffins, Robins, Albanese, & Khantzian, 2008). Longitudinal data suggest
that alcohol and drug problems increase approximately 10% for every additionally experienced PTSD symptom (Haller & Chassin, 2014), and national epidemiological data conclude that about 21.4% of non-institutionalized civilian respondents with PTSD self-medicate with alcohol or drugs to relieve PTSD symptoms (Leeies, Pagura, Sareen, & Bolton, 2010). The self-medication hypothesis supports the temporal structure of the proposed current research by asserting that post traumatic symptomology cultivates negative internal experiences resulting in a desire to self-medicate (Conrod & Stewart, 2003).

Alternative explanations of the PTSD substance use link include the high-risk hypothesis, the shared vulnerability hypothesis, and the susceptibility hypothesis. The high-risk hypothesis posits that drug use is a risky behavior because activities such as buying and selling drugs increase potential exposure to traumatic events (Windle, 1994). The shared vulnerability hypothesis argues that through an unknown common neurochemical mechanism, individuals at risk for PTSD are also at risk for addiction to psychoactive substances (Stewart & Conrod, 2003). The susceptibility hypothesis suggests that substance use can influence the development of PTSD by interfering with essential emotional regulatory mechanisms or increasing arousal and reactivity (Stewart, Pihl, Conrod, & Dongier, 1998). Currently, the self-medication hypothesis is the most researched and supported explanation of post-traumatic stress and substance use comorbidity (Chilcoat & Breslau, 1998; Haller & Chassin, 2014).

Social cognitive theory. Social cognitive theory posits that behavior, cognitions, and other personal factors interact reciprocally to influence learning (Bandura, 2011; Maisto, Carey, & Bradizza, 1999). Coping use motives can predict substance use
consequences beyond substance use behavior (Merrill, Wardell, Read, 2014) because of a failure in adaptive learning. Those with higher coping motivations have learned from past experiences that substance use is effective in temporarily managing and reducing their internal emotional problems. Myopically attending to substance use as a problem-solving strategy interferes with the learning of healthier adaptive coping strategies (Cooper, Frone, Russell, & Mudar, 1995) and impedes problem-solving behaviors (Bandura & Walters, 1977) leading to increased consequences.

**Current Study**

The proposed investigation will test pathways from PTSD symptoms to negative affect and coping motivations to substance use to psychophysiological and behavioral consequences in incarcerated males diagnosed with a substance use disorder. This study, informed by the self-medication hypothesis and social learning theory, is expected to contribute to the literature in several ways. First, PTSD symptoms are documented to be related to higher levels of negative affect (DiMauro, Renshaw, & Kashdan, 2016), coping use motivations (Berenz, Kevorkian, Chowdhury, Dick, Kendler, & Amstadter, 2016), and substance use (Cross, Crow, Powers, & Bradley, 2015). Although PTSD symptoms could facilitate consequences of substance use (Ramchand, Rudavsky, Grant, Tanielian, & Jaycox, 2015), previous research has rarely tested mediating variables and processes that explain this connection. Second, much of the literature has focused on connections between PTSD and substance use in veteran (Possemato et al., 2015) and college student samples (Bachrach & Read, 2017), with fewer studies examining these interrelations in incarcerated prisoners (Howard, Karatzias, Power, & Mahoney, 2017). A search of the
literature failed to disclose any study that tested associations from PTSD to substance use motivations and consequences in an incarcerated sample.

Third, previous studies have tested coping use motivations (Cooper, 1994) and negative affect (Brandon, 1994; Stasiewicz & Maisto, 1993), but rarely control for both constructs in the same statistical model. Simultaneous analysis of both constructs is of relevance because negative affect represents the emotional aftermath and coping use motivations represent the cognitive aftermath of PTSD symptoms. Martens, Neighbors, Lewis, Lee, Oster-Aaland, and Larimer (2008) examined negative affect and coping use motivations as moderators of alcohol use problems, but no study has simultaneously examined both constructs as mediating the effects of PTSD symptoms on substance use consequences.

Fourth, this study intends to examine the extent that PTSD symptoms, negative affect, and coping use motivations are related to the number of different psychoactive substances used prior to incarceration and the extent that polysubstance use is incrementally related to substance use consequences. Independently, many psychoactive substances have been associated with PTSD symptoms and use motivations (Bonn-Miller, Vujanovic, Feldner, Bernstein, & Zvolensky, 2007; Lehavot, Stappenbeck, Luterek, Kaysen, & Simpson, 2014) using quantity and frequency measures. Examining the total number of substances used prior to incarceration is of practical and theoretical relevance for inmates because polysubstance consumption has been associated with increased consequences (Connor, Gullo, White, & Kelly, 2014) and incarceration risk (Morley, Lynskey, Moran, Borschmann, & Winstock, 2015) but is rarely examined as an
outcome of PTSD symptoms, negative affect, and coping use motivations or as a predictor of substance use consequences.

