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TREATMENT MATCHING IN PTSD: A CONFIRMATORY FACTOR ANALYSIS BASED ON THERAPEUTIC MECHANISMS OF ACTION

by

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ABSTRACT

The current study takes an initial step toward deriving a method for empirically based, theory-driven treatment matching in a military population suffering from PTSD. Along with the more overt symptoms of PTSD (e.g., persistent hyperarousal), secondary cognitive symptoms have also been shown to be significantly associated with avoidance and intrusive symptoms, as well as contribute to functional impairment. Based on the factor analytic and treatment literature for PTSD, it appears that there are two central mechanisms associated with beneficial therapeutic change that underlies both CPT and PE treatments (i.e., habituation, changes in cognitions). Additionally, different traumatic events and peritraumatic responses may be associated with unique symptom profiles and may necessitate targeted treatment. The present study proposes a novel approach to treatment matching based on the factor structure of PTSD and underlying mechanisms of treatment response. More broadly, this paper provides evidence for a broader understanding of peritraumatic responses and the potential implications of these responses for symptom profiles and illness trajectories.

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LIST OF ACRONYMS

AMOS	Analysis of Moment Structures
CAPS	Clinician Administered PTSD Scale
CFA	Confirmatory Factor Analysis
CFI	Comparative Fit Index
CPT	Cognitive Processing Therapy
CR	Cognitive Restructuring
DSM	Diagnostic and Statistical Manual of Mental Disorders
GLS	Generalized Least Squares
GFI	Goodness of Fit Index
HAM_D	Hamilton Rating Scale for Depression
IBM	International Business Machines
ICC	Intraclass Correlation Coefficient
IED	Improvised Explosive Devise
MDD	Major Depressive Disorder
ML	Maximum Likelihood
OEF	Operation Enduring Freedom
OIF	Operation Iraqi Freedom
PCFI	Parsimony Comparative Fit Index
PE	Prolonged Exposure
PTSD	Post-Traumatic Stress Disorder
QOLI	The Quality of Life Inventory

RMSEA	Root Mean Square Error of Approximation
SCID-I	Structured Clinical Interview for the DSM-IV, I
SCID-II	Structured Clinical Interview for the DSM-IV Axis II Disorders
SRMR	Standardized Root Mean Square Residual
TBI	Traumatic Brain Injury

CHAPTER ONE: INTRODUCTION

Armed-service members are subjected to a wide array of potentially traumatic events (Hoge et al., 2004; Litz et al., 2009), and exhibit varying peritraumtic responses (Brewin et al., 2000) that may be associated with different clinical presentations and symptom profiles. For example, traumatic experiences characterized by prolonged/unexpected feelings of helplessness (e.g., IED explosion, direct gun fire) may lead to a distinctly different subset of symptoms than traumas associated with guilt/shame (e.g., witnessing the death of a fellow soldier, harming a child). Researchers have attempted to devise methods to categorize types of traumatic events (Litz et al., 2009) and match these events to specific symptoms (Stein et al., 2012). For example, Stein and colleagues (2012) found that events related to "moral injury" (i.e., events that were in-contrast with the individuals self-schema) predicted re-experiencing and guilt symptoms, whereas trauma related to the aftermath of violence predicted negative cognitions about the world. However, the type of trauma the individual experiences may not be as relevant to treatment as the symptom profile the event elicits.

Based on the PTSD treatment literature, sustained peritraumatic hyperarousal and the formation of persistent negative cognitions have been identified as two distinct, but potentially co-occurring, maladaptive patterns of responding to a traumatic event. These response patterns have been identified as primary sources of functional impairment (Resick & Miller, 2009) and potential targets of therapeutic interventions. Despite the clinical utility of conceptualizing PTSD in these terms, physiological and cognitive symptom clusters have never previously been subject to confirmatory factor analysis. This is likely because traditionally, CFA is used to identify the

factors that comprise the construct of PTSD to assist in theorizing about the origins, prevention, and treatment of the disorder. However, Marshall and colleagues (Marshall et al., 2013) have stated that it is unlikely that one model of PTSD will emerge that best characterizes maladaptive responding to traumatic events. Instead, Marshall and colleagues recommended devising models that are useful for specific purposes. The present confirmatory factor analysis (CFA) aims to devise a method for treatment matching based on the PTSD treatment literature and, specifically, existing factor analytic studies regarding PTSD, which have been the subject of considerable debate.

Currently, the DSM-5 criteria for PTSD (see Table 1) are comprised of four main symptom categories (i.e., intrusive, avoidance, negative cognitive/mood, and hyperarousal) (American Psychiatric Association, 2013). Although there is support for this factor structure (for summary see Friedman et al., 2011), alternative two- (Asmundson et al., 2003), three- (American Psychiatric Association, 2000), four-(Rademaker et al., 2012; Simms, Watson & Doebbelin, 2002), and five-factor (Elhai et al., 2011) models have been proposed. Of these models, the dysphoric and numbing models have received the most attention in the literature, have garnered the most empirical support, and were a primary source of recent changes to the DSM-5 PTSD criteria. The dysphoric and numbing models identify an internalizing symptom presentation that is characterized by symptoms related to a reduction in emotional experience and general emotional distress. The numbing model closely resembles the DSM-IV criteria for PTSD (see Table 1), with the exception of separating the avoidance symptom cluster into effortful avoidance (C1-C2), defined as the willful avoidance of thoughts, people, and places, and emotional numbing (C3-C7). The latter includes amnesia, anhedonia, detachment from others,

restricted range of affect, and a sense of foreshortened future. The dysphoria model also includes this division of the avoidance factor, but combines the numbing symptoms with three arousal symptoms to create a dysphoric factor. The resulting dysphoric factor is based on the clustering of these eight symptoms (i.e., D1-D3 and C3-C7) that include the numbing symptoms along with difficulty sleeping, irritability, and difficulty concentrating. By combining elements of the avoidance and arousal factor, the dysphoria model also creates an additional two-item hyperarousal factor comprised of the remaining arousal symptoms (i.e., hypervigilance and an exaggerated startle response). The authors of the dysphoria model have argued that the clustering of the dysphoric symptoms is associated with a general distress and dysphoria construct common to many anxiety and depressive disorders (Simms et al., 2002) whereas the remaining factors are more characteristic of an anxious traumatic response.

In a recent meta-analysis, the dysphoric model was found to provide marginally superior fit than the numbing model (Yufic & Sims, 2010). However, other researchers have demonstrated that although the dysphoric and numbing models differ conceptually and the overall fit of the dysphoric model has been shown to be marginally superior, the mathematical difference between these two models hinges on the estimation of the correlation between the symptoms in the arousal factor and not on the clustering of the dysphoric symptoms (Elhai et al., 2011; Marshall et al., 2013). The arousal factor in the dysphoric model contains only two symptoms, whereas the arousal factor in the numbing model contains all five symptoms present in the DSM-IV arousal criteria. The superiority of the dysphoric model relies on the underestimation of this two-item correlation in the numbing model and not on the clustering of

the symptoms in the dysphoric factor (Marshall, 2013). Therefore, each of these models equally account for the latent PTSD construct, despite their conceptual difference.

Based on the dysphoric and numbing models, a five-factor model has also been posited (Elhai et al., 2011). This model separates the dysphoric factor into the numbing symptoms (C3-C7) and three general dysphoric symptoms (D1-D3) common to many anxiety and depressive disorders. The five-factor model demonstrated better fit than the dysphoric and numbing models. The researchers suggested that this was because the five-factor model better accounts for the separation of fear-based symptoms present in the re-experiencing and the two-item arousal factors, as well as accounts for the depression-related symptoms represented by the emotional numbing factor (Elahi et al., 2011).

Despite numerous studies investigating the latent and factor structure of PTSD, the evidence is still unclear as to whether the cognitive and dysphoric symptoms of PTSD represent a unique symptom profile necessitating targeted PTSD treatment or whether these symptoms are attributable to comorbid depression (Moore et al., 2009). Due to the conceptual overlap between the internalizing symptoms of PTSD and depression, some researchers have suggested the removal of these symptoms (Brewin, 2009). However, there is increased recognition of the diverse presentation of PTSD (Friedman et al., 2011) and the contribution of dysphoric and anhedonic symptoms in predicting the duration, severity, and functional impairment associated with PTSD (Friedman et al., 2013; Meiser-Stedman et al., 2009; Moser et al., 2007). Additionally, there is substantial evidence for the presence of distinct maladaptive cognitions specifically associated with traumatic stress (Friedman et al., 2011; Resick et al., 2009). Longitudinal evidence has shown a bi-directional relationship between depression and PTSD,

suggesting that depressive symptoms may be a core element of a maladaptive traumatic response (Dekel et al., 2014). Based partially on this evidence a “negative alterations in cognitions and mood” symptom cluster has been included in the DSM-5 comprised of persistent cognitions and numbing symptoms (American Psychiatric Association, 2013).

