THE IMPACT OF COMORBID DEPRESSION ON ANGER, QUALITY OF LIFE, AND POSTTRAUMATIC COGNITIONS IN COMBAT VETERANS WITH POSTTRAUMATIC STRESS DISORDER

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ABSTRACT

Posttraumatic Stress Disorder (PTSD) is a psychological disorder that can develop after exposure to traumatic experiences, and shares significant comorbidity with depression. This is of concern given the array of negative correlates associated with PTSD and depression comorbidity, and various explanations have been proposed to explain the large degree of overlap between disorders. These include the existence of a general traumatic stress construct, examination of symptom overlap between disorders, as well as models that attempt to explain the broader overlap between depression and anxiety, such as the Quadripartite Model. While there has been substantial research in the field of PTSD and depression comorbidity, there are still many areas in need of further empirical examination, including comorbid PTSD and depression’s impact on anger, quality of life, association with posttraumatic cognitions, as well as its relationship with PTSD numbing symptoms. The purpose of this dissertation was to address these gaps in the literature across three related studies. The first study examined the impact of comorbid MDD and PTSD numbing symptoms on state and trait anger, while the second study examined the associations of comorbid MDD, depressive symptoms, and PTSD symptom clusters with objective and subjective quality of life indices. The third study examined the differential associations of comorbid depressive symptoms and PTSD symptom clusters with posttraumatic cognitions. Taken together, the results of these three studies address the relationship between comorbid depression and PTSD numbing symptoms, inform the clinical care of comorbid individuals, and contribute to the ongoing development of explanations for this comorbidity.
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List of Abbreviations

Beck Depression Inventory II (BDI-II), Cognitive Processing Therapy (CPT), Clinician-Administered PTSD Scale (CAPS), Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV), Diagnostic and Statistical Manual of Mental Disorders 5 (DSM-5), major depressive disorder (MDD), National Vietnam Veteran Readjustment Study (NVVRS), Operation Enduring Freedom/Operation Iraqi Freedom (OEF/OIF), posttraumatic stress disorder (PTSD), Posttraumatic Stress Disorder Checklist (PCL), PCL-Military Version (PCL-M), PCL-Stressor Specific Version (PCL-S), Quality of Life Inventory (QOLI), Short Form 12 Health Survey (SF-12), SF-12 Physical Component Summary (PCS), SF-12 Mental Component Summary (MCS), State-Trait Anger Expression Inventory-II (STAXI), Structured Clinical Interview for DSM-IV Axis I Disorders (SCID), Vietnam Experience Study (VES)
**Overall Introduction**

Posttraumatic Stress Disorder (PTSD) is a psychological disorder that can develop after exposure to traumatic experiences, such as natural disaster, rape, or military combat. To warrant a diagnosis of PTSD according to the criteria established by the Diagnostic and Statistical Manual of Mental Disorders-IV-Text Revision (DSM-IV-TR; American Psychiatric Association, 2000), an individual must experience fear, helplessness, or horror during or after the traumatic event, as well as symptoms from three separate clusters. These clusters include reexperiencing symptoms (e.g. recurrent involuntary memories of the event, traumatic nightmares), avoidance (e.g. avoiding trauma-related thoughts and feelings, experiencing feelings of detachment from others, restricted affect), and increased arousal (e.g. hypervigilence, exaggerated startle response). These symptoms must persist for longer than a month, not be attributable to a medical illness, medication, or substance use, and result in functional impairment.

Recently, the American Psychiatric Association released the DSM-5, which modified PTSD’s diagnostic criteria. The revisions included eliminating the requirement of fear, helpless, or horror surrounding the trauma, as well as delineating two new subtypes of PTSD. Arguably the largest change, however, was dividing avoidance symptoms into two separate clusters. These included a modified avoidance cluster, containing just two items focused on avoiding thoughts, feelings, people, and places, as well a new cluster with symptoms related to negative alterations in cognitions and mood, such as feelings of detachment from others and restricted affect. This cluster also contains several new items, including persistent negative beliefs about oneself and
the world, distorted blame related to the trauma, and persistent trauma-related emotions, such as anger, guilt, or shame (American Psychiatric Association, 2013)

**PTSD Prevalence Estimates**

Large epidemiological studies have provided estimates of the prevalence of PTSD in both the general and veteran populations. In the National Comorbidity Survey Replication Study, researchers observed a lifetime prevalence of 6.8% for PTSD in the general population (Kessler et al., 2005). Additionally, a review of general population epidemiological studies of PTSD found lifetime rates varying from 5% to 6% for men, and 10% to 14% for women (Breslau, 2002). Current PTSD exhibits a much lower prevalence, and one large study found current rates of 1.2% for men and 2.7% for women (Stein, Walker, Hazen, & Forde, 1997).

Epidemiological research has also been conducted with veteran samples, and produced somewhat incongruent findings. In the Vietnam Experience Study (VES), researchers found a 2.2% prevalence for current PTSD, while the National Vietnam Veteran Readjustment Study (NVVRS) produced a rate of 15.2% for current PTSD (see Thompson, Gottesman, & Zalewski, 2006). This discrepancy appears to be the result of methodological differences across studies, as the VES used more specific cut scores, a one month prevalence estimate, and a single indicator of PTSD, while the NVVRS used more sensitive cut scores, a six month prevalence estimate, and multiple indicators of PTSD (Thompson et al., 2006). Studies with Operation Enduring Freedom/Operation Iraqi Freedom (OEF/OIF) veterans have produced rates that correspond
more closely to the higher estimate of the NVVRS. In a study involving data from 289,328
veterans, researchers found a two-year prevalence of 18.2% for new PTSD diagnoses (Seal et al.,
2009). Likewise, a RAND Cooperation study, which interviewed returning OEF/OIF service
members, reported a rate of 14% for current PTSD (Tanielian & Jaycox, 2008). While the
question of PTSD’s prevalence is still undergoing empirical examination, the existing research
appears to suggest that PTSD rates are higher in the veteran population than in the general
population.

**PTSD Negative Associations and Comorbidity**

Research has also documented the many negative correlates of PTSD. PTSD has been
associated with suicide (Ferrada-Noli, Asberg, Ormstad, Lundin, & Sundbom, 1998), anger
(Novaco & Chemtob, 2002), problems with intimacy, separation, and divorce (Riggs, Byrne,
Weathers, & Litz, 1998), parenting and familial conflict (Samper, Taft, King, & King, 2004;
Sayers, Farrow, Ross, & Oslin, 2009), occupational impairment (Rona et al., 2009; Schnurr &
Lunney, 2011), financial problems (Elbogen, Johnson, Wagner, Newton, & Beckham, 2012),
decreased quality of life (Mendlowicz & Stein, 2000; Mogotsi, Kaminer, & Stein, 2000), and
impaired functioning across many other domains. PTSD also has a high degree of comorbidity
with other disorders, an issue highlighted by the National Comorbidity Survey, which found that
59% of men and 44% of women with PTSD met criteria for three or more other psychiatric
diagnoses (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). PTSD has been associated
with comorbid psychotic disorders (Mueser et al., 1998), eating disorders (Dansky, Brewerton,
Kilpatrick, & O'Neil, 1997), substance use disorders (Brown, Campbell, Lehman, Grisham, & Mancill, 2001), other anxiety disorders (Zayfert, Becker, Unger, & Shearer, 2002), and affective disorders (Brown et al., 2001; Zayfert et al., 2002).

Perhaps the disorder possessing the highest rate of comorbidity with PTSD is depression. A recent meta-analysis involving 57 studies found rates of PTSD and MDD comorbidity ranging from 19% to 89%, with a mean prevalence of 52% across studies (Rytwinski, Scur, Feeny, & Youngstrom, 2013). This is highly concerning, as PTSD and depression comorbidity has been associated with more severe impairment and poorer outcomes than the individual disorders alone. This includes more PTSD symptoms (Gros, Price, Magruder, & Frueh, 2012; Tural, Onder, & Aker, 2012) and MDD symptoms (Campbell et al., 2007; Ikin, Creamer, Sim, & McKenzie, 2010), as well as more dissociation, trauma-related beliefs (Taft, Resick, Watkins, & Panuzio, 2009), suicidality (Campbell et al., 2007; Chan, Cheadle, Reiber, Unützer, & Chaney, 2009), and risky sexual behavior (Holmes, Foa, & Sammel, 2005; Plotzker, Metzger, & Holmes, 2007). PTSD and depression comorbidity has also been empirically tied to diminished psychosocial functioning (Blanchard, Buckley, Hickling, & Taylor, 1998; Ginzburg, Ein-Dor, & Solomon, 2010), decreased social support (Chan et al., 2009; Tural et al., 2012), and increased health-care utilization (Chan et al., 2009).

PTSD and Depression Comorbidity: General Traumatic Stress Construct

A large amount of attention has been devoted to theories explaining the high degree of co-occurrence between PTSD and depression. One prominent explanation is that the disorders
are different manifestations of a single general traumatic stress construct (O’Donnell, Creamer, & Pattison, 2004), a theory based on earlier research suggesting an underlying shared vulnerability for the disorders (Breslau, Davis, Peterson, & Schultz, 2000; Breslau, Davis, Peterson, & Schultz, 1997). There is substantial research supporting this theory, as well as refuting it, across several different domains, including predictors, course of the disorders, and factor analytic studies. With regard to predictors, researchers suggest that if PTSD and depression were manifestations of an underlying construct, they would overlap in their risk factors (Norman et al., 2011). Research supports this position to some extent, as studies examining predictors of the individual disorders have identified a number of commonalities. Two large studies drawing data from the Virginia Twin Registry found that, among other factors, childhood sexual abuse, past history of depression, low education, and low social support were all predictors of a major depressive episode (Kendler, Gardner, & Prescott, 2002, 2006). A meta-analysis across 85 datasets identified low education and social support as modest predictors of PTSD, while childhood trauma and past psychiatry history were robust predictors (Brewin, Andrews, & Valentine, 2000). Research that has compared predictors of PTSD and depression in the same study also provides support for this theory. In the study that laid the groundwork for the theory of a general traumatic stress construct, there were no significant differences in the relative odds of being diagnosed with PTSD or comorbid PTSD-MDD across a variety of predictors at 12 months post-injury (O’Donnell et al., 2004). Likewise, in a sample of patients recruited at a trauma center, post-injury pain and PTSD symptoms measured within 48 hours of the trauma were both associated with higher rates of PTSD and MDD symptoms at one month.
follow-up (Norman et al., 2011). However, all of these studies (Brewin et al., 2000; Kendler et al., 2002, 2006; Norman et al., 2011) also found a number of predictors unique to either PTSD or depression. Indeed, even in the foundational general traumatic stress construct study, at three months post-injury depression evidenced so many unique predictors that the researchers theorized about the existence of a brief reactive depression construct (O’Donnell et al., 2004).

Another domain which has relevance to evaluating this theory is the courses of the two disorders, as researchers have outlined several cases which would support the idea of a general traumatic stress construct. Breslau et al. (2000) noted that if studies demonstrated that PTSD preceded and increased the risk for depression, and depression preceded and increased the risk for PTSD, it would suggest a shared underlying vulnerability. There is research that supports these conditions (Breslau et al., 1997), however it is noteworthy that many studies have found that PTSD proceeds depression (see Shalev et al., 1998), including the National Comorbidity Study, in which 78.4% of individuals with comorbid PTSD-MDD reported that the onset of PTSD came before that of MDD (Kessler et al., 1995). Other research has demonstrated that the disorders follow a similar pattern of occurrence and remittance (Norman et al., 2011; Shalev et al., 1998), which could be interpreted both in favor of and against the theory of an underlying general traumatic stress construct.

Moreover, another aspect of the course of the disorders that has bearing on this theory is whether depression develops independently of PTSD following a traumatic experience. If individuals only develop depression following or in conjunction with PTSD, it would provide additional support for the general traumatic stress construct. However, if depression developed
without PTSD following a trauma, it would more strongly support the idea of independent disorders (O’Donnell et al., 2004). Studies have found that risk for MDD increased only in individuals who developed PTSD, and that trauma exposure not resulting in PTSD did not significantly increase the risk of MDD (Breslau et al., 2000; Breslau et al., 1997). However, other research has discovered subsets of individuals who only develop depression following a traumatic experience (Shalev et al., 1998), including the research which originally promulgated the general traumatic stress construct theory (O’Donnell et al., 2004).

Factor analytic research also speaks to the question of a general traumatic stress construct. Numerous studies have found that PTSD loads with MDD, even more so that with over other anxiety disorders (Cox, Clara, & Enns, 2002; Slade & Watson, 2006), and research has demonstrated that PTSD and MDD, along with generalized anxiety disorder, form a higher order factor (Grant, Beck, Marques, Palyo, & Clapp, 2008). While this provides some support for the general traumatic stress construct theory, it is important to note that this research also concluded that PTSD and MDD are highly related, but distinct, disorders (Grant et al., 2008). Similarly, researchers who conducted factor analytic procedures on National Comorbidity Study data, and who argued strongly in favor of the general traumatic stress construct, still found a secondary sub-factor that could be labeled depression (Elhai et al., 2011). In sum, the current research does not support the general traumatic stress construct theory as a means of fully explaining PTSD’s high degree of comorbidity with depression. Rather, it seems to suggest that the two disorders are separate but highly related constructs, and if there is an underlying vulnerability shared by the disorders, it only accounts for a portion of their comorbidity.
PTSD and Depression Comorbidity: Symptom Overlap

There is also a simpler, alternate explanation for PTSD and depression comorbidity: overlap in symptoms. In the DSM-IV-TR, PTSD had three symptom clusters (i.e. reexperiencing, avoidance, and hyperarousal), and one cluster (i.e. avoidance) contained a number of items similar to those of depression (e.g. markedly diminished interest or participation in significant activities, feeling of detachment or estrangement from others, restricted range of affect; American Psychiatric Association, 2000). While some research suggests that these overlapping symptoms are not the cause of the high rates of PTSD and depression comorbidity (Blanchard et al., 1998; Elhai et al., 2011; Franklin & Zimmerman, 2001), other studies identify symptom overlap as a contributing factor (see Gros et al., 2012). Research has demonstrated that overlapping symptoms actually load more strongly onto a depression factor than onto a PTSD factor, and are predictive of PTSD-MDD comorbidity (Gros, Simms, & Acierno, 2010). Additionally, numerous factor analytic studies using the DSM-IV-TR PTSD criteria have identified four factor solutions which partition these overlapping symptoms into their own cluster, alternately labeled dysphoria (Simms, Watson, & Doebbelling, 2002) or numbing (King, Leskin, King, & Weathers, 1998) depending on the exact symptom configuration. Both models have been supported in a large metaanalytic study (Yufik & Simms, 2010).

PTSD numbing symptoms demonstrate a similar pattern of correlates to depression, with negative associations to domains including psychosocial functioning and quality of life (Hassija, Jakupcak, & Gray, 2012; Judd et al., 2000; Schnurr, Lunney, Bovin, & Marx, 2009). PTSD numbing symptoms do, however, demonstrate unique effects above and beyond those associated
with depression, including increased cigarette smoking (Cook, Jakupcak, Rosenheck, Fontana, & McFall, 2009), decreased parenting satisfaction (Samper et al., 2004), as well as problems in other domains. These findings suggest that, even if symptom overlap is a primary driver of PTSD and depression comorbidity, these symptoms still are distinct from comorbid depression. Much like the work examining the general traumatic stress construct, these studies argue for a model in which PTSD and depression significantly overlap, but retain unique dimensions.