A structural equation model will be tested in the proposed research. The model asserts that pathways starting from PTSD symptoms and ending in psychophysical and behavioral consequences will be statistically mediated by negative affect, coping use motivations, and substance use. In other words, PTSD symptoms are hypothesized to predict greater negative affect, greater coping use motivations, greater substance use, and greater psychophysiological and behavioral use consequences. Furthermore, negative affect and coping use motivations will be specified to explain substance use, psychophysiological consequences, and behavioral consequences. Finally, substance use will be designated to predict psychophysiological and behavioral use consequences.
CHAPTER II

METHOD

Participants

Following listwise deletion, the incarcerated male sample (N = 397) ranged from 18 to 62 years of age (M = 35.64, SD = 11.81) and was recruited from the El Paso County Colorado Sheriff’s Department between the years of 2015 and 2017. Racial composition was 55% White, 22% Hispanic, 18% Black, 3% Native American, 1% Asian/Pacific Islander. The sample consisted of 13% veterans and 15% who lived transiently prior to their assessment.

Procedure

Participants were recruited and administered assessments approximately two weeks following incarceration as part of the Reintegration and Recovery (R&R; a substance abuse and mental health treatment program for criminal offenders) program clinical intake process. Permissions were obtained from the R&R program and the El Paso County Colorado Sheriff’s Department prior to data collection. An institutional IRB approved the research protocols (IRB# 17-174).

Measures

Post-Traumatic Stress Disorder Checklist. The Post-Traumatic Stress Disorder Checklist (PCL) is a self-report scale consisting of 17 items based on DSM-IV-TR diagnostic criteria. Scores on the PCL range from 17 to 85 with higher scores indicating greater symptom severity (Cusack, Herring & Steadman, 2013). The PCL has
demonstrated acceptable internal consistency, test retest reliability, and convergent validity in both veteran and civilian samples (Ruggiero, Ben, Scotti, & Rabalais, 2003; Weathers, Herman, Huska & Keane, 1993) and is considered appropriate for assessing PTSD in criminal offenders (Cusack, Herring & Steadman, 2013). The PCL has exhibited one, two, three, and four factor structures across varying demographic groups (Conybeare, Behar, Solomon, Newman, & Borkovec, 2012) with most evidence supporting a four-factor solution (Elklit & Shevlin, 2007). The PTSD construct, however, is commonly examined in structural equation modeling as a single latent factor represented by indicators from each of the four DSM-5 symptom clusters (Marshall, Miles, & Stewart, 2010; Miller, Vogt, Mozley, Kaloupek, and Keane, 2006).

Respondents indicated how much they have experienced symptoms in the past month using response anchors ranging from 1 (not at all), 2 (a little bit), 3 (moderately), 4 (quite a bit) and 5 (extremely).

**Adult Substance Use Survey Revised.** The Adult Substance Use Survey Revised (ASUS-R) is part of the Standardized Offender Assessment Package designed to assess substance use, substance use outcomes, and risk factors in criminal offenders (Van Voorhis & Salisbury, 2013; Wanberg, 2015). The ASUS-R has demonstrated desirable criterion, concurrent, and predictive validity, in addition to optimal internal consistency reliability based on Cronbach’s alpha and Principal Component Analysis (Wanberg, 2015).

**Negative Affect.** A 9-item ASUS-R subscale measured negative affect independently of substance use. The scale gathers information about emotional problems including depression, worry, anxiety, and anger (e.g., “Have you felt down and
Participants endorsed items with four response choices ranging from 1 (no), 2 (yes sometimes), 3 (yes a lot), and 4 (yes, all the time).

**Coping Motivations.** The ASUS-R measures cognitive motivations to use psychoactive substances to manage and reduce stress, forget problems, and change undesirable emotions (e.g., “To feel less tense or stressed”). Coping motivations were assessed with ten items with response choices ranging from 1 (no), 2 (sometimes), 3 (often) and 4 (very often).