One potential method for resolving these issues is to uncover a factor structure of PTSD by examining the extensive PTSD treatment literature and underlying mechanism of therapeutic action. Limited empirical evidence exists that supports efficacious treatment for combat veterans (Frueh et al., 2007). Data from available treatment studies suggests that the effect sizes of available psychological treatments are moderate, are associated with high dropout rates, and are minimally beneficial or ineffective for a substantial portion of patients (Bradly et al., 2005; Roberts et al., 2009). Given the complex role of both heightened physiological arousal and cognitive distress in PTSD, two main cognitive-behavioral treatments have emerged from the PTSD treatment literature. Cognitive Processing Therapy (CPT) and Prolonged Exposure Therapy (PE) are the most widely studied and supported treatments for PTSD, and are primary psychological interventions utilized by the Veteran Affairs Health facilities for the treatment of combat veterans (Foa, Keane, Friedman, & Cohen, 2009). Multiple randomized control trials (RCTs) and meta-analyses have provided evidence that CPT and PE have relatively equivalent beneficial outcomes (Foa et al., 2009; Foa, Rothbaum, Riggs, & Murdock, 1991; Resick & Schnicke, 1992) despite different theoretical mechanisms of change. The Emotion Processing Theory, underlying PE, posits that repeated exposure to the anxiety-provoking stimuli facilitates the naturally occurring process of habituation (Foa & Kozak, 1986). This process allows new learning to occur, resulting in traumatic memories no longer eliciting heightened physiological

responses and emotional distress (Foa, Hearst, Dancu, Hembree, & Jaycox, 1994). Utilizing a different approach, and based on social cognitive theory, CPT attempts to promote recovery through an examination of the meaning of the traumatic event (Resick & Schnicke, 1992). Repeated altering of maladaptive cognitions and the integration of the traumatic event into the patient's "self-schema" is theorized to reduce the secondary emotions (e.g., depression, guilt, self-blame) and intrusive recollections associated with PTSD (Resick, Monson, & Chard, 2010).

Although different mechanisms of change are suggested by these theories, empirical evidence suggests that these changes do not occur in isolation (Gallagher & Resick, 2012; Zalta et al., 2013). Meta-analytic (Benish, Imel, & Wampold, 2008; Resick et al., 2002) and individual research studies have shown that, through a yet unidentified mechanism, cognitive changes occur during PE (Zalta et al., 2013), and that some techniques used in CPT (e.g., writing of traumatic scenes) have been likened to exposure and habituation. This evidence supports the reduction of both cognitive and physiological symptoms of PTSD, regardless of treatment type (Gallagher & Resick, 2012; Resick et al., 2002). Recent empirical evidence suggests that although PE and CPT both provide beneficial changes in cognitions and physiological arousal, the different underlying mechanisms of change promote increased recovery in their respective domain (Gallagher & Resick, 2012). Specifically, Gallagher and Resick found that PE resulted in a decrease in PTSD symptoms as mediated by habituation, independent of cognitive changes in hopelessness, whereas CPT resulted in similar decreases in PTSD symptoms, as mediated by greater changes in hopelessness (Gallagher & Resick, 2012). However, in this study, PE was still shown to significantly reduce hopelessness, and more recent research has provided further evidence for adaptive changes in cognitions occurring during PE treatment (Zalta et al., 2013).

Although these results appear to suggest different changes that may be best utilized by combining treatments, several studies have explored combining PE with cognitive restructuring (Foa & Rauch, 2004; Moser, Cahill, & Foa, 2010). Unfortunately, these studies have shown that this approach did not enhance the efficacy of either treatment. One potential explanation for these findings may be that different PTSD patients respond to different treatment modalities. For example, several researchers have suggested that a possible explanation for a patient's lack of improvement after PE may be an over-fixation on the meaning of the trauma that interferes with the habituation process (Tarrier et al., 1999). However, a precise method for separating individuals with PTSD into distinct categories has proven difficult.

One study providing information relevant to treatment matching in PTSD retrospectively examined the effects of PE and Cognitive Restructuring (CR), with PTSD patients reporting higher levels of negative trauma-related cognitions (Moser, Cahil, & Foa, 2010). The results from this study suggest that individuals receiving the combined treatment fared worse than those patients receiving PE alone. However, several confounds could explain the results from this study. Although patients did not differ on initial assessments of PTSD symptom severity, it is possible that patients who report heightened physiological symptoms coupled with secondary cognitive symptoms associated with PTSD are experiencing a greater level of overall distress as compared to patients with primarily physiological symptoms. Additionally, in this particular study, the length of treatment sessions was identical in each condition despite the addition of therapy content. As a result, content from the PE condition (e.g., discussions of anxiety) was sacrificed in order to accommodate CR. Given the absence of dismantling studies in the literature, essential components of PE may have been removed in this study as research has

shown that affective labeling is associated with greater gains in exposure therapy (Kircanski, Leiberman, & Craske, 2012). Additionally, not providing adequate time for CR suggests that perhaps neither treatment was provided adequately. Despite these methodological limitations, the findings from numerous studies suggest that PE alone has a beneficial effect on cognitions (Foa & Rauch, 2004; Gallagher & Resick, 2012; Resick et al., 2013; Zalta et al., 2013), and that this effect may be a potential mechanism of beneficial therapeutic change (Zalta et al., 2013). However, the administration of CPT appears to achieve greater changes in these cognitions by directly addressing them (Gallagher & Resick, 2012; Resick et al., 2013), and combining these treatments may be contraindicated.

Existing studies point to adjustments in maladaptive cognitions, and the facilitation of habituation as primary targets of intervention and essential components of achieving desired treatment outcome (Keane & Barlow, 2002; Resick, 2001). However, treatments targeting these two mechanisms do not work for all individuals with PTSD (Schnurr et al., 2007), and both CPT and PE report a significant dropout rate (≈20 percent) (Bryant et al., 2007). The existing literature indicates that individual characteristics associated with dropout include catastrophic cognitions and higher avoidance (Bryant et al., 2007). Thus, the perception that these symptoms are not being targeted or improved upon in treatment may lead an individual to withdraw early from the intervention.

The empirical evidence summarized above suggests that distinct response patterns to PTSD therapy may be associated with different underlying mechanisms that may necessitate targeted treatment. The first step in disentangling the contribution of different trauma types or maladaptive response patterns is, therefore, to identify a reliable way to detect specific symptom

profiles that may respond differently to empirically supported interventions. In response, the current study conducted a CFA based on mechanisms of beneficial therapeutic change that may inform treatment matching.

CHAPTER TWO: METHOD

Participants

Data was collected as part of a Department of Defense-funded RCT that is treating Operation Enduring Freedom (OEF, Afghanistan) and Operation Iraqi Freedom (OIF) combat veterans diagnosed with PTSD. Veterans participated in either a 3-week or 17-week treatment condition that began with a variety of pre-treatment symptom and diagnostic measures. Participants were paid $50 for completing the pre-treatment assessment. The sample consisted of both third-party mandated and treatment-seeking veterans. Inclusion criteria required a PTSD diagnosis confirmed by supervised clinicians. Twenty percent of diagnostic interviews were randomly selected for Inter-rater reliability analysis. This analysis revealed excellent consistency on rating CAPS total scores (ICC=.996 ~~K~~=1.00) and PTSD diagnosis (k=1.00). To collect a representative sample, minimal exclusion criteria were used to derive the original sample. Participants were only excluded if they had a significant history of cardiac symptoms that could have potentially interfered with treatment, an acute substance abuse disorder that prevented the participant from demonstrating two weeks of abstinence, medications that could not be stabilized for two weeks, or the participant met criteria for antisocial personality disorder. Due to the nature of the OEF and OIF conflicts resulting in high rates of Traumatic Brain Injury (TBI) (Shively & Perl, 2012; Vasterling, Verfaellie, & Sullivan, 2009), veterans diagnosed with a TBI were included in the original sample.

The original sample included 150 OEF/OIF veterans, (139 males; 11 females) between the ages of 21 and 63 years (M_{Age}= 35.32 *SD*=9.54). Among the sample, 26% were on active

duty, 42% reported a history of a TBI diagnosis, and 51.3% received service-connected disability at the time of pre-assessment. In regards to their service, 61.3% had served in the Army, 26.7% in the Marines, 6% in the Air Force, 5.3% in the Navy, and 0.7% as Civilian Contractors. For 52% of the veterans in the original sample, high school was their highest level of education, whereas, 12.7% completed some high school. 25.3% had completed some college, 4.7 % had received a bachelor's degree, and 1.3% had a master's degree. 4% of the sample did not report their highest level of education. Of the veterans, 55.2% identified as White, 13.4% as Black/African American, 24.7% as Hispanic, 2% as Asian, 2% as Biracial, and 2.7% identified as Other. In regards to their relationship status, 33.7% were Single, 43.3% were Married 8.7% were Separated, 13.6% were Divorced and 0.7% were in a Domestic Partnership.