**PTSD and Depression Comorbidity: Quadripartite Model**

A broader theoretical framework, which captures this large degree of overlap while still allowing for disorder-specific symptoms, is the Quadripartite Model (Watson, 2005). The theory represents one of the most recent incarnations of over four decades of research attempting to explain the relationship between depression and anxiety disorders. Initial studies, during the 1970s and 80s, focused mainly on correlations among whole scales, and found a large degree of overlap among depression and anxiety measures (Watson, 2009). Following this, researchers began to focus on specific symptoms, and one notable theory of this phrase was the Tripartite Model (Clark & Watson, 1991). This model grouped depression and anxiety symptoms into three factors: a general distress factor comprised of anxious and depressed mood symptoms, as well as symptom factors unique to anxiety (e.g. somatic hyperarousal) and depression (e.g. anhedonia/low positive mood). However, as a result of unique associations between different anxiety disorders and depression, as well as research suggesting the need for more disorder-specific factors, researchers began examining disorder-based analyses. This included Barlow
and colleagues’ Hierarchical Model (Brown & Barlow, 1992), as well as the Integrative Hierarchical Model, the latter of which attempted to quantitatively model a general distress component (Mineka, Watson, & Clark, 1998).

While the disorder-based approach had many benefits, including specifically placing research within the framework of DSM disorders, it also had numerous shortcomings, such as susceptibility to low base rates, changing diagnostic criteria, and diagnostic inconsistency and unreliability (Watson, 2009). Watson’s Quadripartite Model (Watson, 2005) attempted to address these limitations by integrating the DSM disorder framework with a more specific symptom focus, as well as quantitative elements of the Integrative Hierarchical Model. It proposed classifying symptoms of DSM-IV disorders on two quantitative dimensions: magnitude of general distress, as well as magnitude of specificity in terms of degree of relationship with depression and anxiety disorders. Within this framework, PTSD was classified as a “distress” disorder, alongside depression and dysthymia, given its large general distress component and small degree of specificity. Watson also suggested that it may not be necessary to have such a large distress component (i.e. numbing symptoms) built into PTSD’s diagnosis, and others have more explicitly advocated for its removal (Spitzer, First, & Wakefield, 2007).

The Quadripartite Model does not explain PTSD and depression’s comorbidity so much as it proposes a new taxonomy for grouping the disorders. But, given the cited research indicating the independent but highly correlated nature of PTSD and depression, the Quadripartite Model does provide a means for conceptualizing the unique and overlapping
elements the disorders, and a useful framework for future research into the causes and correlates of their comorbidity.

**Areas In Need of Further Empirical Examination**

While there has been significant interest in the field of PTSD and depression comorbidity in recent years, there are still many areas in need of further empirical examination. Specifically, despite research demonstrating that anger accounts for over 40% of the variance in PTSD symptom scores (Novaco & Chemtob, 2002), as well as theories and evidence which connect depression to anger (Berkowitz, 1990), very little research has examined comorbid PTSD and depression’s impact on anger. In the only relevant study to date, PTSD symptoms were indirectly related to aggression through dysphoria symptoms (Taft et al., 2009). However, this study examined only aggression, not anger, and utilized dysphoria symptoms, rather than comorbid depression.

Another area in need of further research is the connection between PTSD and depression comorbidity and quality of life. While there has been some important examination of this topic (Ginzburg et al., 2010; Ikin et al., 2010), more work is needed in a variety of areas. Specifically, most studies to date have examined a limited number of quality of life indicators, and few studies have attempted to disentangle the unique contributions of each disorder to quality of life. The research that has addressed this issue has significant limitations, including the failure to explore the influence of individual PTSD symptom clusters (Pittman, Goldsmith, Lemmer, Kilmer, & Baker, 2012; Rauch et al., 2010). Furthermore, there is sparse research examining the pathways
in which comorbid PTSD and depression may lead to decreased quality of life (e.g. Gudmundsdottir, Beck, Coffey, Miller, & Palyo, 2004).

Additionally, further research is needed examining the content specificity of depressive and posttraumatic cognitions. Content specificity holds that disorders have unique sets of cognitions that predict their development and maintenance (Ehring, Ehlers, & Glucksman, 2008). While the cognitions associated with PTSD and depression significantly overlap, the existent research exploring the specificity of these cognitions to each disorder is sparse (Gonzalo, Kleim, Donaldson, Moorey, & Ehlers, 2012). To date, no research has addressed this issue in a veteran sample, nor has existing research examined the associations of maladaptive cognitions with individual PTSD symptoms clusters.

A final domain requiring additional research is the connection between PTSD numbing symptoms and PTSD and depression comorbidity, which has both clinical and theoretical implications. In the DSM-IV, emotional numbing symptoms were placed together with avoidance, and PTSD could be diagnosed largely in the absence of numbing symptoms (American Psychiatric Association, 2000). However, the new DSM 5 criteria require two symptoms from the negative alterations in cognition and mood cluster, a cluster which significantly overlaps with numbing symptoms. From a clinical utility perspective, individuals diagnosed with PTSD under the new DSM 5 criteria are more likely to evidence PTSD numbing symptoms, and as such, it is important to understand the impact of these symptoms on domains such as anger, quality of life, and response to treatment. Moreover, individuals with comorbid PTSD and depression evidence higher levels of numbing symptoms than individuals with PTSD.
alone (Gros et al., 2012), which suggests that this diagnostic shift could be even more consequential for comorbid individuals.

From a theoretical perspective, understanding if the pattern of relationships for PTSD numbing symptoms varies from that of comorbid depression might inform the debate concerning the removal of PTSD’s numbing symptoms. The goal of such an alteration would be to increase PTSD’s specificity, and an assumption of this approach is that no useful information would be lost by the elimination of the numbing items. This assumption can be tested through comparison of the pattern of relationships that both PTSD numbing symptoms and comorbid depression demonstrate with the same set of clinical correlates. If PTSD numbing symptoms and comorbid depression evidence a nearly identical pattern of correlations, it would argue for removal of PTSD’s numbing symptoms. However, if they were to demonstrate a differential pattern of correlates, it would support retaining PTSD’s numbing symptoms, or it would alternately support including some of these symptoms in a general distress metric proposed by Watson in his discussion of the Quadripartite Model (Watson, 2009).

**Purpose of this Study**

The purpose of this dissertation was to address these gaps in the literature across three related studies. The first study examined the mediating role of comorbid MDD, as well as PTSD numbing and dysphoria symptoms, on the relationships between PTSD symptom clusters and state and trait anger. The second study examined the impact of comorbid PTSD and depression across objective and subjective quality of life domains (social-material, functioning, and
satisfaction), and also explored the unique contributions of each disorder to decreased quality of life. Furthermore, this study looked at the possible mediating influence of comorbid depressive symptoms on PTSD cluster-quality of life relationships. The third study was concerned with the content specificity of posttraumatic cognitions, and examined their associations with comorbid depressive symptoms and individual PTSD symptom clusters. Taken together, the results of these studies inform the clinical care of comorbid individuals and contribute to the ongoing theoretical development of the models seeking to explicate PTSD and depression comorbidity.
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Paper #1: Influence of Depression on State and Trait Anger in Veterans with Posttraumatic Stress Disorder

Please note that results of this study have already been published. Citations for the full manuscript are:


Abstract

Anger is one of the most important symptoms of posttraumatic stress disorder (PTSD), and is associated with many of the adverse correlates of PTSD. Researchers have proposed theories to explain the relationship between anger and PTSD, but no study to date has examined the mediating role of depression. The purpose of this study was to explore the mediating effects of current major depression disorder (MDD), as well as PTSD numbing and dysphoria symptom clusters (King, Leskin, King, & Weathers, 1998; Simms, Watson, & Doebbeling, 2002) on the relationship between PTSD and anger. There were 98 participants in the study, and all were male veterans with combat-related PTSD taking part in a clinical trial.
Introduction

Anger is perhaps one of the most important symptoms of posttraumatic stress disorder (PTSD). PTSD-related anger is associated with a number of the adverse correlates of PTSD, including poor physical health (Ouimette, Cronkite, Prins, & Moos, 2004), poor response to evidence-based treatment (Rizvi, Vogt, & Resick, 2009), and aggressive behavior (Teten, Miller, & Stanford, 2010). Research has also identified anger as the single largest symptom predictor of overall PTSD symptom severity, accounting for as much as 40% of the variance in PTSD scores (Novaco & Chemtob, 2002). Additionally, the strong correlations between anger and PTSD remain even when the PTSD anger symptom is removed from analyses (Novaco & Chemtob, 2002). Together, these findings suggest that the effect of anger on PTSD cannot be solely attributed to its inclusion in the disorder’s diagnostic criteria (Orth & Wieland, 2006).

Researchers have proposed several theories to explain the relationship between anger and PTSD. One school of thought holds that trauma-related intrusive thoughts trigger fear, and anger is adopted as an alternative, preferable emotional state (Feeny, Zoellner, & Foa, 2000). Another recent theory argues that anger, within the context of PTSD, is a unique construct that is differentiated from other forms of anger by the influence of visual imagery. In this model, visual imagery has connections to brain regions associated with threat detection, and also reciprocally influences both anger and PTSD symptoms (McHugh, Forbes, Bates, Hopwood, & Creamer, 2012). One particularly influential theory of the relationship between PTSD and anger was developed by Chemtob and colleagues, and emphasizes “survival mode” in which cognitive biases are activated in response to life threatening situations (Chemtob, Novaco, Hamada, Gross,
& Smith, 1997). Anger is conceptualized as an important survival response associated with heightened arousal and threat-detection biases that evoke anger responses with minimal provocation. Although adaptive in life-threatening situations, “survival mode” becomes maladaptive when activated in non-threatening contexts, and is a phenomenon that Chemtob has suggested is a hallmark of PTSD (Chemtob et al., 1997).

There is also theoretical reason to suspect that depression may influence the relationship between PTSD and anger. The Cognitive Neoassociationistic Model (Berkowitz, 1990) postulates that negative affect can influence anger through links in associative networks in which it is connected to anger-related feelings, thoughts, and memories. When an individual has an unpleasant experience, it induces negative affect which automatically and simultaneously gives rise to fear and anger responses (Berkowitz, 1990). Olatunji, Ciesielski, and Tolin (2010) have supported the applicability of this theory to the PTSD and anger relationship, as they theorize that a higher-order vulnerability to negative affect may serve a mediating role. Empirical studies have also provided some indirect support for this possibility, and in one study, depressive symptoms had a significant indirect effect on the relationship between PTSD and aggression (Taft, Vogt, Marshall, Panuzio, & Niles, 2007), a related but distinct construct from anger (Averill, 1983; Chemtob et al., 1997).

To date, no study has evaluated the mediational role of depression on the PTSD and anger relationship. Examining depression’s role as a potential mediator could result in a better understanding of the factors that influence the relationship between PTSD and anger, and also identify potential treatment targets to reduce PTSD-related anger. However, this analysis is
complicated by the symptom overlap between PTSD and major depressive disorder (MDD). A recent study found that the factor-analytically derived numbing (DSM-IV C3-C7 symptoms; King et al., 1998) and dysphoria (DSM-IV C3-C7 and D1-D3 symptoms; Simms et al., 2002) PTSD clusters primary loaded on a MDD factor rather than on a PTSD factor (Gros, Simms, & Acierno, 2010). It is possible that depression would not significantly affect the relationship between total PTSD symptom severity and anger because many depressive symptoms are already inherent in the PTSD diagnostic criteria. Additionally, the numbing and dysphoria clusters may act as mediators of the PTSD and anger relationship, suggesting the possibility that the depression-related symptoms of PTSD itself may be sufficient to influence anger levels.

To address these possibilities in the present study, we evaluated PTSD and depression in several ways. In addition to defining PTSD by total symptom severity, we also evaluated the PTSD clusters identified by King et al. (1998) and Simms et al. (2002) that do not overlap with depression. These include reexperiencing, avoidance, and hyperarousal (Simms et al., 2002). Additionally, to assess depression we used both clinician-derived diagnosis of MDD, as well as self-report measures of PTSD numbing and dysphoria clusters. Furthermore, we assessed anger with the State-Trait Anger Expression Inventory-II (STAXI) state and trait scales, which are conceptualized as the two major components of the experience of anger (Spielberger, 1999).

The purpose of the present study was to examine the mediational role of depression on the PTSD and anger relationship in a sample of male combat veterans participating in a PTSD treatment trial. On the basis of the Cognitive Neoassociationistic Model, we hypothesized that PTSD symptom clusters not overlapping with depression (reexperiencing, avoidance, and
hyperarousal) would act as the initial unpleasant event necessary to evoke negative affect in the form of depression, which in turn would lead to anger. Because of evidence for other factors that influence the relationship between PTSD and anger (e.g. Chemtob et al., 1997; McHugh et al., 2012), we hypothesized depression would partially, rather than fully, mediate this relationship.

We hypothesized that: (1) MDD would not significantly mediate the relationships between total PTSD symptom severity and state and trait anger because of inherent overlap in diagnostic criteria (2) MDD would partially mediate the relationships between the PTSD reexperiencing, avoidance, and hyperarousal clusters and state and trait anger, and (3) PTSD numbing and dysphoria clusters would partially mediate the relationships between PTSD reexperiencing, avoidance, and hyperarousal clusters and state and trait anger.

Method

Procedure

For this study, we drew data from baseline assessments of a large, ongoing clinical trial evaluating the efficacy of the telehealth modality for delivering Cognitive Processing Therapy to veterans with combat-related PTSD (for more information, see Morland, Hynes, Mackintosh, Resick, & Chard, 2011). Data were collected at VA clinics across the Hawaiian Islands during baseline assessments. Inclusion criteria for the study were a diagnosis of current PTSD based on the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995) and stability of psychiatric medications for 2 months or more. Exclusion criteria were active psychotic symptoms, active suicidal or homicidal ideation, significant cognitive disorder or organic mental disorder,
unwillingness to refrain from substance use during treatment, or current active substance dependence.

Participants
Participants in this study consisted of 98 male combat veterans who were who primarily Caucasian (N = 47, 48.5 %), Asian (N = 16, 16.5 %), or Pacific Islander (N = 15, 15.5 %), and who were largely married (N = 56, 57.1 %), divorced (N = 28, 28.6 %), or single (N = 12, 12.2 %). Additionally, participants served in the Vietnam (N = 67, 68.4 %), Operation Enduring Freedom/Operation Iraqi Freedom (N = 22, 22.4 %), and Desert Storm/Shield (N = 16, 16.3 %) war eras. Participants had a rate of MDD (N = 32, 33.7 %) which was low compared to rates in other samples of veterans with PTSD (Gros, Price, Magruder, & Frueh, 2012). The mean PCL score for this sample (M = 59.28, SD = 11.06) was low compared to other samples of veterans enrolled in PTSD-related clinical trials (Morland et al., 2010; Gros, Yoder, Turek, Lozano, & Acierno, 2011). Percentages differ across demographics because of a small amount of missing demographic data (0.7 %).

Measures
Clinician-Administered PTSD Scale. The CAPS (Blake et al., 1995) is a clinician-administered diagnostic interview for DSM-IV PTSD. The CAPS was used to establish diagnosis of PTSD with the “1/2” rule, meaning that symptoms had to have occurred in the last month and be of at least a moderate intensity (Weathers, Ruscio, & Keane, 1999). The CAPS has good convergent and divergent validity, as well as strong reliability (Blake et al., 1995).
**Structured Clinical Interview for DSM-IV Axis I Disorders (SCID).** The SCID is a clinician-administered interview for diagnosis of DSM-IV Axis I disorders (First, Spitzer, Gibbon, & Williams, 2002). For the purposes of the present study, an abbreviated version of the SCID was administered, and only data on diagnosis of current MDD (i.e. currently experiencing a depressive episode) was used. The SCID has superior agreement with longitudinal, multi-informant derived diagnoses when compared to standard clinical interview (Basco et al., 2000).

**Posttraumatic Stress Disorder Checklist (PCL).** The PCL (Weathers, Litz, Herman, Huska, & Keane, 1993) is a 17-item self-report questionnaire that uses a 5-point Likert scale to measure the severity of the 17 DSM-IV diagnostic criteria over the last month. The PCL has excellent internal consistency (alphas > .94) and test–retest reliability (r = .96; for review see Orsillo, 2002). In addition, the PCL has excellent convergent validity with alternative measures of PTSD (rs range from .77 to .93; Orsillo, 2002).