**Substance Use.** The ASUS-R measures different psychoactive substances used in the past six months including alcohol, marijuana, cocaine, amphetamines, hallucinogens, inhalants, heroin, prescription opiates, barbiturates, and tranquilizers. Participants indicated if they had used each substance in the past six months. A total substance use score was compiled from binary responses by summing each substance used in the past six months with scores ranging from 0 to 10.

**Psychophysiological Consequences.** Psychologically and physically disruptive outcomes of substance use were captured through a series of 10 ASUS-R items assessing consequences due to intoxication or withdrawal that clients had previously experienced in the past six months. Participants rated the frequency of problems (e.g., hallucinations, confusion, or rapid or fast heartbeat) attributed to alcohol or drug use. Items were assessed with response anchors including 1 (never), 2 (1-3 times), 3 (4-6 times), 4 (7-10 times), and 5 (more than 10 times).

**Behavioral Consequences.** Behavioral disruptions attributed to substance use were captured through a series of six ASUS-R items indicating potential harm to self or others. Participants rated the frequency of problems (e.g., violence, blackout, sickness,
and overusing) stemming from alcohol or drug use that they had experience in the past six months. Behavioral disruptions were assessed with response anchors ranging from 1 (*never*), 2 (*1-3 times*), 3 (*4-6 times*), 4 (*7-10 times*), and 5 (*more than 10 times*).

**Analytic Plan**

The confirmatory factor analysis and structural equation models were estimated using IBM SPSS AMOS. Listwise deletion was performed prior to analysis to remove 14 participants with data assumed to be missing randomly (Allison, 2003). Skewness of measured variables ranged from 0.46 to 4.23 whereas kurtosis ranged from -0.58 to 20.65. Bootstrapping with bias corrected confidence intervals and 2000 bootstrap samples was applied to correct departures from normality and evaluate direct and indirect effects (Byrne, 2010).

Parceling was implemented to efficiently aggregate many items and to maintain the statistical advantages of latent factors (Little, Cunningham, Shahar, & Widaman, 2002; Matsunaga, 2008). The PTSD factor ($\alpha = .94$) was parceled into four symptom clusters as recommended in prior research applying confirmatory factor analysis (Elklit & Shevlin, 2007) and structural equation modeling (Miller, Vogt, Mozley, Kaloupek, and Keane, 2006). Substance use ($\alpha = .75$) was represented by a measured total composite of each substance used in the past six months. The latent factors of negative affect ($\alpha = .92$), coping use motivations ($\alpha = .94$), and psychophysiological consequences ($\alpha = .92$) were represented by parcels of their measured items. Parceling was not required to estimate behavioral consequences ($\alpha = .81$) represented by five measured indicators.

Recommendations were followed to perform a confirmatory factor analysis to evaluate item loadings and interfactor correlations prior to estimating the structural
equation model (Anderson & Gerbing, 1988). In the initial and final confirmatory factor analyses (Figures 1 & 2), latent factors involving PTSD symptoms, negative affect, coping use motivations, substance use, psychophysiological consequences and behavioral consequences were permitted to be intercorrelated. In the hypothesized model (Figure 3), PTSD was specified to predict negative affect, coping use motivations, substance use, psychophysiological consequences and behavioral consequences. Furthermore, negative affect and coping use motivations were allowed to explain substance use in addition to both consequence factors. Substance use was set to predict psychophysiological and behavioral consequences. Both consequence outcomes were covaried because items were derived from the same scale and assumed to be correlated. Non-significant paths were trimmed to re-estimate the final model (Figure 4). All tests were evaluated at a more conservative $p < .01$ (two-tailed).

Multiple converging fit indices were utilized to determine model fit. The model chi-squared test assesses overall discrepancy between the hypothesized model and the underlying data. A non-significant model chi-squared test is desirable, but, chi-squared tests can be sensitive to sample size (Bollen, 1989). CFI and TLI values range from 0.00 to 1.00 with higher values indicating more desirable fit (Ullman, 2006; Ullman & Bentler, 2003). The RMSEA index is evaluated from 0.00 to 0.05 (close fit), 0.05 to 0.08 (fair fit), 0.08 to 0.10 (mediocre fit), and greater than 0.10 (poor fit) and provides confidence intervals (MacCallum, Browne, & Sugawara, 1996).
Figure 1. Confirmatory factor analysis involving PTSD symptoms, negative affect, coping motivations, substance use, psychophysiological consequences, and behavioral consequences in criminal offenders. All factor loadings are significant at $p < .001$. All interfactor correlations (except for correlations between PTSD symptoms and substance use and PTSD symptoms and behavioral consequences) use are significant at $p < .01$. e = error.
Figure 2. Confirmatory factor analysis involving PTSD symptoms, negative affect, coping motivations, substance use, psychophysiological consequences, and behavioral consequences in criminal offenders. One item has been deleted (V20). All factor loadings are significant at $p < .001$. All interfactor correlations (except for correlations between PTSD symptoms and substance use and PTSD symptoms and behavioral consequences) use are significant at $p < .01$. $e =$ error.
Figure 3. Hypothesized structural equation model of pathways involving PTSD symptoms, negative affect, coping motivations, substance use, psychophysiological consequences, and behavioral consequences in criminal offenders. e = error. d = disturbance.
Figure 4. Final structural equation model of pathways involving PTSD symptoms, negative affect, coping motivations, substance use, psychophysiological consequences, and behavioral consequences in criminal offenders. All displayed paths are significant at $p < .001$. e = error. d = disturbance.
CHAPTER III
RESULTS