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Measures

Clinician-Administered PTSD Scale (CAPS)

The CAPS (Blake et al., 1990; Weathers & Litz, 1994) is a 30-item semi-structured interview that assesses the DSM-IV criteria for PTSD. Additionally, the CAPS includes dual (i.e., frequency and severity) ratings of the 17 PTSD symptoms and questions targeting the social and occupational impairment associated with PTSD. The CAPS interview allows clinicians to gain additional detail and insight into the patient's trauma and subsequent impairment in functioning.

Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I)

The SCID-I (First et al, 1996) is a semi-structured clinical diagnostic interview that includes major DSM-IV-TR Axis I diagnostic classes. The SCID was administered to the original sample to assess for comorbid diagnoses such as depression, as well as to confirm the diagnosis of PTSD.

Structured Clinical Interview for DSM-IV Axis II Disorders (SCID-II)

The SCID-II Self-Report (First et al., 1997) is a 119-item measure for assessing the 11 Axis II personality disorders from the DSM-IV-R (4th edition, revised). Specific questions were selected (see analytic strategy below) that best represented maladaptive cognitions associated with PTSD. The DSM-5 Negative Cognitions and Mood criteria and the CPT treatment manual were used to inform the selection of these items. Although the SCID-II is most often used to assess the occurrence of personality based psychopathology, this self-report measure contains a wealth of information related to a participant's clinical presentation. A limited number of items were drawn from each of the SCID-II categories, and, therefore, the amount of items selected was not

sufficient to qualify for a SCID-II personality diagnosis. Additionally, the SCID-II self-report was designed as a screening measuring and is not recommend for use in isolation to diagnose personality disorders (SCID-II User's Guide; First et al., 1997) due to the conceptual overlap between distress-related Axis-I disorders and personality-based psychopathology.

The Quality of Life Inventory (QOLI)

The QOLI (Frisch et al., 1993) is a 32-item measure assessing the importance and satisfaction of 16 life domains: Health, Self-Esteem, Goals and Values, Money, Work, Play, Learning, Creativity, Helping, Love, Friends, Children, Relatives, Home, Neighborhood, and Community. These 16 items are rated on a 3-point Likert scale for importance and a 6-point Likert scale for satisfaction. The item representing self-esteem was selected to assess the esteem component of CPT and the maladaptive self-blame cognition.

Hamilton Rating Scale for Depression (HAM_D)

The HAM_D (HAMD; Hamilton, 1959) is a 17-item measure assessing the symptoms of depression and is one of the most widely used scales to assess depressive symptomatology. Specific items were selected (see analytic strategy) to differentiate maladaptive cognitions related to the traumatic event and items related to comorbid depressive symptomatology.

Overall Assessment Strategy

Analytic Strategy

The original sample is thought to be highly representative of a veteran population. Researchers have recommended the inclusive sampling method implemented in this study to best generalize research findings to veteran populations (Frueh, Mirabella, & Turner, 1995). The

sample size of 150 was supported based on best-practices for Confirmatory Factor Analysis (CFA) suggesting the practicality of conducting a CFA with lower sample sizes when examining an established factor structure in a unique population (MacCallum, et al., 1999). The CFA was conducted using IBM SPSS AMOS 21(Arbuckle, 2006) to perform estimation techniques, fit indices, and comparative evaluations of competing models. Items from the CAPS assessing PTSD symptoms were supplemented with selected items from the SCID-II, QOLI, and HAMD.

Many of the existing factor-analytic models examining PTSD have been based on establishing the underlying structure of the disorder. Although these theoretical models are essential for establishing diagnostic criteria and uncovering new targets of intervention, the direct treatment utility of these models is limited. Therefore, the present analysis aimed to build on aspects of the established dysphoric and numbing models proposed by Simms and colleagues (2002) and King and colleagues (1998). Additionally, questionnaire items were incorporated to establish a cognitive subset of symptoms corresponding to the central tenets of CPT (see Table 2) and consistent with DSM-5 negative cognition criteria for PTSD (see Table 3).

The present CFA aimed to construct a model (see Figures 1 & 2) that was based on the mechanisms of action of PE and CPT interventions with the goal of treatment matching. The proposed model contained two hypothesized latent variables comprised of cognitive or physiological symptoms; both stemming from a larger PTSD construct. Although PE and CPT attribute the occurrence and maintenance of symptoms to different underlying causes (fear conditioning vs. maladaptive cognitions), some shared symptoms could theoretically result from either of these causes. Therefore, symptoms shared between the cognitive and hyperarousal constructs are represented in Figure 1 by three latent variables (e.g., re-experiencing symptoms,

anxious arousal, and effortful avoidance) that have been validated in Elhai and colleagues' five-factor model (2011). However, other CFA studies have proposed a two factor model that combines re-experiencing and hyperarousal symptoms (Buckley, Blanchard, & Hickling, 1998). Therefore, two separate models were examined to test the validity of the shared symptom hypothesis. The shared symptom model hypothesizes shared symptom clusters (anxious arousal & re-experiencing symptoms); the specified symptom model identifies these symptoms as part of the physiological symptom construct.

Previous factor-analytic studies have also validated the distinct factor loadings for effortful avoidance symptoms demonstrating that criterion C1 (efforts to avoid thoughts, feelings…associated with the trauma) and C2 (efforts to avoid activities….that arouse recollections of the trauma) load separately from other, more cognition-based avoidance strategies. These cognition-based avoidance strategies (C4-C7) are referred to in other models as emotional numbing, and are therefore clustered under the latent variable of "emotional numbing" with the omission of C3 (inability to recall important aspects of the trauma). Criterion C3 was included as an "anxious arousal" shared variable because it has previously demonstrated poor fit with the emotional numbing construct (King, Leskin, King, & Weathers, 1998), and could theoretically result from either sustained hyperarousal or rumination on trauma related cognitions. Previous studies have also demonstrated evidence for memory impairments associated with pathological anxiety (Boldrini et al., 2005). Therefore, in the specified symptom model, C3 falls under the physiological symptom cluster.

In addition to emotional numbing, the cognitive symptom construct was based on the theoretical mechanisms of action for CPT as well as the DSM-5 criteria for negative alterations

in cognitions and mood. This construct was comprised of several SCID-II items in addition to items derived from other measures including the QOLI and HAM_D, which correspond to symptoms targeted by CPT (e.g., negative beliefs, distorted blame of self and others, guilt). In CPT treatment, these cognitions are addressed through specific modules targeting concerns over guilt, self-blame, safety, trust, control, and esteem.

The physiological symptom construct (see Table 4) has also been partially supported in previous studies examining the dysphoria model which have demonstrated that symptom D4 (hypervigilance) and D5 (exaggerated startle response) load onto a separate hyperarousal construct. Additionally, these models have also shown that D1 (difficulty falling asleep), D2 (irritability and outburst of anger), and D3 (difficulty concentrating) also load independently from the main hyperarousal variables. Therefore, theory justified the separation of these constructs. In addition to the established hyperarousal construct, criterion B5 (physiological reactivity to exposure cues) was included in the physiological arousal factor as exposure therapy is theorized to best target this type of conditioned fear response. This symptom is traditionally subsumed under intrusion/re-experiencing symptom category; however, for the purposes of this model, it was included as a physiological symptom.

Some researchers have suggested that the cognitive symptoms observed in patients with PTSD are more attributable to comorbid depression (Brewin et al., 2009), whereas others have suggested that these cognitive symptoms are unique to PTSD (Friedman et al., 2011). Therefore, the proposed model also included a latent depression variable that was comprised of four manifest variables that are more consistent with a depression diagnosis, but are not part of the DSM-5 criteria for PTSD (e.g., depressed mood, loss of appetite, loss of weight, and

psychomotor retardation). The first of these variables was the SCID-I diagnosis of Major Depressive Disorder (MDD), and the others were symptom-derived from the HAM_D depression items.