In this study, PTSD symptom severity was assessed by both the PCL-Military Version (PCL-M) and the PCL Stressor Specific Version (PCL-S). The PCL-M was used with the first cohort of the study, and the PCL-S was used in all subsequent cohorts. The scales have virtually identical item content but differ in their instructions, and research within the military population suggests that changes to PCL-S instructions do not affect symptom reporting (Riviere et al., 2011). When accounting for depressive symptoms, there were no significant differences found between PCL-M and PCL-S scores in this sample (p > .05).

For analyses we derived PTSD symptoms scores from the PCL-M for the first cohort and from the PCL-S for all subsequent cohorts. All analyses involving the PCL were conducted with
the anger item removed. For analyses involving PTSD symptom clusters, we used the four-factor models developed by King et al. (1998) and Simms et al. (2002). Both models share identical reexperiencing (DSM-IV PTSD symptoms B1-B5) and avoidance (C1-C2) symptom clusters. However, the models differ in their organization of a negative affect cluster, conceptualized as numbing in the King model (DSM-IV PTSD symptoms C3-C7) and dysphoria in the Simms model(C3-C7 and D1-D3). They also differ in their organization of a hyperarousal cluster (King model: DSMIV PTSD symptoms D1-D5; Simms model: D4-5). Both models have been supported in a large meta analytic study (Yufik & Simms, 2010).

State Trait Anger Expression Inventory-2 (STAXI). The STAXI (Spielberger, 1999) is a self-report instrument which assesses the experience, expression, and control of anger. Items are measured on a 4-point Likert scale. For this study we used the 15-item STAXI state anger and 10-item trait anger total scale scores for analyses. The STAXI anger control and behavioral expression of anger scales were not included due to battery length concerns. State trait anger expression inventory-2 (STAXI) state anger measures the intensity of anger a person is currently experiencing, as well as the intensity of current feelings related to verbal and physical expression of anger. STAXI trait anger assesses perceptions of general anger responses experienced over time, as well as the frequency that angry feelings are experienced in situations that involve frustration and negative evaluation. The STAXI has good concurrent validity and has been used extensively in veteran samples (Spielberger, 1999).
**Statistical Analysis**

To explore mediation of the PTSD-anger relationship by depression, we conducted a series of path analyses. Path analysis is a subcategory of structural equation modeling, the main difference being that structural equation modeling involves use of latent variables, while path analysis uses observed variables that are directly measured (Schumacker & Lomax, 2004). The study’s sample size was not sufficient for structural equation modeling, but was appropriate for path analysis (Kline, 1998). This approach also provides a lower standard error and more power than traditional regression approaches to mediation (Iacobucci, 2008). All analyses were conducted using MPlus, Version 6.1 (Muthén & Muthén, 2010) and a small amount of missing data (2.65%) was addressed using maximum likelihood and robust weighted least squares estimators.

There is significant debate about what constitutes a mediated relationship (Mathieu & Taylor, 2006; Shrout & Bolger, 2002). For this study, we defined mediation as occurring when two variables are significantly related to each other (i.e. the total effect), both are significantly related to a mediator, the product of the paths to the mediator (i.e. the indirect effect) is significant, and the relationship between the variables is attenuated when controlling for the mediator (Kenny, 2012). However, if two variables are not significantly related to each other but there are significant pathways from both variables to the mediator, the relationship is termed an indirect effect (Mathieu & Taylor, 2006). Although less convincing than mediation in terms of establishing causality (Shrout & Bolger, 2002), an indirect effect can still establish pathways of interest (Mathieu & Taylor, 2006).
We used the recommendations of Kenny (2012) to define complete and partial mediation, as well as establish effect size guidelines. Complete mediation occurs when a significant relationship is reduced to zero by inclusion of a mediator in analyses. Partial mediation occurs when a significant relationship is reduced in size by the inclusion of a mediator, but is still greater than zero (Kenny, 2012). Effect sizes in mediation analysis can be measured by the indirect effect, and were defined for dimensional mediators (i.e. numbing and dysphoria clusters) as .01 for small effects, .09 for medium effects, and .25 for large effects. For categorical mediators (i.e. diagnosis of MDD), effect sizes were defined as .02 for small effects, .15 for medium effects, and .40 for large effects.

In our path analyses, we first examined whether the presence of a diagnosis of MDD mediated the relationships between total PTSD symptom severity and state and trait anger. Following this, we conducted path analyses exploring whether a diagnosis of MDD mediated the relationships between each PTSD symptom cluster not related to depression (i.e. reexperiencing, avoidance, and hyperarousal) and state and trait anger. Finally, these analyses were repeated using the PTSD numbing cluster as the mediator, and also using the PTSD dysphoria cluster as the mediator.

Results

For analyses using MDD diagnostic status as a mediator, the standardized pathway estimates, as well as total and indirect effects, are listed in Table 1. MDD partially mediated the relationship between total PTSD symptom severity and state anger, as well as the relationship
between reexperiencing and state anger. Avoidance and hyperarousal had significant indirect effects on state anger through MDD. Effect sizes were small. There was no significant mediation or indirect effects for any of the analyses involving MDD and trait anger.

The standardized pathway estimates, total effects, and indirect effects of analyses using numbing and dysphoria as mediators are listed in Table 2. Numbing partially mediated the relationships between all PTSD symptom clusters (reexperiencing, avoidance, hyperarousal) and trait anger. Avoidance and hyperarousal also had significant indirect effects on state anger through numbing. Dysphoria partially mediated the relationships between reexperiencing and trait anger, as well as between hyperarousal and trait anger. Effect sizes were small to medium.

**Discussion**

Past research has indicated that anger, within the context of PTSD, is associated with many negative outcomes (Teten et al. 2010; Ouimette et al. 2004) and high overall PTSD symptom severity (Novaco and Chemtob 2002). A number of theories (Feeny et al. 2000; Chemtob et al. 1997) have attempted to explain the relationship between PTSD and anger; however, no study to date has examined the mediating influence of depression. Based on the Cognitive Neoassociationistic Model (Berkowitz 1990), this study evaluated depression as a mediator of the PTSD and anger relationship. Analyses were conducted with PTSD defined as total symptom severity, as well as PTSD reexperiencing, avoidance, and hyperarousal clusters (King et al. 1998; Simms et al. 2002). Depression was defined as both diagnosis of MDD and as the numbing and dysphoria clusters of PTSD (Simms et al. 2002).
Results partially supported our first and second hypotheses, as MDD did not mediate the relationship between total PTSD symptom severity and trait anger, or the relationships between the reexperiencing, avoidance, and hyperarousal clusters and trait anger. However, MDD did partially mediate the relationship between total PTSD symptom severity and state anger, as well as between the reexperiencing cluster and state anger, and the avoidance and hyperarousal clusters both had significant indirect effects on state anger through MDD. Findings also partially supported our third hypothesis, as numbing symptoms partially mediated all relationships between PTSD clusters and trait anger, and there were also indirect effects of PTSD clusters on state anger through numbing symptoms. Dysphoria demonstrated less of an influence, and only partially mediated two relationships between PTSD clusters and trait anger.

Taken together, this pattern of findings suggests that a diagnosis of current MDD has a significant influence on the relationship between PTSD and state anger, while the numbing and dysphoria clusters of PTSD seem to primarily influence trait anger. These findings are consistent with the Cognitive Neoassociationistic Model, which posits that unpleasant events (i.e. trauma and PTSD symptoms) result in negative affect (i.e. depression), which in turn activates networks with associative links to anger. Based on this theory, temporarily elevated levels of negative affect should result in more activation of associative networks and higher levels of transient anger, while longer periods of lower-level negative affect should result in prolonged anger activation of lesser intensity. This study produced a consistent pattern of findings, as current MDD consists of a transitory, discrete period of heightened negative affect, and partially mediates the relationship between PTSD and state anger. It did not, however, mediate the
relationship between PTSD and trait anger. Conversely, numbing and dysphoria symptoms are clusters of PTSD that are present during the often chronic course of the disorder (Wittchen et al. 2009), and partially mediate the relationship between other PTSD symptom clusters and trait anger. The indirect effect of numbing on several PTSD cluster-state anger relationships also suggests that numbing symptoms can, in some cases, become severe enough to substantially elevate current anger levels.

The implication of these findings is that in veterans with PTSD, posttraumatic symptomatology can increase levels of negative affect and led to depressive symptoms reaching clinical thresholds for MDD, which in turn increases the probability that cognitive structures associated with anger will be activated and result in increased levels of current anger. Even if PTSD symptoms do not result in depressive symptoms reaching the clinical threshold, they can lead to increased trait anger levels through the mediating influence of numbing and dysphoria symptoms. This is not to imply certain PTSD clusters always lead to other clusters in a sequential fashion, but rather to suggest one pathway which can result in increased anger. The results also suggest modalities that treat both PTSD and depression (e.g. Farchione et al. 2012; Gros, Price, Strachan, Yuen, Milanak, & Acierno, in press) should be very effective in reducing anger through targeting both the direct and indirect pathways that lead to anger.

One unexpected finding of this study was that the dysphoria cluster had less of an influence on PTSD-anger relationships than did the numbing cluster. This is somewhat surprising given that the only difference between clusters is that dysphoria contains three extra symptoms, one of which is the PTSD anger item which was removed from analyses. However,
one of these items (i.e. sleep disturbance) has been shown to load more strongly onto PTSD than MDD, and all items of the numbing cluster load more strongly onto MDD (Gros et al. 2010). The stronger mediating and indirect effects of the numbing cluster could therefore be attributed to the fact that it is a purer measure of negative affect and depressive symptoms.

This study has a number of limitations that must be noted. The study’s small sample size may have limited its power to detect significant relationships. Additionally, rates of comorbid anxiety disorders, which highly co-occur with depression (Brown et al. 2001), were not controlled for in these analyses. All participants in this study were volunteers in a clinical research trial, which may limit generalizability. In addition, the causal direction of relations between PTSD symptoms, depression and anger cannot be definitely determined without longitudinal research designs. Furthermore, there are several limitations involving this study’s anger indicators. Anger measures were administered during baseline assessment sessions, which sometimes involved assessment of PTSD. As such, STAXI state findings could reflect frustration with the assessment procedure, or be a result of discussing traumatic events and PTSD symptoms. Additionally, we were not able to assess STAXI scales related to anger control or behavioral expression.

In conclusion, this study found preliminary evidence that comorbid MDD partially mediates the relationship between PTSD and state anger, while numbing and dysphoria clusters partially mediate the relationship between other PTSD clusters and trait anger. Future research should explore whether evidence-based treatment that addresses depressive symptoms in individuals with PTSD also reduces associated anger levels. Additionally, research should
explore these relationships in non-military populations, with other kinds of potentially traumatic events, across gender, and as across various mood and anxiety disorders.
Original Paper 1 Tables

Table 1  Standardized path estimates, total effects, and indirect effects for analyses with current MDD as the mediator

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XY' indicates the relationship between X and Y while controlling for M
SE standard error, Tot Eff total effect, Indir Eff indirect effect, PTSD total PTSD symptoms, MDD current major depressive disorder, State State-Trait Anger Expression Inventory-II state scale; Trait State-Trait Anger Expression Inventory-II trait scale; Reexp PTSD reexperiencing cluster; Avoid PTSD avoidance cluster; Hyper PTSD hyperarousal cluster

* p < .05
** p < .01

Table 2  Standardized path estimates, total effects, and indirect effects for analyses with numbing and dysphoria clusters as mediators

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SE standard error, Tot Eff total effect, Indir Eff indirect effect, PTSD total PTSD symptoms, State-State-Trait Anger Expression Inventory-II state scale, Trait State-Trait Anger Expression Inventory-II trait scale, Reexp PTSD reexperiencing cluster; Avoid PTSD avoidance cluster; Hyper PTSD hyperarousal cluster; Numb PTSD numbing cluster; Dysp PTSD dysphoria cluster

* p < .05
** p < .01
Erratum

A veteran with sub-threshold PTSD was erroneously included in our sample of veterans with a current PTSD diagnosis. Upon correcting the error and rerunning analyses, we found that while the magnitudes of relationships among most variables were not meaningfully altered, some statistical results were slightly changed (see Tables 1, 2). In the new analyses, PTSD numbing no longer partially mediates the relationships between all PTSD clusters and trait anger. Instead, numbing partially mediates the relationships between the reexperiencing cluster and trait anger, and between the avoidance cluster and trait anger. Hyperarousal now has a significant indirect effect on trait anger through numbing symptoms. Additionally, PTSD dysphoria symptoms now partially mediate the relationships between all PTSD clusters and trait anger.

The corrected results continue to support the main findings of our study; however, contrary to our original discussion, we can now conclude that numbing and dysphoria have a similar impact on PTSD cluster–anger relationships.
Corrected Paper 1 Tables

Table 1  Standardized path estimates, total effects, and indirect effects for analyses with current MDD as the mediator

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XY* indicates the relationship between X and Y while controlling for M
SE standard error, Tot Eff total effect, Indir Eff indirect effect, PTSD total PTSD symptoms, MDD current major depressive disorder, State State–Trait Anger Expression Inventory-II state scale, Trait State–Trait Anger Expression Inventory-II trait scale, Reexp PTSD reexperiencing cluster, Avoid PTSD avoidance cluster, Hyper PTSD hyperarousal cluster

* p < .05
** p < .01

Table 2  Standardized path estimates, total effects, and indirect effects for analyses with numbing and dysphoria clusters as mediators

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<th>XY* SE</th>
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SE standard error, Tot Eff total effect, Indir Eff indirect effect, State State–Trait Anger Expression Inventory-II state scale, Trait State–Trait Anger Expression Inventory-II trait scale, Reexp PTSD reexperiencing cluster, Avoid PTSD avoidance cluster, Hyper PTSD hyperarousal cluster, Numb PTSD numbing cluster, Dysp PTSD dysphoria cluster

* p < .05
** p < .01

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Appendix: Cronbach's Alphas for Paper 1's Measures

PCL Total: .89
PCL Reexperiencing: .82
PCL Avoidance: .59
PCL Numbing: .72
PCL Hyperarousal: 76

STAXI State Anger: .96
STAXI Trait Anger: .88
References


treatment-seeking Veterans. *Journal of Nervous and Mental Disease, 198*(12), 885-890. doi: 10.1097/NMD.0b013e3181fe7410


Paper #2: Impact of Comorbid Depression on Quality of Life in Male Combat Veterans with Posttraumatic Stress Disorder

Please note that portions, but not the entirety, of this study are currently in press in *Journal of Rehabilitation Research & Development*, an Open-Access journal freely available in the public domain. Citation for the article is:

Abstract

For Veterans with posttraumatic stress disorder (PTSD), depression is a highly comorbid condition. Both conditions have been associated with decreased quality of life, and research suggests that comorbid PTSD and depression may result in worse quality of life than PTSD alone. However, research is needed to elucidate the impact of comorbidity on a broader variety of quality of life domains. In this study, we used baseline data of 158 male combat Veterans taking part in a PTSD treatment trial. We examined the unique relationships between quality of life domains and PTSD symptom clusters, major depressive disorder (MDD) diagnosis, and self-reported depressive symptoms, and explored the mediating role of depressive symptoms on PTSD-quality of life relationships. Veterans with comorbid PTSD-MDD reported significantly worse satisfaction-related quality of life than those with PTSD alone, although this finding was largely attributable to PTSD numbing symptoms. Subsequent analyses comparing the impact of numbing symptoms to depressive symptoms revealed that depression exerted a stronger influence, although numbing symptoms were still uniquely associated with quality of life. Depressive symptoms partially and fully mediated many of the PTSD-quality of life relationships. We discuss implications for treatment and the need to address negative affect in Veterans with PTSD.
Introduction

Research has demonstrated that for Veterans with posttraumatic stress disorder (PTSD), depression is a highly comorbid condition, with a recent large meta-analysis reporting a rate of 52% across military and civilian samples (Rytwinski, Scur, Feeny, & Youngstrom, 2013). The underlying reason for this high degree of comorbidity is unclear. One theory postulates that the disorders represent differing symptom manifestations of an underlying stress construct, a position supported by the common diatheses of the two disorders (O’Donnell, Creamer, & Pattison, 2004). Researchers have also highlighted the lack of specificity of PTSD’s symptoms as a possible cause, and a recent study with a Veteran sample found that a subset of PTSD symptoms related to numbing loaded more strongly on a depression factor than on PTSD. Furthermore, in that study, individuals endorsing these numbing-related symptoms reported significantly more comorbid PTSD and depression and significantly higher depressive symptom severity (Gros, Simms, & Acierno, 2010).