Descriptives

According to the Life Events Checklist, 89% of participants were exposed to a “happened to me” traumatic event prior to their incarceration reporting an average of 4.35 different “happened to me” traumatic event exposures per inmate. At the time of assessment, mean Post-Traumatic Stress Disorder Checklist (PCL) scores were 35.41 with a standard deviation of 17.72. Participants reported varying levels of substance use in the past 6 months for “non-medical reasons” such that 64% reported using alcohol, 53% reported using marijuana, 15% reported cocaine use, 37% reported amphetamine use, 10% reported using hallucinogens, 2% reported using inhalants, 13% reported using heroin, 14% reported using other opiates, 4% reported using barbiturates, and 6% reported use of tranquilizers.

Confirmatory Factor Analysis

Confirmatory factor analysis was performed to evaluate the factor structure of the latent constructs (Figure 1). The measurement model demonstrated acceptable fit, $\chi^2 = 467.99, df = 156, p < .001, \chi^2/df = 3.00, \text{CFI} = .95, \text{TLI} = .94, \text{RMSEA} = .07$ [90% CI: 0.06 to 0.08]. All factor loadings emerged as significant ($p < .001$) All constructs were significantly intercorrelated ($p < .01$) except for PTSD symptoms and substance use $r = .02, p < .61$ and PTSD symptoms and behavioral consequences $r = .14, p < .02$. 
A variable (V20) was removed from behavioral consequences for demonstrating a multiple r^2 value below .20 according to recommendations for structural equation modeling (Hooper, Daire, Coughlan, Joseph, and Mullen, 2008). After deletion of this item, the confirmatory factor analysis was reestimated. The reestimated confirmatory factor analysis demonstrated desirable fit $\chi^2 = 403.15$, $df = 138$, $p < .001$, $\chi^2/df = 2.92$, CFI = .96, TLI = .95, RMSEA = .07 [90% CI: 0.06 to 0.08] and is presented in Figure 2. Only interfactor correlations between PTSD symptoms and substance use $r = .02$, $p < .61$ and PTSD symptoms and behavioral consequences $r = .14$, $p < .02$ maintained non-significance.