The first phase of the analysis evaluated the fit of the shared symptom model as compared to the specified symptom treatment matching model to determine the best model for continued model evaluation. The next phase of the proposed analysis evaluated the validity of the treatment matching model by comparing it to three additional models. As a primary point of comparison, the proposed model was first compared to a simplistic model containing all 17 PSTD symptoms with the addition of the cognitive items and no latent variables. The treatment matching model was then compared to the DSM-IV and DSM-5 models. Other researchers have employed this method to demonstrate the validity of their model (Simms et al., 2002). In the second phase of analysis, the proposed model was then compared to four models including depression with the goal of establishing the role of depression in the occurrence of cognitive symptoms. The second phase of analysis initially evaluated if the correlation hypothesis depicted in Figure 5 and 6 between depression and PTSD was accurate. The series of models (Figures 5 to 8) also evaluated the independent nature of the cognitive symptom construct to determine if the variance in this construct was more attributable to PTSD or comorbid depression.

Data Preparation

The analysis was estimated using Generalized Least Squares (GLS) as this method has been shown to be robust to smaller sample sizes and non-normal data (Hu & Bentler, 1999). However, after a simplification of the hypothesized treatment matching model, maximum likelihood (ML) estimation was also appropriate and reported. Consistent with SEM best

practices (Hu & Bentler., 1999), model fit was assessed using several fit indices. Model fit was assessed primarily using the comparative fit index (CFI) and standardized root mean square residual (SRMR), as both have been shown to be robust to smaller sample sizes (Bentler, 1988). The root mean square error of approximation (RMSEA) was also examined to assist with interpretation. Cases with more than 10% missing data were excluded resulting in 3 participants be excluded from the analysis. The remainder of the missing data was estimated using the series mean for CAPS, HAM_D and the QOLI and the mean of nearby points for the SCID-II. The SCID-II nearby points estimation technique was implemented because the grouped dichotomous item structure of the measures where in questions are grouped to reflect related constructs.

CHAPTER THREE: RESULTS

Latent variables representing symptom categories (e.g., re-experiencing, effortful avoidance, emotional numbing) were removed to reduce the complexity of the proposed model. The removal of these latent variables allowed for a more direct examination of the validity of physiological and cognitive symptom constructs. A comparison of two proposed models revealed that fit was similar whether hypothesized shared symptoms were specified uniquely (CFI=.815; RMSEA=.057; SRMR=.08; Figure 4) or simultaneously (CFI=.816; RMSEA= .057; SRMR=.08; Figure 3) to the cognitive and physiological symptom constructs. Only effortful avoidance symptoms (C6 and C7) were shared between constructs in the unique specification model, whereas emotional numbing symptoms and re-experiencing symptoms were modeled as indicators of the cognitive construct and physiological construct, respectively. However, since more specified paths always lead to better model fit, parsimony fit indices were examined to account for the number of paths drawn. Parsimony fit indices revealed that the unique specification model (PCFI=.742) demonstrated better fit than the shared symptoms model (PCFI=.707). As a result, the unique specification model was used for the remainder of model testing.

The hypothesized treatment matching model was then compared to three well-established models: a simplistic model containing all 17 PSTD symptoms with the addition of the cognitive items, the DSM-IV 3-factor model and the DSM-5 4-factor model. The chi square for the hypothesized treatment matching model was significant ($\chi^2(311)=460.374$, $p<.001$), which typically indicates poor fit. However, chi square is an absolute fit index that may be overly sensitive to small differences between the observed and predicted covariance matrices

(Tabachnick & Fidell, 2013). Therefore, the standardized root mean square residual (SRMR) and the root mean square error of approximation RMSEA were also examined. They indicated marginal (SRMR=.08) and good fit (RMSEA=.057), respectively. Due to the small sample size, the Generalized Least Squares (GLS) estimation method was also implemented and revealed a goodness of fit index (GFI) that suggested marginal fit (GFI=.829), whereas RMSEA and SRMR suggested good fit (RMSEA=.027) and poor fit (.10), respectively. The inconsistency between these fit indices usually reflects a model requiring additional specification or revision. Overall, across several estimation techniques and fit indices, the hypothesized treatment matching model demonstrated marginal fit; however, two measure items did not significantly contribute to the model. Specifically, the cognitive items judging others harshly (SCID-II_37), believing that most people are no good (SCID-II _38) and not being able to forgive people for past grievances (SCID -II_46) did not account for a significant portion of the variance. However, these items did account for variance in the DSM-5 Model. Compared to alternative models, however, the treatment matching model demonstrated better fit than the simplistic PTSD model the DSM-IV model, and the DSM-5 model (See Tables 6 & 7).

Models were also examined to uncover the relationship between PTSD, depressive symptomatology, and cognitive symptoms. This analysis revealed that models containing a depressive construct demonstrated equally marginal fit (See Tables 8 & 9) regardless of correlation or causal hypotheses. Therefore, a statistical comparison of these models was not conducted.

CHAPTER FOUR: DISCUSSION

Researchers have begun to acknowledge a diverse range of post-trauma symptoms and the subsequent impairment associated with multiple types of traumatic events. In particular, combat soldiers experience multiple types of potentially traumatic events such as direct combat, hostage situations, and violations of personal moral standards (i.e., moral injury). Recent evidence suggests that different types of traumatic events may elicit different symptoms profiles (Stein et al., 2009). Moreover, two theoretically distinct treatments, PE and CPT, have emerged from the literature as two efficacious methods for treating these symptoms in veterans (Foa, Keane, Friedman, & Cohen, 2009) and appear to have relatively equivalent outcomes. Our investigation attempted to validate a treatment matching model that contained factors directly related to the central mechanisms of action within PE and CPT. The physiological symptom factor in the proposed model included symptoms (e.g., hyperarousal, flashbacks, physiological reactivity to exposure cues) theorized to be most amenable to Emotion Processing Theory and habituation-based treatments such as PE. The cognitive symptom factor included items representing the DSM-5 PTSD maladaptive cognition criteria (e.g., guilt & negative expectations) and aligned with social cognition theory as well as therapeutic mechanisms essential to CPT.

The resulting treatment matching model demonstrated marginal fit; however, multiple fit indices revealed that our model fit the data better than several established models including the DSM-IV and DSM-5 models. Furthermore, specific symptom loadings varied in magnitude (See Tables 10 & 11) whereas physiological (β=.654) and psychological (β=.650) reactivity and difficulty concentrating (β=.597) demonstrated the best fit with the physiological construct (in

treatment matching model). The highest loadings for the cognitive construct were items reflecting a sense of foreshortened future (β=.555), self-esteem (β= -.560) and anhedonia (β=.613).

Although this study provides some initial support for the treatment matching model and can be utilized to refine further model testing, there are several limitations worth noting. The first of these limitations is associated with the items that defined the cognitive symptom construct. In a recent revision to the DSM criteria for PTSD, several cognitive symptoms were added that more broadly defines the disorder to include multiple cognitive distortions in addition to heightened physiological reactivity. Given this recent revision, several cognitive symptoms were not measured during the ongoing PTSD randomized clinical trial from which data for this investigation were drawn. As a result, items from other measures (HAM_D, SCID-II) were extracted instead to best represent these constructs. Although these items did appear to match the DSM-5 criteria, the items were not specifically related to PTSD nor previously established as valid measures of trauma-related cognitions. Additionally, several of these items were dichotomous and may have limited the variance captured by the model.