While there is no definitive explanation for the high degree of depression comorbidity observed in individuals with PTSD, there is reason to suspect that it may be associated with reduced Veteran quality of life, which has been conceptualized as a person’s physical, mental, and social well-being (World Health Organization, 1948). Research has shown that PTSD has a deleterious impact on Veteran life quality, as Veterans with PTSD report increased rates of unemployment due to disability (Magruder et al., 2004), decreased social (Rona et al., 2009) and occupational functioning (Breslau, Lucia, & Davis, 2004), and lower life satisfaction (Lunney & Schnurr, 2007). There is also literature documenting the significant negative impact of
depression on quality of life. Similar to PTSD, depression has been associated with financial problems (Elbogen, Johnson, Wagner, Newton, & Beckham, 2012) and decreased life satisfaction (Rapaport, Clary, Fayyad, & Endicott, 2005).

Several studies suggest that individuals with PTSD who also have comorbid depression have worse quality of life than those with PTSD alone. Research in non-Veteran samples has found that PTSD and depression comorbidity is associated with significantly worse major role functioning than PTSD alone (Blanchard, Buckley, Hickling, & Taylor, 1998), and significantly worse social and occupation functioning than is associated with only PTSD or neither disorder (Momartin, Silove, Manicavasagar, & Steel, 2004). The few studies conducted with Veterans have produced a similar pattern of findings. In a study with Israeli Veterans of the 1982 Lebanon War, individuals with comorbid PTSD, depression, and anxiety reported worse psychosocial functioning than individuals with only PTSD (Ginzburg, Ein-Dor, & Solomon, 2010). Additionally, in a large sample of Korean War Veterans, individuals with comorbid PTSD and depression reported poorer satisfaction with their physical and psychological health, social relationships, and environment compared to Veterans with only PTSD, only depression, or neither disorder (Ikin, Creamer, Sim, & McKenzie, 2010). These studies provide preliminary evidence that PTSD and comorbid depression can have a significantly more detrimental impact on Veteran quality of life than PTSD alone. However, further research is needed to elucidate this impact, given the multifaceted nature of quality of life.

Quality of life has been operationalized in a variety of ways and using various measures, with researchers advocating the importance of assessing both subjective life satisfaction (Frisch,
as well as more objective indices (Gladis, Gosch, Dishuk, & Crits-Christoph, 1999). In their review of the quality of life literature for Iraq and Afghanistan War Veterans, Schnurr and colleagues (Schnurr, Lunney, Bovin, & Marx, 2009) adopted a framework focusing on subjective and objective indices across three domains: social-material conditions, functioning, and satisfaction (Gladis et al., 1999). Social-material quality of life includes areas such as employment, income, and marital status. Functioning involves an individual’s role performance, or their ability to function at work, relationships, and in different life domains. Satisfaction is concerned with individual well-being and subjective appraisal of life quality (Schnurr et al., 2009). This tripartite approach shares similarities to other conceptualizations of quality of life (Mogotsi, Kaminer, & Stein, 2000), as well as the World Health Organization’s definition of the construct (World Health Organization, 1948). Existing studies have examined the impact of comorbid PTSD and depression on these domains (Ginzburg et al., 2010; Ikin et al., 2010), but to our knowledge few studies to date have explored indicators of all three domains in a single study utilizing a Veteran sample.

Additionally, further empirical investigation is needed to disentangle the unique contributions of PTSD and depression to the decreased quality of life associated with comorbidity of the disorders (Schnurr et al., 2009). One study involving OEF/OIF combat Veterans found that PTSD and depression both independently contributed to mental health-related quality of life, with depression predicting more variance than PTSD (Pittman, Goldsmith, Lemmer, Kilmer, & Baker, 2012). In another study of combat Veterans, depressive symptoms had a significant impact on satisfaction-related quality of life, although PTSD was not
significantly related (Rauch et al., 2010). Likewise, in a sample of Veterans with PTSD, researchers found that depression predicted overall physical health-related quality of life, while PTSD did not, although analysis of the quality of life subscales provided a more nuanced picture of the findings. While depressive symptoms predicted physical and role functioning scales, both depressive symptoms and PTSD independently predicted general health and bodily pain scales, and depression and PTSD interacted to predict vitality and social functioning scales (Aversa et al., 2012). Taken together, the existent research suggests that PTSD and depression make unique contributions to decreased quality of life, but that depression exerts a stronger influence. Further research is needed however, especially to examine possible differences in the impact of depression and individual PTSD symptom clusters. Studies have found that various PTSD symptom clusters impact quality of life (Kuhn, Blanchard, & Hickling, 2003; Rona et al., 2009), perhaps most notably the PTSD numbing symptom cluster (Kuhn et al., 2003; Lunney & Schnurr, 2007; Schnurr & Lunney, 2008) identified in the emotional numbing four-factor structure (King, Leskin, King, & Weathers, 1998) and later incorporated into the structure of the DSM-5 diagnostic criteria for PTSD (American Psychiatric Association, 2013). Because the numbing cluster shares significant overlap with depression (Gros, Price, Magruder, & Frueh, 2012; Gros et al., 2010), understanding its relationship with quality of life, and how this resembles or differs from that of the other symptom clusters, is essential to understanding the unique contributions of PTSD and depression to decreased quality of life.

Empirical attention is also needed to examine potential pathways that may lead to Veteran quality of life problems associated with comorbid PTSD and depression, as improved
understanding of such pathways could facilitate more targeted treatment planning and result in improved clinical outcomes for Veterans. One possible pathway involves mediation of PTSD and quality of life relationships by comorbid depression. In a study of motor vehicle accident survivors, depressive symptoms mediated the relationship between PTSD and subjective appraisal of quality of life (Gudmundsdottir, Beck, Coffey, Miller, & Palyo, 2004). Also, in a sample of OEF/OIF Veterans, significant relationships between PTSD symptoms and role functioning problems were rendered non-significant when controlling for depression symptoms (Erbes, Westermeyer, Engdahl, & Johnsen, 2007). However, more research is needed to examine the mediational impact of comorbid depression across a variety of quality of life indicators.

The purpose of the current study was to conduct exploratory analyses on the impact of PTSD and comorbid depression on social-material, functioning, and satisfaction-related quality of life domains in a sample of male combat Veterans with PTSD. We used the *Diagnostic and Statistical Manual of Mental Disorders* (DSM)-IV PTSD symptom clusters of the emotional numbing model (King et al., 1998) which share significant overlap with the new DSM-5 diagnostic criteria (American Psychiatric Association, 2013). For all domains where comorbid depression was significantly associated with poorer quality of life, we explored the unique contributions of PTSD symptom clusters and depression. Furthermore, we explored the mediating role of comorbid depression on the relationships between PTSD clusters and quality of life domains when the unique associations of the disorders suggested the possibility of mediation. Finally, while we initially defined depression by categorical diagnosis of major
depressive disorder (MDD), we also conducted post-hoc analyses operationalizing depression using the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996), which provided a dimensional measure of depressive symptoms.

We predicted that individuals with comorbid PTSD and MDD would have significantly worse quality of life than individuals with PTSD alone across all social-material, functioning, and satisfaction-related quality of life domains. Given the research connecting a variety of PTSD symptom clusters to poorer quality of life, we predicted that both PTSD symptom clusters and comorbid MDD would uniquely influence each quality of life domain. We also predicted that comorbid MDD would play a mediating role for many of the PTSD and quality of life relationships of the study.

Method

Participants

We drew data for this study from the baseline assessments of a clinical trial evaluating the effectiveness of the telehealth modality for delivering Cognitive Processing Therapy (Resick, Monson, & Chard, 2007) to combat Veterans with PTSD (Morland et al., 2014); this included baseline data from a pilot cohort (Morland, Hynes, Mackintosh, Resick, & Chard, 2011). Data were collected at Veterans Affairs clinics, and all participants provided written informed consent prior to participation. Participants in the current study consisted of 158 male combat Veterans with PTSD, and the inclusion criterion for the current study was a diagnosis of current PTSD based on the Clinician Administered PTSD Scale for DSM-IV (CAPS; Blake et al., 1995). Participants were primarily White (n=70, 45.2%), Pacific Islander (n=31, 20.0%), or Asian
American (n=23, 14.8%). They mainly served in the Vietnam (n=100, 63.3%), OEF/OIF (n=47, 29.7%), and Operations Desert Storm/Shield (n=24, 15.2%) theatres, and in the Army (n=109, 69.0%), Marines (n=22, 13.9%), Navy (n=21, 13.3%), and Air Force (n=14, 8.9%). Because some Veterans served in multiple war eras, or in multiple military branches, numbers and percentages are larger than the total sample size.

Measures

Participants completed psychometrically-sound self-report measures and clinician-administered interviews to assess PTSD and depression symptoms, as well as quality of life domains. For clinician-administered interviews, masters and doctoral-level assessors were provided initial training in interview administration and required to demonstrate sufficient reliability and fidelity before conducting independent assessments. All assessors were also provided ongoing supervision to maintain fidelity to assessment protocols, and a random 20% of CAPS protocols in the parent study were independently reviewed for fidelity and reliability.

Posttraumatic Stress Disorder

*The Clinician Administered PTSD Scale for DSM-IV.* The CAPS is a clinician-administered interview for diagnosis of DSM-IV PTSD (Blake et al., 1995). The CAPS was used in this study to establish diagnosis of PTSD with the “1/2” rule, in which symptoms must have occurred minimally within the last month and be of at least a moderate intensity (Weathers, Ruscio, & Keane, 1999). The CAPS has good divergent and convergent validity, and strong reliability (Blake et al., 1995).
Posttraumatic Stress Disorder Checklist (PCL). The PCL (Weathers, Litz, Herman, Huska, & Keane, 1993) is a 17-item self-report questionnaire that uses a 5-point Likert scale to measure severity of the 17 DSM-IV PTSD symptoms. The PCL-Military Version (PCL-M) was used with the first two cohorts of the parent study, and the PCL-Stressor Specific Version (PCL-S) was utilized in all other cohorts. The scales have nearly identical items but differ in their instructions, and research with a military sample suggests that changes to PCL-S instructions do not affect reporting of symptoms (Riviere et al., 2011). There were no significant differences found between PCL-M and PCL-S scores in this sample when controlling for depression (p>.05).

For analyses involving PTSD symptom clusters, we used the emotional numbing four-factor model (King et al., 1998). The model has reexperiencing (B1-B5) and hyperarousal (D1-D5) clusters identical to those in the DSM-IV (American Psychiatric Association, 2000). However this model partitions the DSM-IV avoidance symptoms into two separate clusters: an avoidance cluster (C1-C2) and an emotional numbing cluster (C3-C7) which primarily captures negative affect. The model has been supported in a large meta-analytic study (Yufik & Simms, 2010).

Research has demonstrated that the PCL has excellent convergent validity with other measures of PTSD (rs range from .77 to .93) (Orsillo, 2002. Chapter 19, Measures for acute stress disorder and posttraumatic stress disorder). Additionally, the PCL has strong test-retest reliability and internal consistency (Orsillo, 2002. Chapter 19, Measures for acute stress disorder and posttraumatic stress disorder). Internal consistency for the PCL total score in the current sample was good (α=.90), and the reexperiencing (α=.84), numbing (α=.73), and hyperarousal
(α=.76) clusters all evidenced acceptable reliability. The internal consistency of the avoidance cluster (α=.65) was somewhat low, likely because the cluster is only composed of two items.

**Depression**

*Structured Clinical Interview for DSM-IV Axis I Disorders (SCID).* The SCID is a clinician-administered diagnostic interview for DSM-IV Axis I disorders (First, Spitzer, Gibbon, & Williams, 2002), and has strong agreement with multi-informant based diagnoses (Basco et al., 2000). In the parent study, an abbreviated version of the SCID was given, and only data on diagnosis of current MDD (i.e. currently experiencing a major depressive episode) was used in the present study. The rate of comorbid MDD in this sample (33%) was smaller than the reported rate of a recent meta-analysis (Rytwinski et al., 2013), possibly a result of stringent application of the DSM-IV criteria.

*Beck Depression Inventory II (BDI-II).* Depressive symptom severity was assessed for post-hoc analyses using the BDI-II (Beck et al., 1996), a 21-item self-report inventory of depression symptoms. Items are rated on a 4-point Likert scale, and higher total scale scores are indicative of greater symptom severity. The BDI-II has been used frequently in Veteran samples (Walter, Barnes, & Chard, 2012) and possesses strong validity and reliability (Beck et al., 1996). Internal consistency of the BDI-II total score in the current sample was good (α=.93).

**Quality of Life**

Quality of life was assessed across the domains of social-material conditions, functioning, and satisfaction (see Schnurr et al., 2009). Quality of life related to social-material conditions was operationalized in this study as marital status and employment income, both of
which came from the parent study’s demographic questionnaire. Data on employment income was drawn from an open-ended question inquiring about current sources of income. For this study’s analyses, we defined these domains using dichotomous variables (i.e. married/not married; employment income/no employment income).

*Short-Form 12 Health Survey (SF-12):* Functioning was assessed using the SF-12, version 1 (Ware Jr, Kosinski, & Keller, 1996), a brief 12-item measure which uses both dichotomous and Likert-scale items to assess role functioning and health-related quality of life. The SF-12 asks questions about physical health limitations, the degree to which pain interferes with activities, and the impact of emotional problems on activities during the past month. The measure produces two summary scores, the physical component summary (PCS) and the mental component summary (MCS), which respectively assess the degree to which physical health and mental health problems have interfered with role functioning. The SF-12 has good reliability and validity (Ware Jr et al., 1996) and has been used in military samples (McKenzie et al., 2004).

*Quality of Life Inventory (QOLI).* Satisfaction was assessed using the QOLI (Frisch, 1994), a psychometrically valid and reliable 32-item self-report inventory which assesses subjective life quality across 16 areas of life (Frisch, Cornell, Villanueva, & Retzlaff, 1992). Each area of life on the QOLI is measured with two items, a three-point Likert scale which assesses importance and a 6-point Likert scale which assesses satisfaction. Importance and satisfaction ratings are multiplied to produce a weighted satisfaction score for each area of life.

For this study, we organized areas of life assessed by the QOLI into four domains that have been supported in a large factor analytic study with a male Veteran sample (Lunney &
Schnurr, 2007). These domains include achievement, self-expression, relationships, and surroundings. The achievement domain consists of the areas of health, self-esteem, goals and values, money, and work. The self-expression domain is composed of play, creativity, and learning areas of life, while the relationships domain consists of helping, love, friends, children, and relatives. Finally, the surroundings domain contains home, neighborhood, and community areas of life. Weighted satisfaction scores for the areas of life were averaged across each domain to produce a domain score, a metric with good internal consistency (Schnurr & Lunney, 2008). In the current study, the internal consistency across all domain scores was acceptable (achievement, $\alpha=.81$; self-expression, $\alpha=.76$; relationships, $\alpha=.76$; surroundings, $\alpha=.79$).