Table 1

*Descriptive Statistics of Measured Variables*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean</th>
<th>Standard Deviation</th>
<th>Skewness</th>
<th>Kurtosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>V1</td>
<td>.00</td>
<td>4.00</td>
<td>1.10</td>
<td>1.10</td>
<td>.82</td>
<td>-.41</td>
</tr>
<tr>
<td>V2</td>
<td>.00</td>
<td>4.00</td>
<td>1.19</td>
<td>1.28</td>
<td>.82</td>
<td>-.58</td>
</tr>
<tr>
<td>V3</td>
<td>.00</td>
<td>4.00</td>
<td>1.05</td>
<td>1.05</td>
<td>.99</td>
<td>.08</td>
</tr>
<tr>
<td>V4</td>
<td>.00</td>
<td>4.00</td>
<td>1.09</td>
<td>1.27</td>
<td>.93</td>
<td>-.38</td>
</tr>
<tr>
<td>V5</td>
<td>.00</td>
<td>3.00</td>
<td>.81</td>
<td>.59</td>
<td>.81</td>
<td>.46</td>
</tr>
<tr>
<td>V6</td>
<td>.00</td>
<td>3.00</td>
<td>1.18</td>
<td>.78</td>
<td>.46</td>
<td>-.45</td>
</tr>
<tr>
<td>V7</td>
<td>.00</td>
<td>3.00</td>
<td>.89</td>
<td>.68</td>
<td>.75</td>
<td>.18</td>
</tr>
<tr>
<td>V8</td>
<td>.00</td>
<td>3.00</td>
<td>1.13</td>
<td>.87</td>
<td>.61</td>
<td>-.56</td>
</tr>
<tr>
<td>V9</td>
<td>.00</td>
<td>3.00</td>
<td>1.15</td>
<td>.81</td>
<td>.50</td>
<td>-.46</td>
</tr>
<tr>
<td>V10</td>
<td>.00</td>
<td>3.00</td>
<td>1.11</td>
<td>.74</td>
<td>.47</td>
<td>-.43</td>
</tr>
<tr>
<td>V11</td>
<td>.00</td>
<td>10.00</td>
<td>2.17</td>
<td>1.87</td>
<td>1.33</td>
<td>2.03</td>
</tr>
<tr>
<td>V12</td>
<td>.00</td>
<td>4.00</td>
<td>.28</td>
<td>.75</td>
<td>3.39</td>
<td>12.42</td>
</tr>
<tr>
<td>V13</td>
<td>.00</td>
<td>4.00</td>
<td>.21</td>
<td>.64</td>
<td>4.23</td>
<td>20.65</td>
</tr>
<tr>
<td>V14</td>
<td>.00</td>
<td>4.00</td>
<td>.51</td>
<td>1.00</td>
<td>2.34</td>
<td>5.01</td>
</tr>
<tr>
<td>V15</td>
<td>.00</td>
<td>4.00</td>
<td>.32</td>
<td>.84</td>
<td>3.22</td>
<td>10.36</td>
</tr>
<tr>
<td>V16</td>
<td>.00</td>
<td>4.00</td>
<td>.38</td>
<td>.87</td>
<td>2.79</td>
<td>7.69</td>
</tr>
<tr>
<td>V17</td>
<td>.00</td>
<td>4.00</td>
<td>.47</td>
<td>.80</td>
<td>2.21</td>
<td>5.15</td>
</tr>
<tr>
<td>V18</td>
<td>.00</td>
<td>4.00</td>
<td>.51</td>
<td>.88</td>
<td>2.10</td>
<td>4.20</td>
</tr>
<tr>
<td>V19</td>
<td>.00</td>
<td>4.00</td>
<td>.31</td>
<td>.72</td>
<td>3.02</td>
<td>9.37</td>
</tr>
</tbody>
</table>
Table 2

“Happened to Me” Traumatic Life Event Exposures

<table>
<thead>
<tr>
<th>Traumatic Life Event</th>
<th>Number Exposed</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Natural Disaster</td>
<td>99</td>
<td>25%</td>
</tr>
<tr>
<td>Fire</td>
<td>78</td>
<td>20%</td>
</tr>
<tr>
<td>Transportation Accident</td>
<td>238</td>
<td>60%</td>
</tr>
<tr>
<td>Serious Accident</td>
<td>105</td>
<td>26%</td>
</tr>
<tr>
<td>Exposure to a Toxic Substance</td>
<td>47</td>
<td>12%</td>
</tr>
<tr>
<td>Physical Assault</td>
<td>213</td>
<td>54%</td>
</tr>
<tr>
<td>Assault with a Weapon</td>
<td>172</td>
<td>44%</td>
</tr>
<tr>
<td>Sexual Assault</td>
<td>43</td>
<td>11%</td>
</tr>
<tr>
<td>Unwanted Sexual Experience</td>
<td>41</td>
<td>10%</td>
</tr>
<tr>
<td>Combat</td>
<td>40</td>
<td>10%</td>
</tr>
<tr>
<td>Captivity</td>
<td>18</td>
<td>5%</td>
</tr>
<tr>
<td>Life Threatening Disease</td>
<td>76</td>
<td>19%</td>
</tr>
<tr>
<td>Severe Human Suffering</td>
<td>47</td>
<td>12%</td>
</tr>
<tr>
<td>Sudden Violent Death</td>
<td>50</td>
<td>13%</td>
</tr>
<tr>
<td>Sudden Unexpected Death of Someone Close to You</td>
<td>214</td>
<td>54%</td>
</tr>
<tr>
<td>Serious us Injury, Harm, or Death Caused to Someone Else</td>
<td>47</td>
<td>12%</td>
</tr>
<tr>
<td>Any Other Stressful Event</td>
<td>164</td>
<td>41%</td>
</tr>
</tbody>
</table>