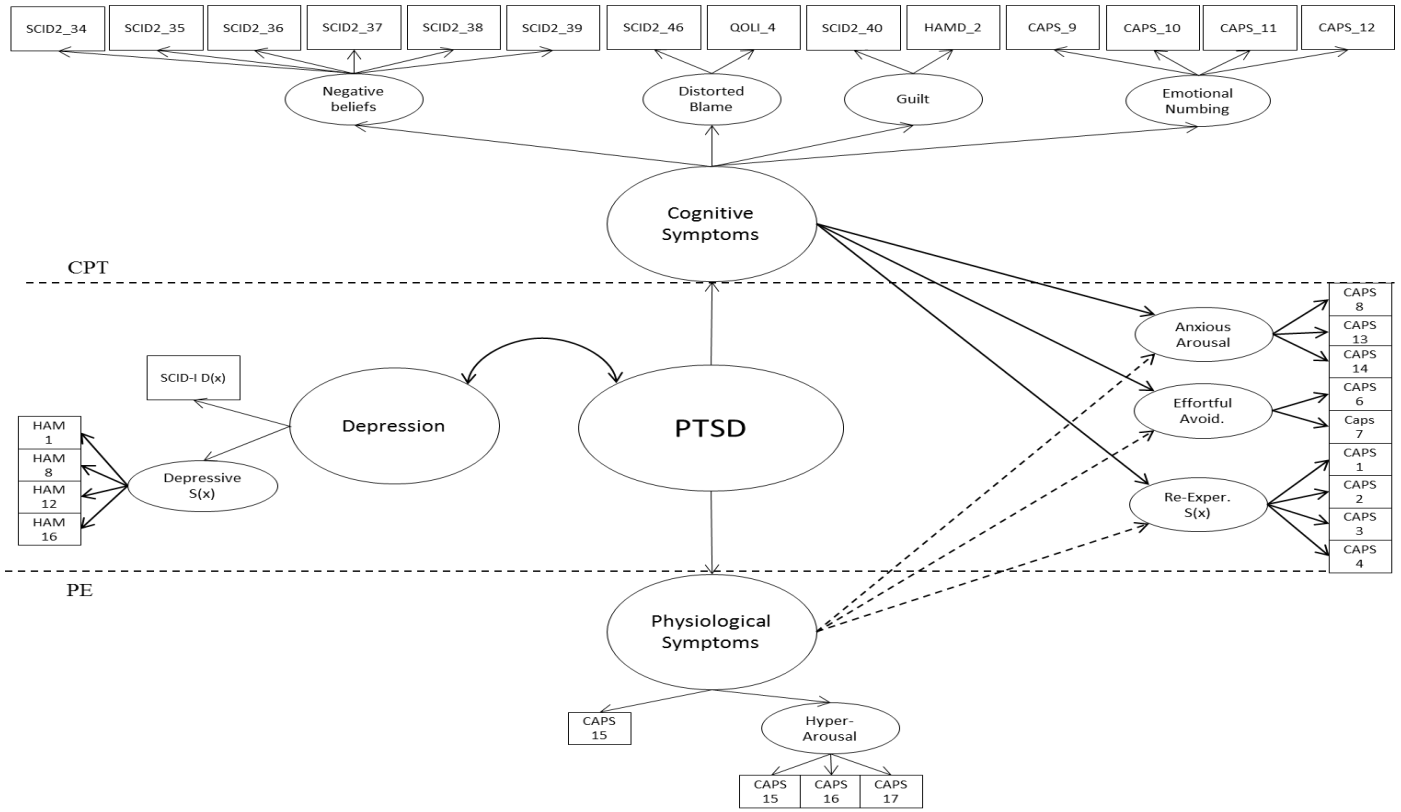
The conclusions of this study may also be limited by the treatment modalities offered to our sample. Data from two treatment conditions (3-week & 17-week) were collapsed to obtain sufficient power for model testing. Examining the samples separately revealed significant differences in overall PTSD severity as well as multiple individual symptoms representing the cognitive construct. It is possible that individuals with greater PTSD severity present with a unique symptom profile not represented by our model. Additionally, the data used for model testing was gathered from a sample of treatment seeking veterans. It is possible that veterans

electing to participate in this trial self-selected based on specific symptoms and the perceived benefits explained to them by study recruiters. Furthermore, individuals with a cognitive based symptom profile may be less likely to seek out treatment trials due to symptoms such as anhedonia and detachment from others.

The results of this study also raise interesting questions regarding the co-occurrence of depression and PTSD. Few conclusions can be drawn due to the marginal fit demonstrated by these models (Figures 5 through 8). With this in mind, some information can be drawn from these models to inform future model testing. For example, the models examining a causal relationship between the depression construct and the cognitive construct demonstrated worse fit relative to models containing an uninfluenced cognitive construct. Furthermore, the addition of the depression factor did not significantly reduce the overall model fit, suggesting that comorbid depressive symptomatology is an important consideration for future model testing. However, these differences were small and interpreting association within marginal fitting models should be done with caution. Overall, the overlap of the cognitive criteria within PTSD and depression remains an open question that requires further investigation.

This is the first study to attempt to devise a model based on therapeutic mechanisms of action. Based on our findings, future research should continue to refine and develop models for the purpose of treatment matching. PTSD is an ideal candidate for this technique due to the development of two theoretically distinct efficacious treatments and need for improved treatment outcomes. Further research should also continue to uncover the mechanisms of action underlying these treatments and better incorporate this information to refine and more effectively deliver existing treatments.

APPENDIX A: FIGURES



Manifest Variables:

SCID2_34	Do you believe that you are basically an inadequate person and often don't feel good about yourself?
SCID2_35	Do you often put yourself down?
SCID2_36	Do you keep thinking about bad things that have happened in the past or worry about bad things that might happen in the future?
SCID2_37	Do you often judge others harshly and easily find fault with them?
SCID2_38	Do you think that most people are basically no good?
SCID2_39	Do you almost always expect things to turn out badly?
SCID2_40	Do you often feel guilty about things you have or haven't done?
SCID2_46	Are there many people you can't forgive because they did or said something to you a long time ago?
QOLI_4	How satisfied are you with your self-esteem?
HAMD_2	Feelings of Guilt: Do you often brood about past mistakes?

CAPS_1	Intrusive Recollections
CAPS_2	Distressing Dreams
CAPS_3	Acting or feeling as if the event were recurring
CAPS_4	Psychological distress at exposure cues
CAPS_5	Physiological reactivity on exposure to cues
CAPS_6	Avoidance of thoughts or feelings
CAPS_7	Avoidance of activities, places, or people
CAPS_8	Inability to recall important aspects of the trauma
CAPS_9	Diminished interest in activities
CAPS_ 10	Detachment & Estrangement
CAPS_11	Restricted range of affect
CAPS_12	Sense of Foreshortened Future
CAPS_13	Difficulty falling or staying asleep
CAPS_14	Irritability or outbursts of anger
CAPS_15	Difficulty Concentrating
CAPS_16	Hypervigilance
CAPS_17	Exaggerated Startle Response

Figure 1. Hypothesized shared symptom treatment-matching model with manifest variables