**Statistical Analyses**

To determine if Veterans with comorbid PTSD and depression demonstrated worse quality of life than those with PTSD only, we conducted a series of linear and logistic regressions between comorbid MDD status (PTSD-MDD vs. PTSD only) and quality of life indicators, including marital status and employment income, the SF-12’s PCS and MCS scales, and the four QOLI domains (achievement, self-expression, relationships, surroundings). For all quality of life indicators for which comorbid MDD was significantly and negatively related to quality of life, subsequent analyses were conducted to better understand the unique contributions of PTSD and depression. Each indicator was separately regressed on to each PTSD symptom cluster (reexperiencing, avoidance, numbing, and hyperarousal). Then, in cases where a PTSD cluster was significantly and negatively related to the quality of life indicator, we conducted a series of simultaneous regressions. We first regressed the quality of life indicator on the PTSD
cluster and comorbid MDD simultaneously to gauge whether inclusion of both the cluster and comorbid MDD would attenuate either variable’s significant relationship with the quality of life indicator. We then regressed the quality of life indicator on to all the PTSD clusters simultaneously to determine if the relationship remained significant when accounting for other PTSD symptoms. Finally, we regressed the quality of life indicator on to all PTSD symptom clusters and comorbid MDD simultaneously, and compared this model’s $R^2$ value to the $R^2$ of the regression not including comorbid MDD. By doing this, we were able to examine the unique variance accounted for by all the PTSD clusters, as well as the incremental variance accounted for by comorbid MDD.

The possibility of mediation of PTSD cluster and quality of life relationships by comorbid depression was also examined. There is significant debate about what constitutes a mediated relationship (Mathieu & Taylor, 2006; Shrout & Bolger, 2002). For this study, we defined mediation as occurring when an independent and dependent variable were significantly related to each other (i.e. the total effect), the pathway from the independent variable to the mediator was significant, the pathway from the mediator to the dependent variable was significant when controlling for the independent variable, the product of these paths to the mediator (i.e. the indirect effect) was significant, and the total effect was attenuated when controlling for the mediator (Kenny). We used the recommendations of Kenny (Kenny) to define complete and partial mediation. Complete mediation occurred when a significant relationship was reduced to zero by inclusion of a mediator in analyses. Partial mediation occurred when a significant relationship was reduced in size by the inclusion of a mediator, but
was still greater than zero (Kenny). Mediation was examined using path analysis, a special case of structural equation modeling that utilizes observed variables and does not account for measurement error through use of latent variables (Schumacker & Lomax, 2004). Many independent variables in this study were converted to single-item latent variables, not to account for measurement error, but only for the purposes of addressing missing data; this technique does not change interpretation of model parameters (Enders, 2010).

Mediation was to be examined in cases where three preliminary conditions were met. These conditions were that the PTSD cluster was significantly associated with a quality of life indicator in the separate regression, comorbid depression was significantly associated with the indicator in the simultaneous regression, and the inclusion of depression in simultaneous analysis decreased the strength of relationship between the PTSD symptom cluster and the quality of life indicator. However, because of unexpected findings, we did not conduct our originally planned mediation analyses. Initial results suggested that decreased quality of life was primarily attributable to PTSD numbing symptoms, and not comorbid MDD. However, the ability to draw this conclusion was limited by differences in reporter (self-report vs. clinician) and level of measurement (categorical vs. dimensional) across PTSD and depression measures. As such, we conducted post-hoc analyses in which we repeated all depression-related regression and mediation analyses using the BDI-II. Because the BDI-II and PCL are both self-report, dimensional measures, this allowed us to reduce the possible influence from method variance and draw more accurate conclusions about the unique contributions of PTSD and depression to decreased quality of life.
All regression and path analyses were conducted with MPlus, Version 6.1 utilizing maximum likelihood estimation to handle missing data (8.27%). Descriptive statistics and reliabilities were calculated using SPSS, Version 19.

Results

Prediction of Quality of Life by Comorbid MDD

Table 1 presents descriptive statistics for the comorbid PTSD-MDD and PTSD only groups, as well as standardized results from regressions between comorbid MDD and the different quality of life indicators. When compared to Veterans with PTSD only, Veterans with comorbid PTSD and MDD evidenced significantly worse satisfaction-related quality of life on all four QOLI domains (achievement, self-expression, relationships, and surroundings). Comorbid MDD did not significantly predict differences in either social-material conditions (marital status, employment income) or functioning (SF-12 PCS and MCS scales).

Separate and Simultaneous Regressions Involving PTSD Symptom Clusters

The top portion of Table 2 contains standardized results from separate regressions between each PTSD symptom cluster and each QOLI domain. All four PTSD symptom clusters were significantly and negatively associated with the achievement and self-expression domains, and the avoidance, numbing, and hyperarousal clusters were significantly negatively associated with the relationships domain. Numbing was the only cluster significantly and negatively associated with the surroundings domain.

The middle portion of Table 2 displays the standardized results from simultaneous regressions involving comorbid MDD and individual PTSD symptom clusters. For the
achievement domain, all PTSD symptom clusters retained their significant negative associations when analyzed together with comorbid MDD, while comorbid MDD retained its significant negative associations when analyzed with the reexperiencing, avoidance, and hyperarousal clusters. Comorbid MDD lost its significant negative relationship with achievement when analyzed jointly with the numbing cluster. Analyses involving the self-expression domain produced a pattern of significant relationships identical to those of the achievement domain. In addition, comorbid MDD lost its significant negative association with the relationship domain when analyzed with PTSD clusters, and only the numbing cluster retained it significant negative association when analyzed with comorbid MDD. There were no significant associations in simultaneous analyses involving the surroundings domain.

The bottom portion of Table 2 presents standardized results from the regressions containing all PTSD symptom clusters regressed simultaneously. The only PTSD symptom cluster that retained significance was numbing, which remained significantly negatively associated with the achievement, self-expression, and relationship domains. These relations remained significant even when comorbid MDD was entered into analyses; comorbid MDD itself evidenced no significant relationships. Examination of model $R^2$ values indicated that a significant amount of variance was accounted for in analyses involving the achievement, self-expression, and relationship domains, and the inclusion of comorbid MDD failed to substantially increase the amount of variance accounted for in analyses.
Post-Hoc Analyses

Results from post-hoc regression analyses using the BDI-II are displayed in Table 3. There were significant negative relationships between the BDI-II and all satisfaction-related quality of life domains in the separate analyses. Simultaneous analyses revealed a striking pattern: the BDI-II retained almost all its significant relationships when regressed jointly with individual PTSD clusters, while the reexperiencing, avoidance, and hyperarousal clusters lost significance across all quality of life domains when analyzed together with the BDI-II. The PTSD numbing cluster, however, retained significant negative relationships with the achievement, self-expression and relationship domains. Simultaneous regressions between all the PTSD clusters and the BDI-II produced similar findings: the BDI-II was significantly and negatively related to the achievement and surroundings domains, and both the BDI-II and PTSD numbing cluster had significant negative associations with the self-expression and relationship domains. There were no significant relationships between other PTSD clusters and quality of life domains in these simultaneous analyses.

The results of the mediation analyses are displayed in Table 4. Comorbid depressive symptoms, as measured by the BDI-II, significantly and partially mediated many of the relationships of the study. The BDI-II partially mediated relationships between all PTSD symptom clusters (reexperiencing, avoidance, numbing, and hyperarousal) and the achievement domain, and between all clusters and the self-expression domain. The BDI-II also partially mediated the relationships between the avoidance cluster and the relationship domain, the
hyperarousal cluster and the relationship domain, and fully mediated the relationship between the numbing cluster and the surroundings domain.

**Discussion**

Previous research has demonstrated that PTSD can have a negative impact on quality of life for Veterans (Magruder et al., 2004), and that the presence of comorbid depression can lead to further life quality problems (Ginzburg et al., 2010). The current study adds to this literature by exploring the impact of comorbid depression on social-material conditions, functioning, and satisfaction-related quality of life, the unique contributions of each PTSD symptom cluster and comorbid depression to quality of life impairment, and the mediating role of comorbid depression on PTSD-quality of life relationships in a sample of male combat Veterans with PTSD.

Our hypotheses were partially confirmed, as our study supported past research documenting, in individuals with PTSD, the detrimental impact of comorbid depression on satisfaction with relationships and the surrounding environment (Ikin et al., 2010) and expanded upon past research by finding negative relationships with achievement and self-expression domains. However, we also found that comorbid depression was not significantly related to social-material condition-related quality of life, nor was it associated with diminished role functioning, the latter of which is in contrast to existing research (Blanchard et al., 1998). When exploring the unique contributions of depression and PTSD to reduced quality of life, PTSD numbing symptoms initially seemed to account for the association with decreased quality of life
across all satisfaction-related domains. However, a very different pattern of findings emerged in post-hoc analyses when operationalizing depression using the self-report, dimensional BDI-II instead of the clinician-administered, categorical MDD SCID diagnosis. Comorbid depression symptoms had more negative associations with decreased quality of life, and constituted the sole significant predictor of quality of life in the achievement and surrounding domains when considered jointly with all PTSD clusters. Furthermore, the BDI-II also predicted decreased quality of life in the self-expression and relationship domains, and the only PTSD cluster that demonstrated significant relationships with these domains was numbing.

These findings were consistent with past research suggesting that depression has a stronger influence on decreased quality of life than does PTSD (Aversa et al., 2012; Pittman et al., 2012). Past studies have also produced similar findings regarding the importance of the PTSD numbing cluster to quality of life in Veterans with PTSD (Lunney & Schnurr, 2007; Schnurr et al., 2009), and this study supports and extends those findings by suggesting that, of all the PTSD symptom clusters, numbing has the largest negative association on quality of life. Medialional analyses also supported and expanded upon past research (Erbes et al., 2007; Gudmundsdottir et al., 2004) by demonstrating that comorbid depression partially and fully mediated the relationships between the various PTSD symptom clusters and all satisfaction-related quality of life domains assessed by the QOLI. Taken together, the results of this study provide compelling evidence that, for Veterans with PTSD, the impact of PTSD on satisfaction-related quality of life is in large part accounted for by negative affect in the form of comorbid depression or numbing cluster symptoms.
Research has highlighted the important role negative affect plays in mood and anxiety disorders, and negative affect has been conceptualized as a higher-order factor that may explain the high rates of comorbidity across these disorders (Watson, 2009). To address these comorbidities, researchers have proposed an empirically-based reorganization of the DSM diagnostic categories to create a superclass encompassing all mood and anxiety disorders. Coined the Quadripartite Model, this overarching framework involves three correlated subclasses, including fear disorders (agoraphobia, panic disorder, social phobia, and specific phobia), distress disorders (dysthymic disorder, generalized anxiety disorder, PTSD, and MDD), and bipolar disorders (bipolar I, bipolar II, and cyclothymia) (Watson, O'Hara, & Stuart, 2008). Proponents of the Quadripartite Model advocate removing negative affect symptoms (i.e. numbing symptoms of PTSD) from diagnostic criteria and measuring them dimensionally, to both improve measurement and also to aid in differential diagnosis (Watson, 2009).

Although not ultimately endorsed by the DSM-5 categorization, the Quadripartite Model categorized PTSD as a distress disorder because of strong correlations with indicators of depression, and a substantial amount of negative affectivity variance. This was largely due to the PTSD numbing symptoms, or alternately PTSD dysphoria symptoms (Watson, 2009), another highly supported factor structure that differs slightly from the numbing factor solution (Simms, Watson, & Doebbellling, 2002). Analysis of large epidemiological samples shows that dysphoria and numbing symptoms are more highly correlated with depression than are other PTSD symptoms clusters (i.e. re-experiencing, avoidance, hyperarousal), and less significantly correlated with anxiety disorders than are other PTSD symptom clusters (Watson, 2009).
Although this finding has not been consistent across all samples (Gros, Magruder, Ruggiero, Shaftman, & Frueh, 2012), the results of the current study further support the connections between PTSD numbing symptoms and comorbid depression by demonstrating their similar negative associations with quality of life.

However, it is important to note that PTSD numbing symptoms and comorbid depression did not have an identical pattern of significant findings across quality of life domains. Rather, in simultaneous analyses, comorbid depression as measured by the BDI-II was significantly negatively related to satisfaction with the achievement, self-expression, relationships, and surroundings domains. PTSD numbing symptoms were significantly related to satisfaction with the self-expression and relationships domains even when accounting for depressive symptoms, and numbing’s association with the relationships domain was stronger than that of depression. This is interesting in that it suggests that the manifestations of negative affect in PTSD numbing symptoms and comorbid depression were distinct enough to result in a differential pattern of associations with quality of life domains. Further empirical research is needed to examine the different manifestations of negative affect in the mood and anxiety disorders as well as their clinical correlates, especially when determining inclusion of items for a general measure of negative affect across disorders.

These findings have significant clinical implications. They suggest that while addressing PTSD with evidence-based treatments should improve life quality, incorporating treatment elements specifically targeting comorbid depression and PTSD numbing symptoms may lead to even more improvement in quality of life outcomes in Veterans with PTSD. One such
intervention, which includes behavioral activation components with PTSD evidence-based treatment, found that the combined treatment lowered both PTSD symptoms and the overlapping symptoms of PTSD and depression (Daniel F Gros, Matthew Price, et al., 2012). While the researchers did not examine quality of life outcomes, given the robust empirical associations between both disorders and quality of life, it is conceivable that this innovative treatment modality could substantially improve life quality for Veterans. Another approach that could potentially address both PTSD and depression are transdiagnostic psychotherapies for mood and anxiety disorders (Barlow et al., 2010). Transdiagnostic treatments for the emotional disorders focus more broadly on the overarching symptoms found across all of the mood and anxiety disorders (e.g., poor emotional regulation) (Mansell, Harvey, Watkins, & Shafran, 2009), and these treatments could potentially address symptoms shared by both PTSD and depression (e.g. negative affect).

This study has several limitations that should be noted. The R² values of significant simultaneous regression analyses indicated that comorbid PTSD and depression only accounted for a portion of the variance across quality of life scores, highlighting the need for further research into predictors of satisfaction-related quality of life. Also, the BDI-II and diagnosis of MDD produced disparate findings in this sample. This is possibly a result of method variance, as the continuous BDI-II may have captured a larger degree of negative affect variance than the dichotomous MDD diagnosis, which may then have resulted in a stronger association with quality of life indicators. Additional research is needed utilizing multiple self-report and clinician-administered measures, across both continuous and dichotomous predictors, to explore
this issue. All measures of quality of life in this study, including objective measures, were obtained through self-report methodology. Use of other reporters, such as employers, spouses, and friends, could enhance the reliability of findings (Gros, Milanak, & Hershenberg, 2013). Furthermore, our study was cross-sectional in nature and therefore causality and temporal precedence cannot be conclusively established. This is most applicable to our mediation analyses, and findings would be greatly enhanced by prospective, longitudinal research designs.

**Conclusions**

The findings of this study suggest that in combat Veterans with PTSD, the presence of comorbid depressive symptoms is associated with lower quality of life across multiple domains of subjective life satisfaction. Results also suggest that quality of life decreases are associated with both PTSD numbing symptoms and comorbid depressive symptoms, and that comorbid depressive symptoms play a partially and fully mediating role in many relationships between PTSD and quality of life indicators. Together, these findings demonstrate the importance of negative affect when assessing quality of life in Veterans with PTSD, and highlight the need to shift to more transdiagnostic assessment (Watson, 2009) and treatment (Barlow et al., 2010) practices. Future research should examine the impact of negative affect on quality of life in other traumatized populations, as well as the impact of comorbid depression on quality of life outcomes following evidence-based PTSD treatment.
Table 1

*Descriptive Statistics and Standardized Regression Results for Differences in Quality of Life Indicators across Comorbid PTSD-MDD and PTSD Only Groups*

<table>
<thead>
<tr>
<th></th>
<th>PTSD-MDD (N=49)</th>
<th>PTSD Only (N=99)</th>
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<th>SE</th>
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<td>M</td>
<td>SD</td>
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<td>2.68</td>
<td>1.44</td>
<td>2.25</td>
</tr>
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</table>

Note: The total sample size for the study is larger than the sum of the PTSD-MDD and PTSD only groups because of participants with missing MDD diagnosis data that were included in some analyses of the study. Additionally, because of participant non-responding, some values were based on smaller sample sizes. Group means and standard deviations are not standardized, whereas betas and standard errors of regression analyses are standardized. PCS= SF-12 physical component summary; MCS= SF-12 mental health component summary; Achievement= QOLI achievement domain; Self-Expression= QOLI self-expression domain; Relationships= QOLI relationships domain; Surroundings= QOLI surroundings domain.