Hypothesized Model

The hypothesized structural equation model involving all the possible combinations of predictive paths was estimated following confirmatory factor analysis. The hypothesized model rendered acceptable fit indices, $\chi^2 = 403.15, df = 138, p < .001, \chi^2/df = 2.92, CFI = .96, TLI = .95, RMSEA = .07$ [90% CI: 0.06 to 0.07]. The item loadings and predictive paths are presented in Figure 3. When controlling for other factors, direct pathways from PTSD symptoms to psychophysiological consequences ($\beta = .02, ns$) and behavioral consequences ($\beta = -.07, ns$), did not maintain significance. Negative affect was not significantly associated with substance use ($\beta = .13, ns$) or behavioral consequences ($\beta = .15, ns$). Coping use motivations was not significantly related to psychophysiological consequences ($\beta = .18, ns$).
Re-estimated Model

In the re-estimated model, the pathway from PTSD symptoms to substance use emerged as nonsignificant ($\beta = -0.14, \text{ns}$) and was trimmed from the model. The final model including only significant pathways was presented in Figure 4 and exhibited desirable fit indices, $\chi^2 = 430.85, df = 144, p < .001, \chi^2/df = 2.99, \text{CFI} = .95, \text{TLI} = .94, \text{RMSEA} = .07 \ [90\% \ CI: .06 \text{ to } .08]$. In the final model PTSD symptoms was associated with higher negative affect ($\beta = .66, p < .001$) and coping use motivations ($\beta = .44, p < .001$). Negative affect explained higher psychophysiological consequences ($\beta = .37, p < .001$) whereas coping use motivations contributed to higher substance use ($\beta = .30, p < .001$) as well as higher behavioral ($\beta = .21, p < .001$) consequences. Substance use explained higher psychophysiological consequences ($\beta = .35, p < .001$) and higher behavioral consequences ($\beta = .39, p < .001$). The model explained 31% of the total variance in psychophysiological consequences and 24% of the total variance in behavioral consequences.

Mediational Tests.

Mediational processes were tested in the re-estimated model (Figure 3) using bootstrapping with bias corrected confidence intervals and 2000 bootstrap samples (Cheung & Lau, 2008; Sobel, 1987). A significant indirect pathway emerged from PTSD symptoms to substance use (indirect $\beta = .13, p < .001$) as mediated by coping use motivations. The pathway from PTSD symptoms to psychophysiological consequences was fully mediated by negative affect (indirect $\beta = .29, p < .001$). The pathway from PTSD symptoms to behavioral consequences (indirect $\beta = .14, p < .001$) was fully and multivariately mediated by coping use motivations and substance use. Substance use
mediated pathways from coping use motivations to psychophysiological consequences
(indirect $\beta = .11$, $p < .001$) and behavioral consequences (indirect $\beta = .12$, $p < .001$).
CHAPTER IV
DISCUSSION

The current study illuminates the understanding of pathways from PTSD symptoms to negative affect and coping use motivations, to substance use, to psychophysiological and behavioral consequences in currently imprisoned inmates. Findings highlight the results of previous investigations emphasizing the mediating role of negative affect and coping use motivations in predicting PTSD to substance use and substance use consequence pathways (Miller, Pedersen, & Marshall, 2017; Miller, Vogt, Mozley, Kaloupek, and Keane, 2006; Yeater, Austin, Green, & Smith, 2010) and contribute to the established literature by evaluating these pathways in a sample of imprisoned criminal offenders. Contrary to some prior investigations (Cottler & Mager, 1992) PTSD symptoms were not directly associated with substance use or substance use consequences when controlling for other variables. In the final model, psychophysiological consequences were explained by negative affect and substance use whereas behavioral consequences were explicited by higher coping motivations and substance use. Notably, PTSD symptoms indirectly predicted increased substance use through coping use motivations but only coping use motivations and not PTSD symptoms or negative affect directly explained increased substance use.

The structural equation model coincided with results from prior studies in that PTSD symptoms directly explained higher negative affect (Miller, Vogt, Mozley, Kaloupek, and Keane, 2006; Olatunji, Ciesilski, & Tolin, 2010) and higher coping use
motivations (Potter, Vujanovic, Marshall-Berenz, Bernstein, & Bonn-Miller, 2011) but reported nonsignificant direct pathways from PTSD to substance use and substance use consequences. Due to the inclusion of negative affect and coping use motivations in the same statistical model, pathways from PTSD symptoms to substance use consequences were fully mediated mirroring results found in non-incarcerated samples (Miller, Pedersen, & Marshall, 2017; O’Hare and Sherrer, 2011) and demonstrating a novel finding for inmates.