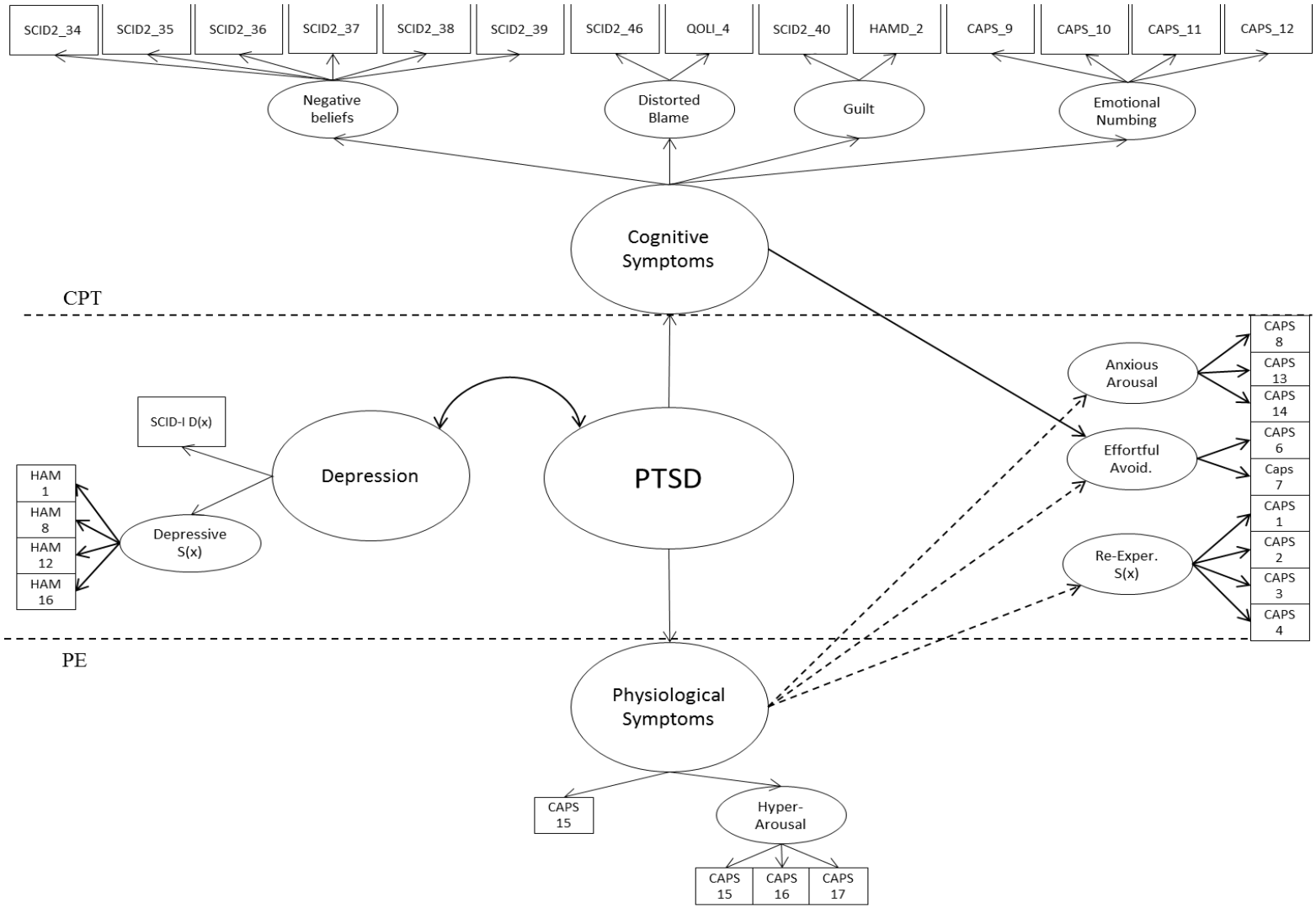


Figure 2. Hypothesized specified symptom treatment-matching model

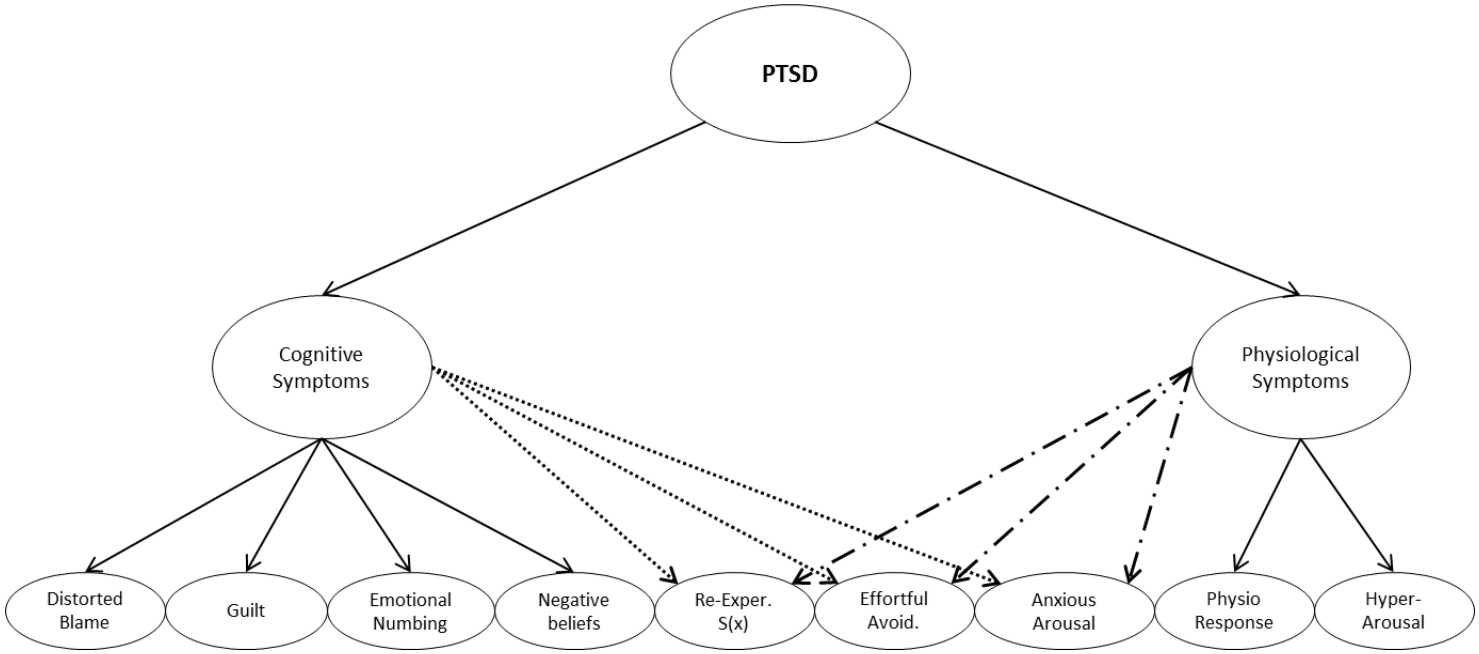


Figure 3. Shared Symptom Model

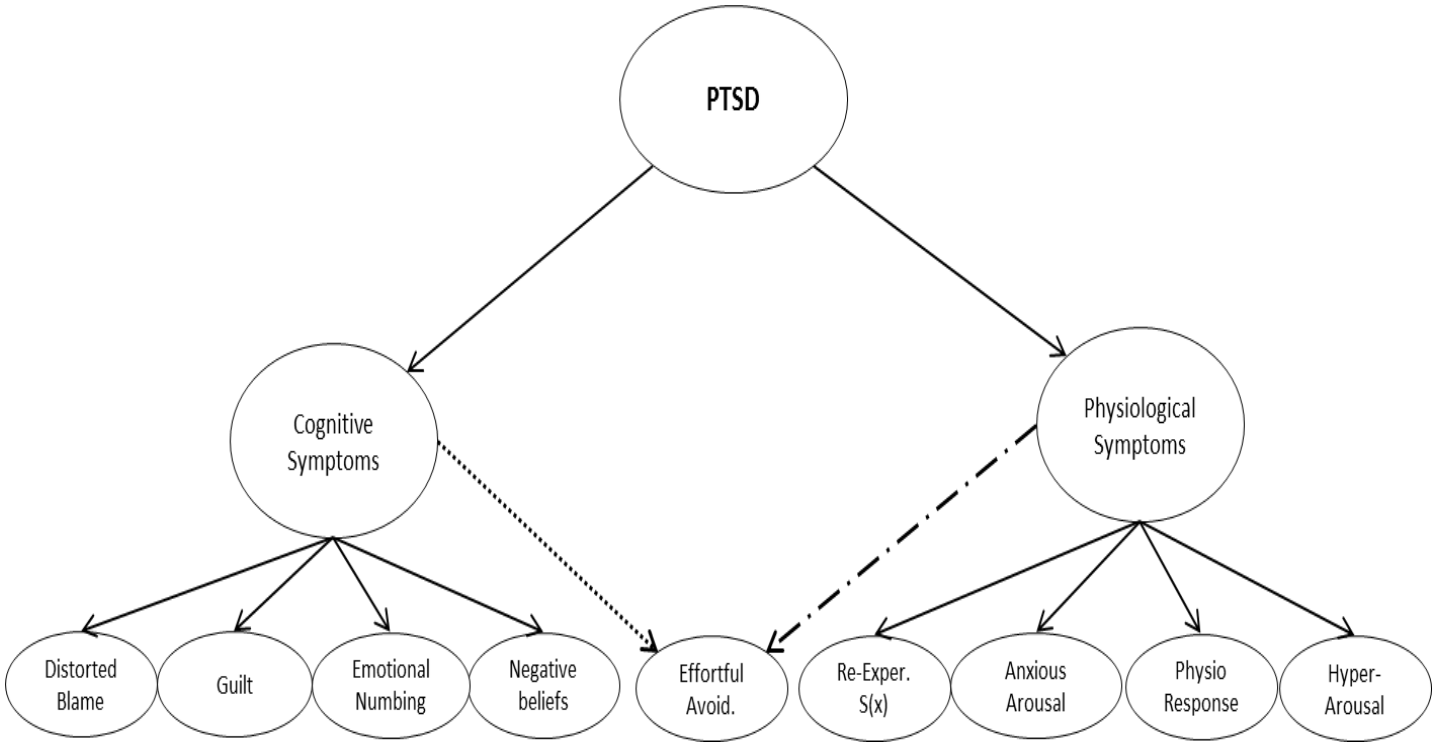
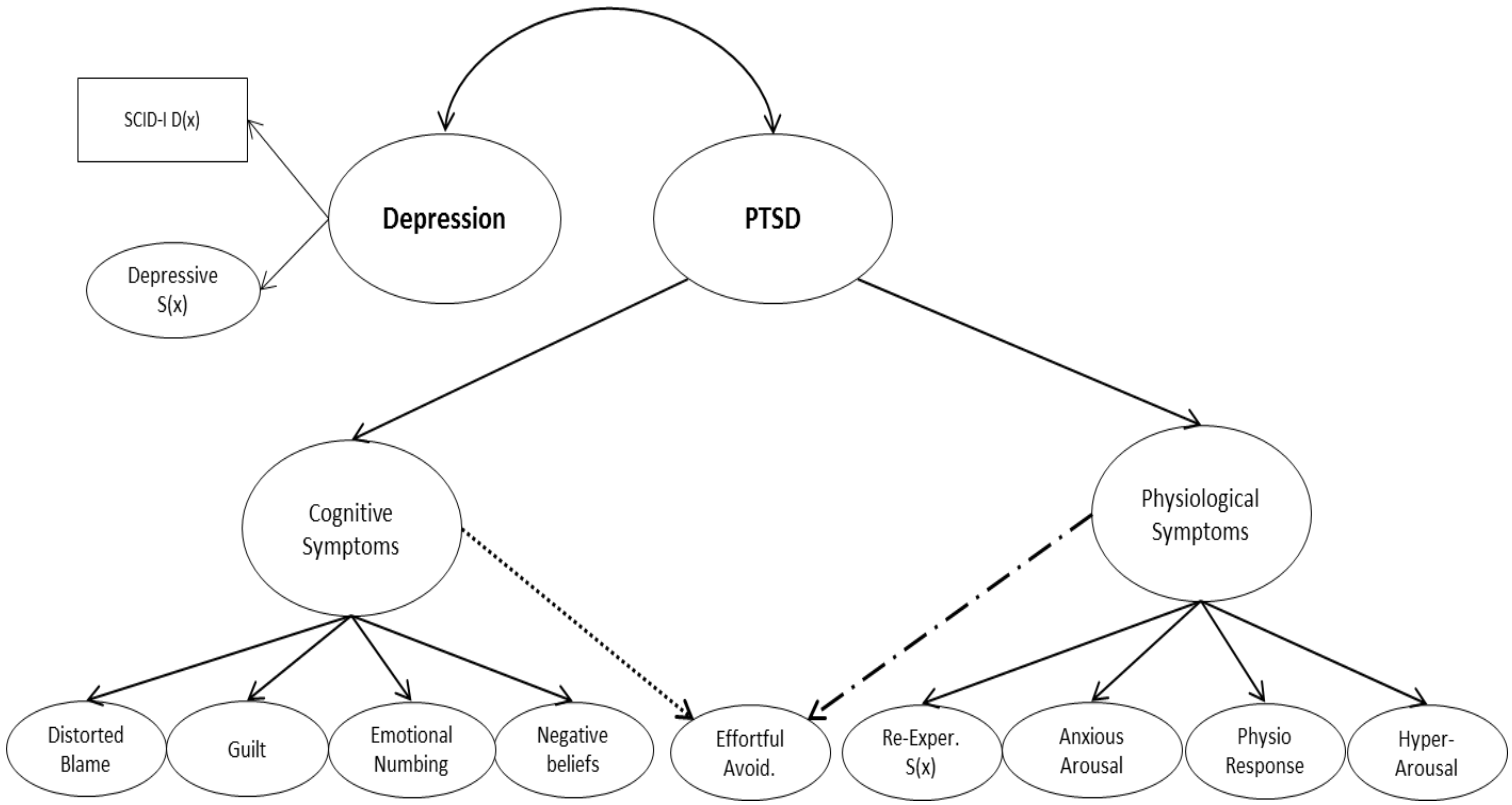