*p<.05  **p<.01  ***p<.001
Table 2

Standardized Results of Separate and Simultaneous Regressions between PTSD Symptom Clusters, MDD, and Quality of Life

<table>
<thead>
<tr>
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<th>Surroundings</th>
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<td>SE</td>
<td>β</td>
<td>SE</td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
</tr>
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<td>.08</td>
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<td>-.28**</td>
<td>.08</td>
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<td>.07</td>
<td>-.42***</td>
<td>.07</td>
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<tr>
<td>Hyper</td>
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<td>.08</td>
<td>-.27**</td>
<td>.08</td>
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<td></td>
<td></td>
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<td>( R^2 )</td>
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<td>.19**</td>
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<tr>
<td>( R^2 )</td>
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<td>.06</td>
<td>.19**</td>
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<tr>
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Note: A dashed line indicates that a value was not computed. Achievement= QOLI achievement domain; Self-Expression= QOLI self-expression domain; Relationships= QOLI relationships domain; Surroundings= QOLI surroundings domain; Reexp= PTSD reexperiencing cluster; Avoid = PTSD avoidance cluster; Numb=PTSD numbing cluster; Hyper= PTSD hyperarousal cluster; MDD= comorbid major depressive disorder.

*p<.05  **p<.01  ***p<.001
Table 3
Standardized Results of Separate and Simultaneous Regressions between PTSD Symptom Clusters, the Beck Depression Inventory-II, and Quality of Life

<table>
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<th>Surroundings</th>
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<td>.09</td>
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Note: A dashed line indicates that a value was not computed. Achievement= QOLI achievement domain; Self-Expression= QOLI self-expression domain; Relationships= QOLI relationships domain; Surroundings= QOLI surroundings domain; Reexp= PTSD reexperiencing cluster; Avoid= PTSD avoidance cluster; Numb =PTSD numbing cluster; Hyper= PTSD hyperarousal cluster; BDI= Beck Depression Inventory-II total score.

*p<.05  **p<.01  ***p<.001
Table 4

Results of Mediational Analyses between PTSD Symptom Clusters, Comorbid Depression Symptoms, and Quality of Life Indicators

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<th>XM</th>
<th>XM'</th>
<th>MY</th>
<th>MY'</th>
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<th>XY</th>
<th>Indir</th>
<th>Indir</th>
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Note: XY' indicates the relationship between X and Y while controlling for M. The MY pathway is calculated while controlling for X. Achievement= QOLI achievement domain; Self-Expression= QOLI self-expression domain; Relationships= QOLI relationships domain; Surroundings= QOLI surroundings domain; Reexp= PTSD reexperiencing cluster; Avoid = PTSD avoidance cluster; Numb = PTSD numbing cluster; Hyper= PTSD hyperarousal cluster; BDI= Beck Depression Inventory-II total score; Indir=indirect effect.

*p<.05  **p<.01  ***p<.001
References


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Paper #3: Examination of the Content Specificity of Posttraumatic Cognitions in Combat Veterans with Posttraumatic Stress Disorder

Please note that results of this study are currently under review. Citation for the manuscript is:

Abstract

Cognitive theories have proposed the idea of content specificity, which holds that emotional disorders are associated with unique sets of negative cognitions. The existent research exploring cognitions related to posttraumatic stress disorder (PTSD) and depression is sparse, and research is needed in veteran samples, especially exploring associations with individual PTSD symptom clusters. In this study of 150 male combat veterans with PTSD, we analyzed the unique associations of comorbid depressive symptoms and PTSD symptom clusters with posttraumatic cognitions, and also performed post-hoc analyses to examine the mediational role of comorbid depressive symptoms. Results showed that posttraumatic negative cognitions about the self and self-blame were most strongly associated with comorbid depressive symptoms and the depression-related PTSD numbing cluster. Comorbid depressive symptoms also partially mediated many of the relationships between posttraumatic cognitions and PTSD symptom clusters. The findings of this study suggest that posttraumatic cognitions about the self and self-blame are not specific to PTSD, but rather are more strongly related to symptoms of depression and negative affect. The results also suggest a potential pathway from posttraumatic cognitions to PTSD through the partially mediating influence of comorbid depression, and highlight the need to assess and treat comorbid depression in veterans with PTSD.

**Keywords:** depression, PTSD, comorbidity, Quadripartite Model, mediation, posttraumatic cognitions
Introduction

Research has demonstrated that maladaptive cognitions and cognitive styles are associated with the onset and maintenance of a number of psychiatric disorders (Alloy et al., 2006; Ollendick & Hirshfeld-Becker, 2002). Beck (1979) hypothesized that negative cognitions have content specificity, that there are unique sets of cognitions associated with each emotional disorder. There is empirical support for this concept (Ehring, Ehlers, & Glucksman, 2008), however one area in need of further research is the content specificity of cognitions related to posttraumatic stress disorder (PTSD) and depression.

Ehlers and Clark (2000) proposed a cognitive theory of PTSD in which the negative appraisal of traumatic events and their consequences can play a key role in maintaining PTSD through producing a sense of current threat. These appraisals can be external, about the world as a dangerous place, or internal, concerning one’s self as an incapable person or to blame for the traumatic event. In a similar vein, Foa and Rothbaum (2001) proposed that cognitions related to the world as completely dangerous, and related to oneself as totally incompetent, mediate the relationship between the experience of trauma and the development of PTSD. Additionally, research has suggested that internal, stable, and global attributions are related to PTSD symptoms (see Gonzalo, Kleim, Donaldson, Moorey, & Ehlers, 2012).

Negative cognitions about one’s self, the world, and the future have been theorized to play a causal role in the development of depression (Beck, 1967). Depressogenic beliefs can be grouped into three thematic categories: beliefs about helplessness (e.g., “I am incompetent”),
unlovability, and worthlessness (Beck, 2011). In the reformulated helplessness theory of depression, researchers argued that a general cognitive style that attributes failure to internal, stable, and global causes predisposes one to depression (Abramson, Seligman, & Teasdale, 1978). In a revision of this theory, researchers postulated the existence of a subtype of depression related to hopelessness, and hypothesized that inferring negative consequences from negative life events, and making stable, global attributions regarding the causes of these events, are proximal contributory causes of hopelessness depression (Abramson, Metalsky, & Alloy, 1989).

The overlap between the cognitions associated with PTSD and depression is noteworthy, especially cognitions related to one’s self as an incapable person, and internal, stable, and global negative attributions. However despite these similarities, to date there has been relatively sparse research addressing content specificity of cognitions related to PTSD and depression. Studies have been conducted in samples of motor vehicle accident survivors (Ehring et al., 2008), assault survivors (Kleim, Ehlers, & Glucksman, 2012), and a combined sample of inpatients, outpatients, emergency patients, and community members (Gonzalo et al., 2012); all of these studies found support for unique sets of cognitive predictors for PTSD and depression, as well as support for overlapping predictors. Notably, Gonzalo et al. (2012) found that posttraumatic cognitions were strongly associated with both PTSD and depressive symptoms, and metaanalytic research has produced comparable results with other disorders, finding that anxiety-related cognitions shared equal variance with anxiety-related and depressive symptoms (Beck & Perkins, 2001).
While this research provides a starting point, there are a number of shortcomings in the literature that need to be addressed. To date, no study has examined this issue in a veteran sample, an important extension given the high degree of PTSD and depression comorbidity observed in this population (Gros et al., 2012; Gros, Simms, & Acierno, 2010). Existing research also largely focuses on categorically comparing disorders rather than dimensionally examining the relationships between cognitive predictors and individual symptom clusters, which could improve theoretical understanding of the disorders (Ehring et al., 2008). To our knowledge, only one study has examined the relationships between posttraumatic cognitions and individual PTSD symptom clusters (Blain, Galovski, Elwood, & Meriac, 2013). While the researchers did not examine comorbid depressive symptoms, they did find that self-blame and negative cognitions about the self were related to the PTSD numbing cluster (Blain et al., 2013). Given that the PTSD numbing cluster shares significant overlap with depressive symptoms (Gros et al., 2010), the findings raise important questions about the specificity of posttraumatic cognitions to PTSD.

In this study we explored the unique associations of PTSD symptom clusters and comorbid depressive symptoms with posttraumatic cognitions in a sample of male combat veterans with PTSD. We hypothesized that 1) PTSD clusters and comorbid depressive symptoms would both be uniquely associated with posttraumatic cognitions and 2) in its relationships with posttraumatic cognitions, the pattern of findings for the PTSD numbing cluster would resemble the pattern of findings for comorbid depressive symptoms more closely than the patterns associated with the other PTSD symptom clusters. We also conducted post-hoc analyses
examining the meditating role of comorbid depressive symptoms on relations between posttraumatic cognitions and PTSD symptom clusters.

**Method**

**Participants**
In this study, we used data from the baseline assessments of a federally funded clinical trial evaluating the effectiveness of the telehealth modality for delivering evidence-based treatment to veterans with PTSD (Morland et al., 2014); this included baseline data drawn from the study’s pilot cohort (Morland, Hynes, Mackintosh, Resick, & Chard, 2011). Participants were recruited at Veterans Affairs clinics and Vet Centers on the Hawaiian Islands, and participants consisted of 150 male combat veterans with PTSD. The inclusion criterion for the present study was a diagnosis of current PTSD derived from the Clinician Administered PTSD Scale for DSM-IV (CAPS; Blake et al., 1995). The rate of comorbid MDD in this sample, as determined by the Structured Clinical Interview for DSM-IV (First, Spitzer, Gibbon, & Williams, 2002), was 33%. This was lower than the rate of a recent metaanalysis (Rytwinski, Scur, Feeny, & Youngstrom, 2013), potentially because of strict application of DSM-IV criteria during assessment.

**Procedures**
The parent study was approved by the Department of Veterans Affairs Pacific Islands Healthcare System IRB, and all participants provided written informed consent prior to initiating study participation. Participants completed clinician-administered interviews and self-report questionnaires during two baseline assessments; in rare instances, unfinished measures were
completed during a subsequent orientation session. All participants were compensated for their travel related costs with a fifty dollar gift card at each assessment session.

Measures

*The Clinician Administered PTSD Scale for DSM-IV (CAPS)*. The CAPS, a clinician-administered interview, was utilized in this study to establish a diagnosis of current DSM-IV PTSD (Blake et al., 1995). The “1/2” scoring rule was used, in which symptoms must have occurred at least once within the last month and be of at least a moderate intensity (Weathers, Ruscio, & Keane, 1999). The CAPS has good validity and strong reliability (Foa & Tolin, 2000).

*Posttraumatic Stress Disorder Checklist (PCL)*. The PCL (Weathers, Litz, Herman, Huska, & Keane, 1993) is a self-report measure that uses a 5-point Likert scale to assess the severity of the 17 DSM-IV PTSD symptoms. The PCL has good internal consistency, strong test-retest reliability, and correlates well with other measures of PTSD (Keen, Kutter, Niles, & Krinsley, 2008).

Internal consistency of the PCL total score in the present study was good ($\alpha=.90$), and the reexperiencing ($\alpha=.84$), numbing ($\alpha=.73$), and hyperarousal ($\alpha=.76$) clusters all had acceptable reliability. The internal consistency of the avoidance cluster ($\alpha=.65$) was low, likely because it consists of only two items.

The PCL-Military Version (PCL-M) was given to the first two cohorts of the parent study, while the PCL-Stressor Specific Version (PCL-S) was used in the other cohorts. The measures differ primarily in their instructions, and research in a military sample found that changes to PCL instructions do not affect symptom reporting (Riviere et al., 2011). In this study, there were no significant differences between PCL-M and PCL-S scores when controlling for depression ($p > .05$).
Beck Depression Inventory-II (BDI-II). The 21-item self-report BDI-II was used to assess depressive symptom severity (Beck, Steer, & Brown, 1996). BDI-II items are rated on a 4-point Likert scale, with higher scores indicative of greater symptom severity. The BDI-II has been used frequently in veteran samples (Strachan, Gros, Ruggiero, Lejuez, & Acierno, 2012) and possesses strong validity and reliability (Beck et al., 1996). In the present study, the BDI-II had good internal consistency (α=.93).

Posttraumatic Cognitions Inventory (PTCI). The PTCI (Foa, Ehlers, Clark, Tolin, & Orsillo, 1999) is a self-report measure which gauges agreement or disagreement with maladaptive trauma-related cognitions. Each item is measured using a 7-point Likert scale, with anchors ranging from 1 (totally disagree) to 7 (totally agree), and high scores are indicative of more endorsement of posttraumatic cognitions. Multiple studies have supported a three-factor structure for the PTCI, including negative cognitions about the self (e.g. “I am inadequate”), negative cognitions about the world (e.g. “The world is a dangerous place”), and self-blame (e.g. “There is something about me that made the event happen”) subscales (Beck et al., 2004; Foa et al., 1999). The PTCI subscales have good test-retest reliability and internal consistency, and the measure on the whole has strong discriminant validity in differentiating between traumatized and non-traumatized populations (Foa et al., 1999). The internal consistency of the three PTCI subscales was acceptable in the present study (self: α=.95; world: α=.86; self-blame: α=.82).

Statistical Analyses
For analyses involving PTSD symptom clusters, we used the four-factor model developed by King, Leskin, King, and Weathers (1998). Termed the emotional numbing model, this
empirically-supported factor structure (Yufik & Simms, 2010) has reexperiencing (DSM-IV B1-B5 symptoms) and hyperarousal (D1-D5) clusters that are the same as those of the DSM-IV (American Psychiatric Association, 2000). However, the emotional numbing model bifurcates the DSM-IV avoidance cluster, producing an avoidance cluster (C1-C2) as well as a new numbing cluster (C3-C7).

To examine the unique associations of posttraumatic cognitions with PTSD symptom clusters and comorbid depressive symptoms, we conducted a series of separate and simultaneous linear regressions. First, we separately regressed each PTCI subscale (self, world, and self-blame) on to each PTSD symptom cluster as measured by the PCL (reexperiencing, avoidance, numbing, and hyperarousal) and comorbid depressive symptoms as assessed by the BDI-II. In all cases where a PTSD symptom cluster and comorbid depressive symptoms were both significantly associated with a PTCI subscale, we performed additional analyses. We first regressed the PTCI subscale on the PTSD cluster and comorbid depressive symptoms simultaneously to determine if the constructs retained their significant associations when analyzed jointly. Then, we regressed the PTCI subscale on to all PTSD clusters simultaneously to determine if the relationship remained significant when accounting for other PTSD symptoms. Following this, we regressed the PTCI subscale on to all PTSD symptom clusters and comorbid depressive symptoms simultaneously. We compared this model’s $R^2$ value to the $R^2$ of the regression that did not include comorbid depressive symptoms, and examined the incremental variance accounted for by comorbid depression.
Initial analyses suggested the possibility of mediation of PTCI subscale-PTSD cluster relationships by comorbid depressive symptoms. As such, for all instances in which comorbid depressive symptoms attenuated the strength of the PTCI-PTSD relationship in simultaneous analyses, we conducted post-hoc mediation analyses. We used path analysis, which is a special case of structural equation modeling that utilizes observed variables and does not account for measurement error through use of latent variables (Schumacker & Lomax, 2004). We defined mediation as occurring when a PTCI subscale and PTSD cluster were significantly related to each other, the PTCI subscale was significantly related to comorbid depressive symptoms (i.e. the mediator), comorbid depressive symptoms were significantly related to the PTSD cluster when controlling for the PTCI subscale, the product of the pathways to the mediator was significant (i.e. the indirect effect), and the relationship between the PTCI subscale and PTSD symptom cluster was attenuated when controlling for comorbid depressive symptoms (Kenny, 2014). We defined complete mediation as occurring when a significant PTCI-PTSD relationship was reduced to zero by the inclusion of comorbid depressive symptoms in analyses, and defined partial mediation as instances when the relationship was attenuated but remained greater than zero (Kenny, 2014).