In the confirmatory factor analysis and the final structural equation model PTSD symptoms were not significantly associated with substance use. This is an unexpected result given that prior findings often describe a positive association between PTSD symptoms and substance use outcomes. Such investigations commonly measure substance use quantity and frequency (Stewart, Mitchell, Wright, & Loba, 2004) but rarely assess the cumulative number of substances used in the past six months. This study suggests that PTSD symptoms do not directly contribute to the number of different psychoactive substances consumed by incarcerated inmates prior to their arrest.

In the structural equation model negative affect was not directly associated with substance use but was predictive of increased psychophysiological consequences. Additionally, negative affect was found to fully mediate the pathway from PTSD to psychophysiological outcomes. These results are consistent with prior findings given that negative affect has been found to mediate the PTSD substance use consequence relation and has been consistently associated with psychological distress and physiological complaints (Watson, 1988; Watson, Clark, & Carey, 1988). Results diverge from previous findings in that negative affect did not predict increased substance use (James &
Taylor, 2007) or externalizing problem behaviors (Eisenberg et al., 2009). Simultaneous examination of both affective and cognitive precursors to substance use likely account for insignificant pathways from negative affectivity to substance use outcomes.

Coping use motivations emerged as a significant predictor of higher substance use as well as behavioral consequences, suggesting that positive direct pathways from coping use motivations to substance use consequences that have previously been reported in undergraduate samples (Blevins, Abrantes, & Stephens, 2016) can now be extended to incarcerated populations. Coping use motivations also emerged as a significant mediator in the structural equation models. Prior investigations have determined that coping use motivations can mediate the PTSD substance use pathway (O’Hare & Sherrer, 2011) and that coping use motivations can moderate the PTSD substance use relationship (Simpson, Stappenbeck, Luterek, Lehavot, & Kaysen, 2014), but have not tested mediational pathways from PTSD symptoms to coping use motivations to substance use to substance use consequences in incarcerated male inmates. Even though PTSD symptoms were not directly related to substance use, positive indirect pathways emerged from PTSD to substance use as mediated by coping use motivations. Furthermore, coping use motivations contributed to fully mediating the association between PTSD symptoms and behavioral consequences.

The number of different psychoactive substances consumed prior to arrest was associated with an increase in psychophysiological and behavioral consequences after controlling for other significant consequence predictors. This finding corroborates prior investigations determining that polysubstance use yields greater consequences than the use of a single substance (Connor, Gullo, White, & Kelly, 2014) and that increased
substance use results in increased psychophysiological consequences (Mirijello, et al., 2015; Moeller, Huttner, Struffert, & Muller, 2016; Swift & Stout, 1992) as well as adverse behavioral outcomes (Kenworthy, Ayyub, Rtveladze, Wright, Xia, & Fordham, 2017; McKetin, Lubmann, Najman, Dawe, Butterworth, & Baker, 2014).

When interpreted as a whole, the structural equation models offer several targets for therapeutic interventions in incarcerated populations. First, the importance of trauma informed care is recommended as PTSD symptoms are predictive of both negative affect and coping use motivations that explain substance use behavior and substance use consequences. Several evidence-based treatment programs have already been adapted for use to treat PTSD in male offenders with promising results to include Seeking Safety (Barrett et al., 2015) and the Male-Trauma Recovery Empowerment Model (Wolff, Huening, Shi, Frueh, Hoover, & McHugo, 2015). Such treatments emphasize the simultaneous treatment of trauma and substance related concerns and have demonstrated positive sustainable outcomes in comparison to conventional psychotherapies (Wolff et al., 2015).

Negative affect should also be the target of therapeutic interventions. Emotional regulation therapies are one set of broadly defined therapeutic techniques that can increase the ability to cope with emotional difficulties. Suppression and avoidance of negative emotions can increase the propensity for harmful self-medicative coping but adaptive emotional regulation strategies including acceptance and problem solving correspond with a reduction in harmful substance abuse behaviors (Aldao, Nolen-Hoeksema, & Schweitzer, 2010). Mindfulness meditation is one effective emotional regulation strategy focused on developing nonjudgmental acceptance of unwanted
emotional experiences (Teasdale, Segal, & Williams, 1995). Application of mindfulness practices in criminal justice settings represent a low-cost treatment alternative associated with reduction of substance use and substance use consequences (Bowen et al., 2006).