Figure 4. Specifified Symptom Model



Manifest Variables (Depression):

SCID_I	MDD diagnosis from SCID I
HAMD_1	Depressed Mood: Sadness, hopeless, helpless, worthless
HAMD_8	Retardation: Slowness of thought and Speech: impaired ability to concentrate: decreased motor activity
HAMD_12	Somatic Symptoms Gastro-intestinal (*Loss of Appetite*)
HAMD_16	Loss of Weight

Figure 5. PTSD Correlated with Depression Model with Manifest Variables

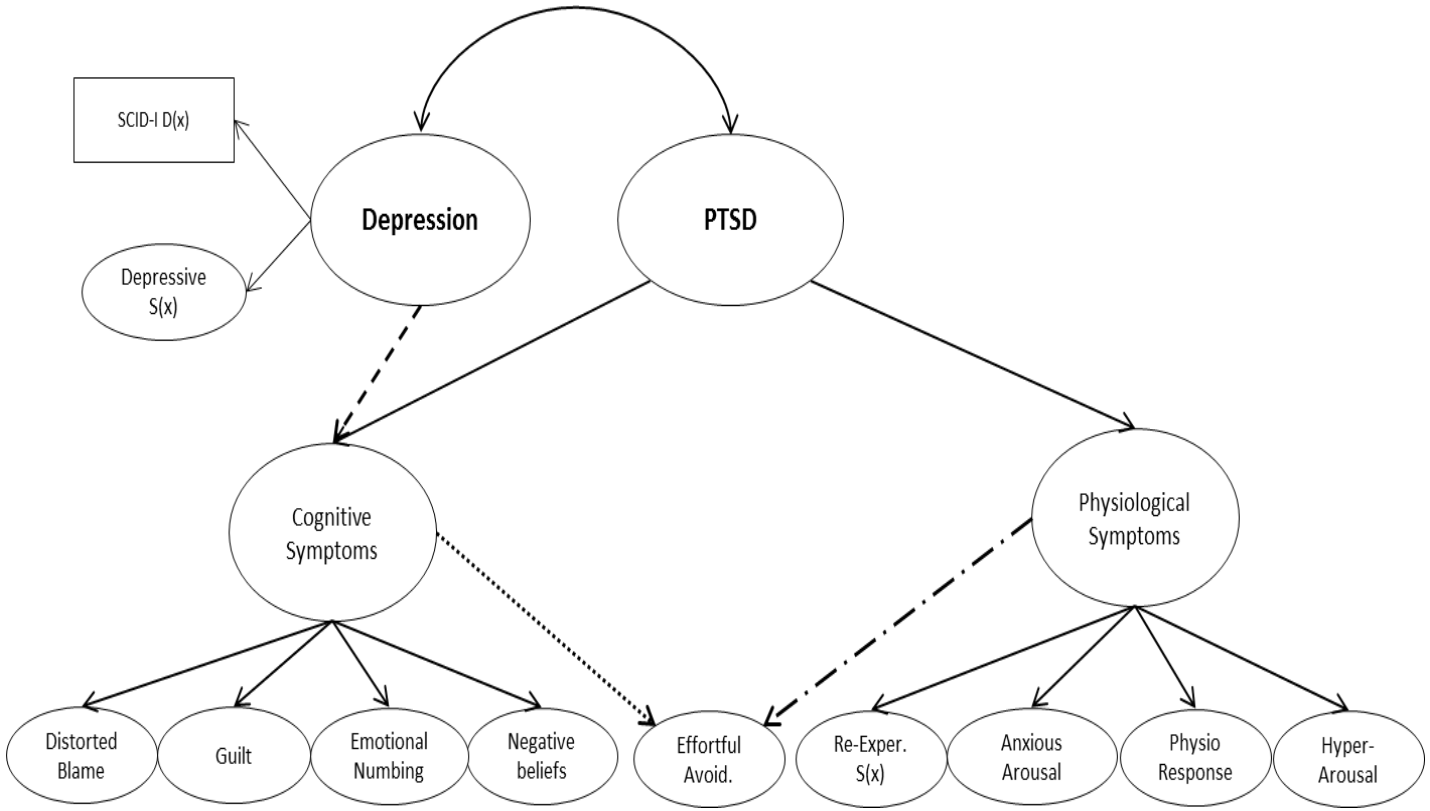


Figure 6. PTSD Predicting Depression Model

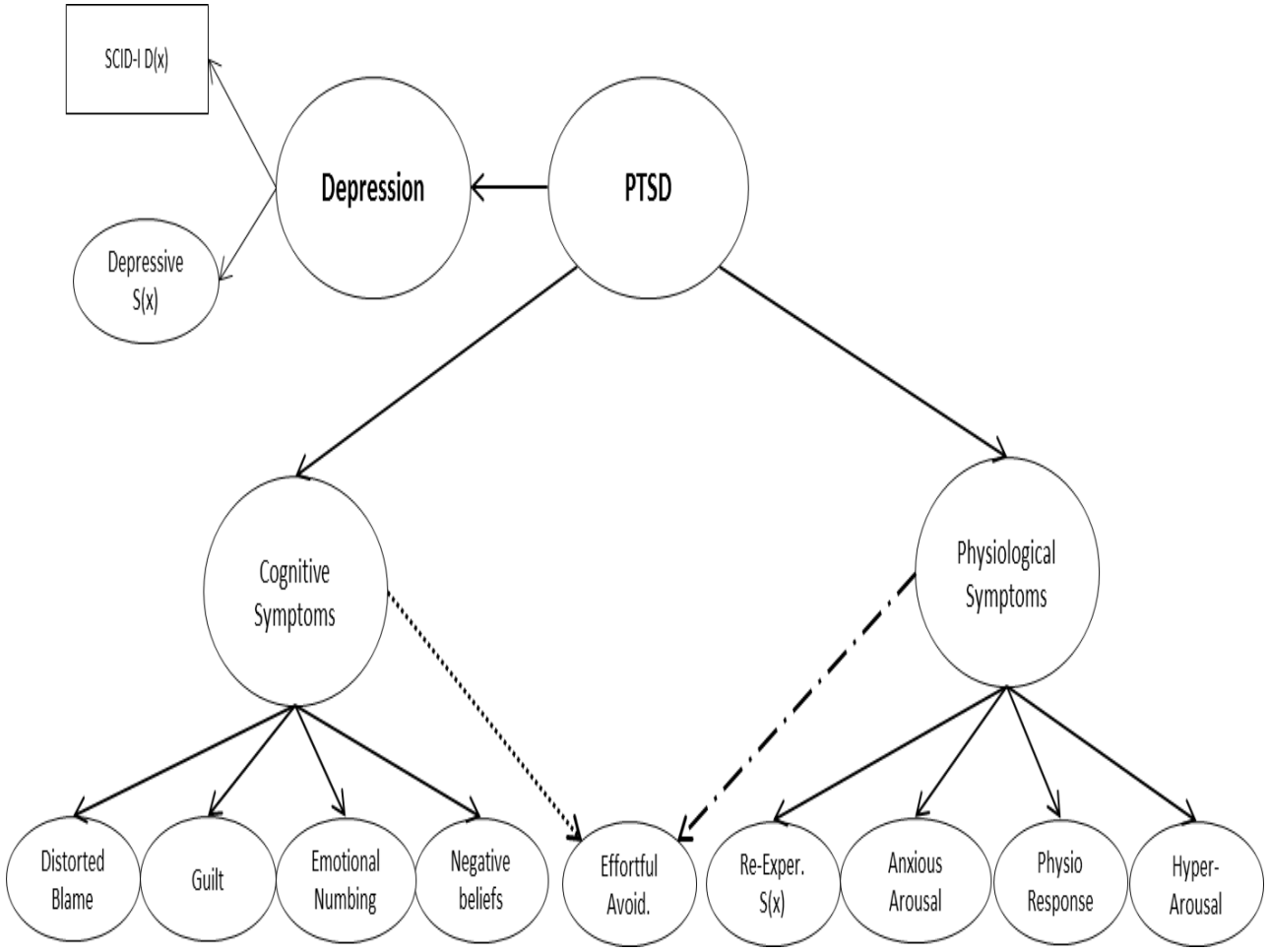


Figure 7.PTSD Correlated with Depression Predicting Cognitive Symptoms Model

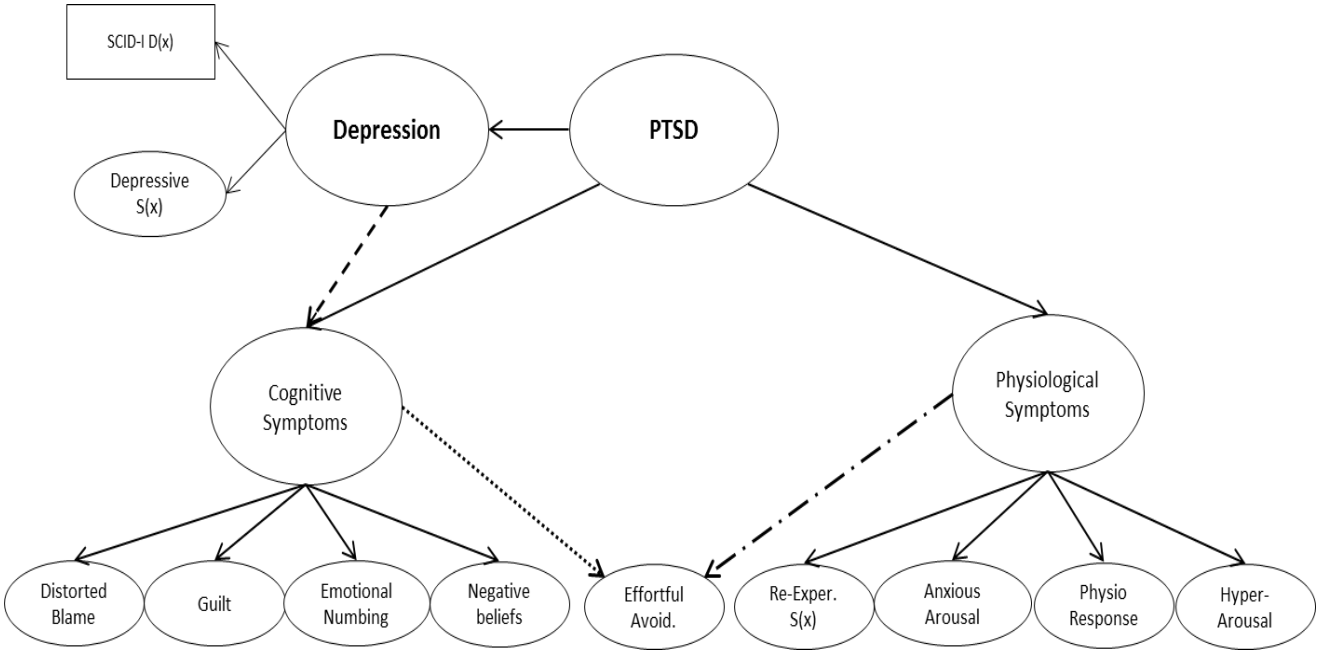


Figure 8. PTSD Predicting Depression Predicting Cognitive Symptoms Model

APPENDIX B: TABLES

Table 1. Changes to DSM Criteria for PTSD

DSM-5 Criteria for PTSD	**DSM-IV Criteria for PTSD**
B1. Intrusive thoughts B2. Nightmares B3. Re-living trauma B4. Emotional cue reactivity B5. Physiological cue reactivity	B1. Intrusive thoughts B2. Nightmares B3. Re-living trauma B4. Emotional cue reactivity B5. Physiological cue reactivity
C1. Avoidance of thoughts C2.Avoidance of reminders	C1. Avoidance of thoughts C2. Avoidance of reminders C3. Trauma related amnesia C4. Loss of interest C5. Feeling detached C6. Constricted affect C7. Hopelessness
D1. Trauma related amnesia D2. Persistent negative beliefs about the world D3. Persistent distorted blame D4. Persistent trauma-related emotions D5. Loss of interest D6. Feeling detached D7. Constricted affect	D1. Difficulty sleeping D2. Irritability/anger D3. Difficulty concentrating D4. Overly alert D5. Easily startled
E1. Irritable or aggressive behavior E2. Self-destructive or reckless behavior E3. Hypervigilance E4. Exaggerated startle response E5. Problems in concentration E6. Sleep disturbance	

Table 2. Measure items that reflect CPT Treatment Themes

Central Themes in CPT	**Items from Measures**
Self-blame/guilt	Do you often feel guilty about things you have or haven't done? *(scid2_40)* Feelings of Guilt: Do you often brood about past mistakes?*(HAMD_ 2)*
Safety	Sense of foreshortened future (*CAPS_C12*) Do you keep thinking about bad things that have happened in the past or worry about bad things that might happen in the future? *(scid2_36)*