Descriptive statistics and reliabilities were calculated using SPSS, Version, 19 (IBM Corporation, 2010), while regression and path analyses were performed with MPlus, Version 6.1 (Muthén & Muthén, 2010). To address a small amount of missing data (4.33%), we used maximum likelihood estimation. We also converted independent variables to single-item latent
variables for the purposes of missing data analysis, a technique which does not change
interpretation of model parameters (Enders, 2010).

Results

Participants in this study were largely married (n=89, 59.3%), divorced (n=33, 22.0%),
and single (n=22, 14.7%), and primarily of Caucasian (N=67, 45.6%), Pacific Islander (N=30,
20.4%), or Asian (N=23, 15.6%) ethnicity. They mainly served in the Vietnam (N=95, 63.3%),
OEF/OIF (N=44, 29.3%), and Operations Desert Storm/Shield (N=22, 14.7%) theatres. Because
some veterans served in multiple war eras, numbers and percentages are larger than the sample
size.

Separate regression analyses are displayed in the top portion of Table 1. All PTSD
symptom clusters, as well as comorbid depressive symptoms, were significantly related to the
PTCI self and world subscales. Comorbid depressive symptoms and all PTSD clusters except
hyperarousal were significantly associated with the PTCI self-blame subscale.

Simultaneous regressions involving individual PTSD symptom clusters and comorbid
depressive symptoms are listed in the middle portion of Table 1. Comorbid depressive
symptoms retained its significant associations with all PTCI subscales when analyzed with PTSD
symptom clusters. Conversely, reexperiencing and avoidance clusters lost their significant
relationships with all PTCI subscales when jointly analyzed with comorbid depressive
symptoms. The numbing cluster retained its significant association with the self subscale in joint
analyses, and hyperarousal was significantly associated with both the self and world subscales when analyzed simultaneously with comorbid depressive symptoms.

Simultaneous regressions involving all PTSD symptoms clusters are displayed in the bottom portion of Table 1. In analyses involving all PTSD clusters, numbing possessed the only significant positive association with the self and self-blame subscales, and hyperarousal had the only significant positive relationship with the world subscale. Hyperarousal was also significantly negatively associated with the self-blame subscale. Addition of comorbid depressive symptoms to analyses did not cause any of these relationships to drop out of significance, and comorbid depressive symptoms demonstrated significant associations with all PTCI subscales in these analyses. The relationships of the PTCI self and self-blame subscales with comorbid depressive symptoms were notably stronger than those with the PTSD clusters, while the relationship between depressive symptoms and the world subscale was weaker than the relation between this subscale and the hyperarousal cluster. Adding comorbid depressive symptoms to analyses substantially raised the $R^2$ value across all PTCI subscales.

Post-hoc mediation analyses are displayed in Table 2. Comorbid depressive symptoms partially mediated the relationships between the PTCI self subscale and all PTSD symptom clusters. The self subscale-numbing and the self subscale-hyperarousal relationships still remained significant even when accounting for the mediating influence of depressive symptoms. Similarly, comorbid depressive symptoms partially mediated the relationships between the PTCI world subscale and all PTSD symptom clusters, but the world subscale-hyperarousal relationship
still retained significance after accounting for depressive symptoms. Finally, comorbid depressive symptoms partially mediated the relationships between the PTCI self-blame subscale and the PTSD reexperiencing, avoidance, and numbing clusters.

**Discussion**

In this study, we examined the content specificity of posttraumatic cognitions by exploring their unique associations with PTSD symptom clusters and comorbid depressive symptoms in a sample of combat veterans with PTSD. The hypotheses of our study were confirmed, as PTSD symptom clusters and comorbid depressive symptoms were both uniquely associated with posttraumatic cognitions. Additionally, the pattern of significant findings between the PTSD numbing cluster and PTCI subscales, most notably the self and self-blame subscales, resembled the pattern of significant findings associated with comorbid depressive symptoms more closely than the patterns associated with the other PTSD clusters.

We found that the hyperarousal cluster was positively associated with negative cognitions about the world, a relationship that has been previously demonstrated (Blain et al., 2013). Surprisingly, in this sample the hyperarousal cluster was negatively associated with self-blame, even when controlling for comorbid depressive symptoms and other PTSD clusters. The negative cognitions about the world subscale contains items concerning the world as a dangerous and unpredictable place, full of untrustworthy people, while the self-blame subscale contains primarily negative, internal attributions related to the causes of traumatic events (Foa et al., 1999). Our study’s findings suggest that high levels of hyperarousal symptoms may be more
related to external, negative attributions about the world, and less related to negative internal attributions about one’s own role in traumatic experiences.

The research to date examining the content specificity of PTSD and depression has found specific factors associated with each disorder, as well as common factors shared across disorders (Gonzalo et al., 2012; Kleim et al., 2012). In this study, we found limited evidence to support the content specificity of posttraumatic cognitions. In simultaneous analyses involving all PTSD clusters and comorbid depressive symptoms, the PTSD hyperarousal cluster possessed the strongest relationship with negative cognitions about the world. However, comorbid depressive symptoms were also significantly associated with these cognitions. Additionally, in these analyses, comorbid depressive symptoms and the PTSD numbing cluster, which shares significant overlap with depression (Gros et al., 2010), possessed the only significant positive associations with the negative cognitions about self and self-blame subscales. Furthermore, in post-hoc mediation analyses, comorbid depressive symptoms partially mediated all analyzed relationships between posttraumatic cognitions and PTSD symptom clusters. Past metaanalytic research has found a lack of content specificity for anxious cognitions (Beck & Perkins, 2001), and the results of this study extend those findings to PTSD. They suggest that posttraumatic cognitions, especially cognitions related to the self and self-blame, are not specific to PTSD, but rather are more strongly related to symptoms of depression and negative affect.

The results of this study are also consistent with models of psychopathology that stress the overlap between PTSD and depression and the importance of negative affect to the disorders,
notably Watson’s Quadripartite Model (Watson, 2009). On the basis of substantial epidemiological research, the model classifies PTSD as a “distress” disorder alongside depression and dysthymia, an arrangement primarily due to the large degree of negative affectivity present in all the disorders (Watson, 2009). To reduce comorbidity, Watson has suggested removing negative affect symptoms from the diagnostic criteria of depression and anxiety disorders and modeling them dimensionally (Watson, 2009). Interestingly, the results of this study suggest that such a step might substantially attenuate the relationship between posttraumatic cognitions and PTSD diagnosis.

This study has important assessment and treatment implications. The findings suggest the need to use more generalized measures of psychopathology that capture both PTSD and depressive symptoms, such as the Inventory of Depression and Anxiety Symptoms (Watson et al., 2007). In the absence of such measures, the results of this study highlight the importance of jointly assessing depression and PTSD. Additionally, prior research examining content specificity of PTSD and depression has suggested the possible benefits of shifting from a primarily disorder-based tack to a dimensional, symptom focused approach (Ehring et al., 2008). The present study supports the need for such a shift, as well as the necessity of considering both posttraumatic cognitions and PTSD as heterogeneous constructs, the subscales and symptom clusters of which are likely to demonstrate differential patterns of association.

With regard to treatment, the current study’s mediation findings propose a potential pathway from posttraumatic cognitions to PTSD symptom clusters through the partially
mediating influence of comorbid depressive symptoms. These findings, along with the results from the study’s regression analyses, argue that PTSD therapies should incorporate treatment elements designed to address negative affect, such as behavioral activation (Gros et al., 2012), and that doing so could reduce the impact of posttraumatic cognitions on PTSD symptoms. Study findings also support the benefits of utilizing transdiagnostic treatments (Barlow et al., 2010) to address negative affect in individuals with comorbid PTSD and depressive symptoms.

This study has several limitations that should be noted. It is cross-sectional, and causal relationships cannot be definitively established without longitudinal research designs. This is most applicable to the mediation analyses, and it is possible that the direction of our pathways is incorrect and PTSD clusters actually result in comorbid depressive symptoms, which in turn lead to posttraumatic cognitions. If this proved to be the case it would still be of interest, as it would suggest that negative affect in the form of comorbid depressive symptoms can lead to the reinforcement of posttraumatic cognitions. This study only examined posttraumatic cognitions, and the findings regarding specificity would be greatly strengthened by examining depressive cognitions as well. Additionally, in analyzing PTSD and posttraumatic cognitions, we utilized empirically-supported factor structures, but did not examine factor structures within the current sample. We also did not employ statistical controls for Type I error, which could impact study findings. Furthermore, this study utilized a male combat veteran sample, and results should be replicated in other populations, notably those with female representation and other trauma types.

Conclusion
Our findings suggest that posttraumatic cognitions do not possess content specificity, but are most strongly associated with comorbid depressive symptoms and the depression-related PTSD numbing cluster. We also found that depressive symptoms mediated the relationships between negative cognitions about one’s self, the world, and self-blame and nearly all PTSD clusters, suggesting that negative affect may play an important role in the pathway from posttraumatic cognitions to PTSD symptoms. Future research should examine these relationships with longitudinal research designs, operationalize depression and PTSD both categorically and dimensionally, include hypothesized cognitive predictors of both depression and PTSD, as well as analyze associations with individual PTSD symptom clusters.
Table 1

Standardized Results of Separate and Simultaneous Regressions Between PTSD Symptom Clusters, Comorbid Depression, and Posttraumatic Cognitions

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Note: A dashed line indicates that a value was not computed. Self=PTCI negative cognitions about self subscale; World =PTCI negative cognitions about the world subscale; Blame=PTCI self-blame subscale; Reexp=PTSD reexperiencing cluster; Avoid=PTSD avoidance cluster; Numb=PTSD numbing cluster; Hyper=PTSD hyperarousal cluster; BDI=Beck Depression Inventory-II Total Score. *p<.05 **p<.01 ***p<.001
Table 2

Results of Mediation Analyses between Posttraumatic Attributions, Comorbid Depressive Symptoms, and PTSD Symptom Clusters

| X      | M   | Y    | X     | Y     | XY   | XY'   | X     | SE   | X     | SE   | M     | SE   | Y     | SE   | MY   | SE   | MY   | SE   | XY'  | SE   | Indir | SE   |
|--------|-----|------|-------|-------|------|-------|-------|------|-------|------|-------|------|-------|------|------|------|------|------|------|-------|------|-------|------|
| Self   | BDI | Reexp| .37***| .07   | .70***| .04   | .45***| .10   | .06   | .11   | .31***| .07  |
| Self   | BDI | Avoid| .40***| .07   | .70***| .04   | .26*  | .11   | .21   | .11   | .19*  | .08  |
| Self   | BDI | Numb | .64***| .04   | .70***| .04   | .30***| .09   | .43***| .08   | .21** | .06  |
| Self   | BDI | Hyper| .44***| .07   | .70***| .04   | .26*  | .10   | .25*  | .10   | .18*  | .07  |
| World  | BDI | Reexp| .20*  | .08   | .37***| .07   | .49***| .07   | .02   | .08   | .18***| .05  |
| World  | BDI | Avoid| .21*  | .08   | .37***| .07   | .38***| .08   | .07   | .09   | .14***| .04  |
| World  | BDI | Numb | .30***| .08   | .37***| .07   | .57***| .06   | .09   | .07   | .21***| .05  |
| World  | BDI | Hyper| .43***| .07   | .37***| .07   | .33***| .07   | .31***| .08   | .12** | .04  |
| Blame  | BDI | Reexp| .23** | .08   | .39***| .07   | .47***| .07   | .05   | .08   | .18***| .05  |
| Blame  | BDI | Avoid| .20*  | .08   | .39***| .07   | .40***| .08   | .04   | .09   | .16** | .04  |
| Blame  | BDI | Numb | .34***| .08   | .39***| .07   | .55***| .06   | .13   | .07   | .22***| .05  |

Note: XY’ indicates the relationship between X and Y while controlling for M (i.e., direct effects). MY is the relationship between M and Y while controlling for X. Self = PTCI negative cognitions about self subscale; World = PTCI negative cognitions about the world subscale; Blame = PTCI self-blame subscale; BDI = Beck Depression Inventory-II total score; Reexp = PTSD reexperiencing cluster; Avoid = PTSD avoidance cluster; Numb = PTSD numbing cluster; Hyper = PTSD hyperarousal cluster; Indir = indirect effect

*p<.05  **p<.01  ***p<.001
Reference


Overall Conclusion
Posttraumatic Stress Disorder (PTSD) is highly comorbid and has been shown to have significant overlap with a number of emotional and anxiety disorders (Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Zayfert, Becker, Unger, & Shearer, 2002). The degree of comorbidity is perhaps highest with depression, and a recent metaanalysis reported that 52% of individuals with PTSD also had comorbid Major Depressive Disorder (MDD; Rytwinski, Scur, Feeny, & Youngstrom, 2013). Various explanations have been proposed to account for this high degree of overlap, including the existence of a general traumatic stress construct (O’Donnell, Creamer, & Pattison, 2004), as well as symptom overlap between the disorders (Gros, Price, Magruder, & Frueh, 2012a; Gros, Simms, & Acierno, 2010). Theorists have also developed new taxonomies for depression and PTSD, including the Quadripartite Model, which places both PTSD and MDD into the same diagnostic class (Watson, 2005, 2009). Comorbidity between PTSD and depression is likely to increase with the creation of the negative alterations in cognitions and mood PTSD cluster in the DSM 5 (American Psychiatric Association, 2013), and as such, more research is needed to inform the clinical care of comorbid individuals and progress the theoretical development of models of this comorbidity. The purpose of this dissertation was to further these ends by examining the implications of comorbid depression in male combat veterans with PTSD across a variety of clinical correlates, include anger, quality of life, and posttraumatic cognitions.
This impact was examined across three related studies. Study one examined the mediating roles of comorbid MDD, as well as PTSD numbing and dysphoria clusters, on the relationships between other PTSD symptom clusters and anger. Results indicated that comorbid MDD partially mediated the relationships between PTSD clusters and state anger, while PTSD numbing and dysphoria clusters partially mediated the relationships between PTSD symptom clusters and trait anger. Study two examined the impact of PTSD symptom clusters and comorbid depression on objective and subjective quality of life domains, and found that veterans with comorbid PTSD-MDD reported significantly worse satisfaction-related quality of life than those with PTSD alone. However, subsequent analyses revealed that this was in large part due to PTSD numbing symptoms, and to better understand the unique contributions of depression and PTSD, post-hoc analyses were conducted using a measure of depressive symptom severity. Results indicated that comorbid depressive symptoms exerted a stronger influence on quality of life indicators than any PTSD symptom cluster, but that the PTSD numbing cluster was still uniquely associated with many of these indicators. Findings also demonstrated that comorbid depressive symptoms partially and fully mediated many of the relationships between PTSD clusters and quality of life indicators. Study three examined the content specificity of posttraumatic cognitions by examining their associations with comorbid depressive symptoms and PTSD symptom clusters. The study did not support content specificity, but rather found that posttraumatic negative cognitions about the self and self-blame were most strongly associated with comorbid depressive symptoms and the PTSD numbing cluster, while negative cognitions about the world were related to both the PTSD hyperarousal cluster and comorbid depressive
symptoms. Post-hoc analyses showed that comorbid depressive symptoms partially mediated many of the relationships between posttraumatic cognitions and PTSD symptom clusters.