Interventions for male inmates should also target coping use motivations. Current evidence-based PTSD treatments can be difficult for clients to complete and result in high rates of client dropout (Imel, Laska, Jakupcak, & Simpson, 2013). Thus, targeting coping use motivations that fully mediate the effects of PTSD on substance use outcomes might represent a more effective and implementable treatment strategy for substance using offenders. Interventions consisting of three one-hour brief cognitive behavioral therapy sessions delivered in a group format have previously demonstrated a sustainable reduction in coping expectancies and substance consumption at ten weeks post treatment (Watt, Stewart, Birch & Bernier, 2006). Coping use motivations might also be attenuated by increasing coping self-efficacy. Social cognitive theory argues that positive and sustainable behavioral change is made possible as human beings develop beliefs in the effectiveness of a behavior within a specific environmental context (Bandura, 2011). As inmates successfully learn and apply positive healthy coping strategies, coping self-efficacy increases allowing for adaptive coping behaviors. Studies of self-efficacy in substance abuse treatment consistently relate lower self-efficacy beliefs to higher levels of substance use (McKay et al., 2004) and higher self-efficacy beliefs to successful treatment outcomes (Dolan, Martin, and Rohsenow, 2008).

The current study includes several limitations. Given that the investigation focused on incarcerated male offenders, findings should be cross-validated using an incarcerated female sample. Furthermore, study pathways should be tested longitudinally
to verify that theoretically explained temporal connections between factors are being accurately described (Crano, Brewer, & Lac, 2015). Moreover, the present investigation assessed only total number of psychoactive substances used in the past six months. Assessing substance use frequency by allowing the respondent to numerically indicate quantity and frequency on a continuous scale might increase the accuracy of the substance use measure. Finally, additional substance use predictors such as antisocial personality, traumatic event exposures, or competing substance use motivations could be statistically controlled for to rule out rival explanatory pathways. In summary, findings emphasize that only coping use motivations remain a risk factor for substance use whereas negative affect, coping use motivations, and substance use differentially predict substance use consequences. The explanatory power of PTSD symptoms on substance use outcomes was found to be fully mediated after statistically controlling for negative affect, coping use motivations, and substance use in incarcerated offenders. The present investigation should inform PTSD and substance abuse treatments for male inmates through a focus on treatment targets including PTSD symptoms, negative affect, and coping use motivations.
REFERENCES


APPENDIX

IRB APPROVAL

University of Colorado
Colorado Springs
Institutional Review Board (IRB) for the Protection of Human Subjects

Date: 3/28/2017

IRB Review

IRB PROTOCOL NO.: 17-174
Protocol Title: Post Traumatic Stress Predicting Substance Use in an Incarcerated Population
Principal Investigator: Nathaniel Brack
Faculty Advisor if Applicable: Andrew Lac
Application: New Application
Type of Review: Expedited
Risk Level: No more than Minimal Risk
Renewal Review Level (if changed from original approval) if Applicable: Expedited
This Protocol involves a Vulnerable Population: Prisoners
Expires: 27 March 2018
Note: if exempt: If there are no major changes in the research, protocol does not require review on a continuing basis by the IRB. In addition, the protocol may match more than one review category not listed.
Externally funded: ☐ No ☐ Yes
OSP #: 
Sponsor:

Thank you for submitting your Request for IRB Review. As Exemption per 45 CFR 46 do not apply to research using prisoners as subjects, this research is reviewed as Expedited under Category 5. The protocol identified above has been reviewed according to the policies of this institution and the provisions of applicable federal regulations. The review category is noted above, along with the expiration date, if applicable.

Once human participant research has been approved, it is the Principal Investigator’s (PI) responsibility to report any changes in research activity related to the project:

- The PI must submit all protocol, recruitment, advertising, and consent form amendments/revisions to the IRB for approval.
- The IRB must approve these changes prior to implementation.
- If you are a student, please note that it is required to include the IRB approval letter in the library when you submit the dissertation/thesis.
- The PI must promptly inform the IRB of all unanticipated serious adverse events (within 24 hours). All unanticipated adverse events must be reported to the IRB within 1 week (see 45CFR46.116(b)). Failure to comply with these federally mandated responsibilities may result in suspension or termination of the project.
- Leave study with the IRB at least 19 business days prior to expiration.
- Notify IRB when the study is complete.

If you have any questions, please contact Research Integrity Specialist in the Office of Sponsored Programs and Research Integrity at 719-255-3503 or irb@uccs.edu.

Thank you for your concern about human subject protection issues, and good luck with your research.

Sincerely yours,

Deborah J. Kenney, Ph.D., RN, FAAN
Deborah J. Kenney
UCCSS IRB Chair

www.uccs.edu/irb
Version 11/11/16

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