The results of these studies provide insight into several issues, including a potential causal pathway, explanations of PTSD and depression comorbidity, issues with the DSM 5, implications for treatment, and defining depression in comorbidity research. One of the more striking findings of this dissertation was that all three studies found strong pathways mediated by comorbid depression. When considered together, the studies suggest a possible causal pathway stretching from risk factors of PTSD all the way to negative outcomes of the disorder. In study three, results indicated that comorbid depressive symptoms partially mediated relationships between posttraumatic cognitions and PTSD symptom clusters. Because posttraumatic cognitions are theorized to lead to the development of PTSD (Foa & Rothbaum, 2001), a position with empirical support (Ehring, Ehlers, & Glucksman, 2008), the findings suggest that after a traumatic experience, comorbid depression may strongly influence whether or not posttraumatic cognitions give rise to PTSD symptoms. Studies one and two raise a similar possibility regarding clinical outcomes, highlighting that depressive symptoms or comorbid MDD may significantly impact whether or not PTSD symptoms lead to increased anger and decreased quality of life. It is extremely important to highlight the cross-sectional nature of these studies, which limits conclusions regarding causality and temporal precedence, and the ability to definitively establish the existence of such a pathway. Additionally, the vast majority of analyses suggested partial, rather than full, meditation of study constructs by comorbid depression, strongly indicating the existence of other important predictors. However, despite
these limitations, the results of this dissertation do suggest that targeting comorbid depression in individuals with PTSD could have a dually beneficial result: it could limit the influence of posttraumatic cognitions on PTSD symptoms, as well as attenuate the impact of these symptoms on negative outcomes related to anger and quality of life.

The results of these studies also have implications for explanations of PTSD and depression comorbidity. The finding of study three that both comorbid depressive symptoms and PTSD numbing and hyperarousal clusters were significantly related to posttraumatic cognitions seems to provide some support for the theory of a general traumatic stress construct, an underlying assumption of which is that PTSD and depression have common risk factors (O’Donnell et al., 2004). However, in this same study, comorbid depressive symptoms were more strongly related to negative cognitions about the self, the PTSD hyperarousal cluster was more strongly associated with negative cognitions about the world, and both comorbid depressive symptoms and PTSD symptom clusters had independent, significant relationships with all posttraumatic cognitions subscales. In comparable fashion, study two found similar but unique patterns of relationships for quality of life indicators with comorbid depression and PTSD symptom clusters, and study one found that the disorders were related to different forms of anger. In short, rather than indicating that PTSD and depression are different manifestations of a general traumatic stress construct, our findings suggest that the disorders are related but highly similar constructs, a conclusion also drawn by factor analytic research examining their comorbidity (Grant, Beck, Marques, Palyo, & Clapp, 2008).
Our results also support explanations of comorbidity that highlight the overlap between PTSD’s numbing symptoms and depression (Gros et al., 2012a). Research has demonstrated that individuals endorsing PTSD numbing symptoms evidence higher rates of PTSD-MDD comorbidity (Gros et al., 2010). Our findings further support these explanations by demonstrating that the pattern of correlates associated with PTSD numbing symptoms bears a strong resemblance to the pattern associated with comorbid depression, and a varies from the patterns of correlates associated with other PTSD symptom clusters. Study three found the pattern of relations between posttraumatic cognitions and comorbid depressive symptoms was the most similar to the pattern of relations between PTSD numbing symptoms and these cognitions. Study two produced a similar pattern of association, as the pattern of relationships with quality of life indicators was highly similar for PTSD numbing symptoms and comorbid depressive symptoms. Study one even provided some support for this explanation, as both PTSD numbing symptoms and comorbid MDD mediated PTSD cluster-anger relationships.

This research also has implications for the Quadripartite Model (Watson, 2009). Similar to other researchers (Spitzer, First, & Wakefield, 2007), the models’ author, Watson, has suggested the need to remove numbing/dysphoria symptoms from the PTSD diagnostic criteria to improve specificity and reduce comorbidity (Watson, 2009). However, acknowledging the important role these symptoms play in the disorder, he proposed that they instead be assessed dimensionally to create a measure of negative affectivity (Watson, 2009). Our results suggest that eliminating the PTSD numbing/dysphoria symptoms entirely from the disorder could result in the loss of clinically relevant information. Specifically, study two suggests that if the PTSD
numbing cluster was removed from the disorder, the disorder would no longer capture the symptoms that are primarily associated with negative satisfaction-related quality of life. Likewise, a significant portion of PTSD’s association with posttraumatic cognitions would also likely be lost. That being said, it merits mention that regulating PTSD numbing/dysphoria symptoms to a non-diagnostic, dimensionally assessed metric could have the unintended consequence of deemphasizing their importance and inadvertently encouraging the conception that these symptoms are not essential to the assessment and treatment of PTSD. Clearly, if this approach were adopted, the importance of PTSD numbing and dysphoria symptoms would need to be highlighted, and their assessment made a required part of the diagnostic process.

However, this may be a moot point for the foreseeable future, as the DSM 5 did not remove these symptoms, but in fact actually expanded the role of negative affect in the diagnostic criteria of PTSD. The DSM 5 took the tack of removing the PTSD numbing symptoms from the DSM-IV avoidance cluster, expanding upon these symptoms, and creating a new symptom cluster required for diagnosis, labeled “negative alterations in cognitions and mood” (American Psychiatric Association, 2013). While this will likely have the effect of either maintaining or increasing comorbidity between depression and PTSD (Lockwood & Forbes, 2014). The shift may also have an unforeseen positive outcome. By separating PTSD numbing symptoms from avoidance symptoms and creating a new symptom cluster, the new diagnostic criteria may capture more clinically relevant information. Specifically, the studies of this dissertation suggests that by increasing the importance of negative affectivity to the diagnosis of PTSD, the disorder as it is conceptualized in the DSM 5 may strengthen relationships with
posttraumatic cognitions and better capture more severe anger and negative quality of life outcomes. The disorder as newly conceptualized may represent a more severe pathological presentation, a potentially positive nosological outcome given criticisms that the disorder can in some cases capture normative stress reactions (Spitzer et al., 2007).

Furthermore, the results of this dissertation speak to the need to treat comorbid depression in individuals with PTSD and point to several possible avenues. One approach could involve incorporating empirically established treatments for depression, like behavioral activation (Dimidjian, Barrera Jr, Martell, Muñoz, & Lewinsohn, 2011), into existing protocols for PTSD (e.g., Resick, Monson, & Chard, 2007). One study took this approach and, interestingly, while treatment did address PTSD and depression symptoms overlapping with PTSD, it did not significantly reduce non-overlapping depression symptoms (Gros et al., 2012b). It may be necessary to incorporate more cognitive treatment elements, which given their overlap with PTSD cognitions (Gonzalo, Kleim, Donaldson, Moorey, & Ehlers, 2012), may also further improve PTSD symptom reduction. It could also be useful to utilize transdiagnostic psychotherapies, which are designed to address symptoms shared across disorders (Barlow et al., 2010). Much of the transdiagnostic treatment research has explored protocols addressing multiple anxiety disorders, with some initial positive findings on the impact of treatment for both anxiety and depression disorders (Norton, Hayes, & Hope, 2004). Future transdiagnostic research should specifically examine the impact of treatments on individuals with comorbid PTSD and depression.
Finally, these three studies also highlight the importance of how one defines depression in comorbidity research. Our findings appear to support previous research demonstrating the differences between clinician-administered and self-report measures of depression (Cuijpers, Li, Hofmann, & Andersson, 2010), as well as categorical and dimensional methods of assessment (Watson, 2009). While the current study does not permit an analysis of the individual contributions of each of these factors to differences in depression ratings, what can be said is method variance in defining depression had a large impact on outcomes. This was most apparent in study two, in which clinician-rendered diagnosis of MDD and dimensionally assessed self-report depressive symptoms produced dramatically different patterns of association with quality of life indicators. If post-hoc analyses utilizing dimensionally-assessed depressive symptoms had not been conducted, results would have led to the conclusion that comorbid depression does not influence satisfaction-related quality of life indicators. Instead, findings demonstrated that comorbid depressive symptoms are not only the strongest predictor of negative satisfaction-related quality of life, but that they also partially mediated many of the relationships explored in the study. Future research examining depression and PTSD comorbidity should ideally utilize multiple methods of depression assessment, and should also examine PTSD symptom clusters, given the strong negative affect inherent in numbing/dysphoria symptoms (Watson, 2009).

There were several limitations across all studies that constrain conclusions and should be highlighted. All three studies of this dissertation were limited by their cross-sectional nature. Longitudinal work would be needed to verify the causal relationships suggested by the mediation analyses of these studies, especially those involving posttraumatic cognitions in which the causal
direction could be interpreted in multiple ways. All the studies of this dissertation, and the conclusions drawn from them, are also limited to the sample’s population, treatment seeking male combat veterans with PTSD. Additional work would be needed in other samples, such as community samples, other trauma types, and women, before conclusions can be generalized. Furthermore, these studies did not statistically control for the presence of other anxiety disorders or substance use problems, which could account for some of the variance observed in our results. This is especially pertinent in Paper 1, where substance use could also be impacting the relationships between PTSD and anger.

Future research should continue to explore the associations of PTSD symptom clusters, comorbid depression, and other clinical correlates of PTSD. This is especially pertinent for negative outcomes that have been found to be associated with both PTSD and depression, such as financial problems (Elbogen, Johnson, Wagner, Newton, & Beckham, 2012). While empirical investigation using DSM-IV assessment methods is likely to provide important insights, especially research examining the PTSD numbing cluster, future research should utilize the DSM 5 criteria when at all possible. Given the increased importance of negative affect in the new conceptualization, understanding the impact of this new symptom cluster on clinical outcomes, as well as its relationship with comorbid depression, will become increasingly important. Likewise, research examining concurrent treatment of comorbid PTSD and depression, both from disorder-specific and transdiagnostic perspectives, is needed. Finally, while the DSM 5 is currently the accepted nosology, research should continue examining the Quadripartite Model, as well as alternate classifications methods. Research focusing on ways to incorporate and assess
the negative affect symptoms of PTSD, while increasing diagnostic specificity and reducing comorbidity, will be especially important.
Supplementary Table 1

**Substance Use and Anxiety Disorder Comorbidity Rates of Study One**

<table>
<thead>
<tr>
<th>Disorder</th>
<th>N</th>
<th>(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Substance Use Disorders</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol Abuse</td>
<td>5</td>
<td>(5.4%)</td>
</tr>
<tr>
<td>Alcohol Dependence</td>
<td>10</td>
<td>(10.8%)</td>
</tr>
<tr>
<td>Marijuana Abuse</td>
<td>1</td>
<td>(1.1%)</td>
</tr>
<tr>
<td>Marijuana Dependence</td>
<td>4</td>
<td>(4.3%)</td>
</tr>
<tr>
<td>Other Substance Abuse</td>
<td>1</td>
<td>(1.1%)</td>
</tr>
<tr>
<td>Other Substance Dependence</td>
<td>2</td>
<td>(2.2%)</td>
</tr>
<tr>
<td><strong>Anxiety Disorders</strong></td>
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<td></td>
</tr>
<tr>
<td>Panic Disorder</td>
<td>9</td>
<td>(9.8%)</td>
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<tr>
<td>Agoraphobia</td>
<td>3</td>
<td>(3.3%)</td>
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<tr>
<td>Social Phobia</td>
<td>11</td>
<td>(12.2%)</td>
</tr>
<tr>
<td>Specific Phobia</td>
<td>3</td>
<td>(3.3%)</td>
</tr>
<tr>
<td>Generalized Anxiety Disorder</td>
<td>9</td>
<td>(9.9%)</td>
</tr>
</tbody>
</table>

*Note: All values listed are for current substance use and anxiety disorders, as assessed by the Structured Clinical Interview for DSM-IV (SCID). Total study sample size was 97; some percentages were based on smaller sample sizes. While current substance dependence diagnoses were exclusion criteria for the parent study, veterans with these diagnoses are included in the current sample because they were given a PTSD diagnosis before being excluded from the parent study, or they demonstrated sufficient sobriety and remission to warrant inclusion in the parent study. Additionally, strict differential diagnosis rules were used when assessing anxiety disorders. Because PTSD symptoms accounted for comorbid anxiety symptoms in most cases, comorbid diagnoses were often not made.*
Supplementary Table 2

**Substance Use and Anxiety Disorder Comorbidity Rates of Study Two**

<table>
<thead>
<tr>
<th>Disorder</th>
<th>N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Substance Use Disorders</strong></td>
<td></td>
</tr>
<tr>
<td>Alcohol Abuse</td>
<td>5 (3.2%)</td>
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<tr>
<td>Alcohol Dependence</td>
<td>19 (12.0%)</td>
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<tr>
<td>Marijuana Abuse</td>
<td>5 (3.2%)</td>
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<tr>
<td>Marijuana Dependence</td>
<td>5 (3.2%)</td>
</tr>
<tr>
<td>Other Substance Abuse</td>
<td>1 (0.6%)</td>
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<tr>
<td>Other Substance Dependence</td>
<td>3 (1.9%)</td>
</tr>
<tr>
<td><strong>Anxiety Disorders</strong></td>
<td></td>
</tr>
<tr>
<td>Panic Disorder</td>
<td>18 (11.6%)</td>
</tr>
<tr>
<td>Agoraphobia</td>
<td>4 (2.6%)</td>
</tr>
<tr>
<td>Social Phobia</td>
<td>12 (7.9%)</td>
</tr>
<tr>
<td>Specific Phobia</td>
<td>4 (2.6%)</td>
</tr>
<tr>
<td>Generalized Anxiety Disorder</td>
<td>9 (5.7%)</td>
</tr>
</tbody>
</table>

*Note:* All values listed are for current substance use and anxiety disorders, as assessed by the Structured Clinical Interview for DSM-IV (SCID). Total study sample size was 158; some percentages were based on smaller sample sizes. While current substance dependence diagnoses were exclusion criteria for the parent study, veterans with these diagnoses are included in the current sample because they were given a PTSD diagnosis before being excluded from the parent study, or they demonstrated sufficient sobriety and remission to warrant inclusion in the parent study. Additionally, strict differential diagnosis rules were used when assessing anxiety disorders. Because PTSD symptoms accounted for comorbid anxiety symptoms in most cases, comorbid diagnoses were often not made.
Supplementary Table 3

**Substance Use and Anxiety Disorder Comorbidity Rates of Study Three**

<table>
<thead>
<tr>
<th>Disorder</th>
<th>N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Substance Use Disorders</strong></td>
<td></td>
</tr>
<tr>
<td>Alcohol Abuse</td>
<td>4 (2.7%)</td>
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<tr>
<td>Alcohol Dependence</td>
<td>17 (11.3%)</td>
</tr>
<tr>
<td>Marijuana Abuse</td>
<td>5 (3.4%)</td>
</tr>
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<td>Marijuana Dependence</td>
<td>4 (2.7%)</td>
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<tr>
<td>Other Substance Abuse</td>
<td>1 (0.7%)</td>
</tr>
<tr>
<td>Other Substance Dependence</td>
<td>2 (1.3%)</td>
</tr>
<tr>
<td><strong>Anxiety Disorders</strong></td>
<td></td>
</tr>
<tr>
<td>Panic Disorder</td>
<td>18 (12.2%)</td>
</tr>
<tr>
<td>Agoraphobia</td>
<td>4 (2.7%)</td>
</tr>
<tr>
<td>Social Phobia</td>
<td>12 (8.3%)</td>
</tr>
<tr>
<td>Specific Phobia</td>
<td>4 (2.8%)</td>
</tr>
<tr>
<td>Generalized Anxiety Disorder</td>
<td>9 (6.2%)</td>
</tr>
</tbody>
</table>

*Note:* All values listed are for *current* substance use and anxiety disorders, as assessed by the Structured Clinical Interview for DSM-IV (SCID). Total study sample size was 150; some percentages were based on smaller sample sizes. While current substance dependence diagnoses were exclusion criteria for the parent study, veterans with these diagnoses are included in the current sample because they were given a PTSD diagnosis before being excluded from the parent study, or they demonstrated sufficient sobriety and remission to warrant inclusion in the parent study. Additionally, strict differential diagnosis rules were used when assessing anxiety disorders. Because PTSD symptoms accounted for comorbid anxiety symptoms in most cases, comorbid diagnoses were often not made.
References


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