Trust	Do you find it hard to be open, even with people you are close to? *(scid2_3)*
Control	Detachment & Estrangement (*CAPS_C10*) Do you keep thinking about bad things that have happened in the past or worry about bad things that might happen in the future? *(scid2_36)* Do you almost always expect things to turn out badly? *(scid2_39)* Is it hard for you to do simple or routine things for yourself? (*HAMD_23)*
Esteem	How satisfied are you with your self-esteem? (*QOLI_4)* How do the problems you have affect your self-esteem; how do you feel about yourself? (*HAMD_24)* Do you believe that you're not as good, as smart, or as attractive as most people *(scid2_6))* Do you believe that you are basically an inadequate person and often don't feel good about yourself? *(scid2_34)*
Intimacy	Detachment & Estrangement (*CAPS_C10*)

Table 3. Basis for Cognitive Variable Items Selection.

DSM-5: Criterion D: Negative alterations in cognitions and mood that began or worsened after the traumatic event.	Items from Measures
1. Inability to recall key features of the traumatic event (usually dissociative amnesia; not due to head injury, alcohol, or drugs).	Inability to recall important aspects of trauma *(CAPS_8)*
2. Persistent (and often distorted) negative beliefs and expectations about oneself or the world (e.g., "I am bad," "The world is completely dangerous").	Do you believe that you are basically an inadequate person and often don't feel good about yourself? *(scid2_34)* Do you often put yourself down? *(scid2_35)* Do you keep thinking about bad things that have happened in the past or worry about bad things that might happen in the future? *(scid2_36)* Do you often judge others harshly and easily find fault with them? *(scid2_37)* Do you think that most people are basically no good? *(scid2_38)* Do you almost always expect things to turn out badly? *(scid2_39)*
3. Persistent distorted blame of self or others for causing the traumatic event or for resulting consequences.	Are there many people you can't forgive because they did or said something to you a long time ago? *(scid2_46)* How satisfied are you with your self-esteem? (*QOLI_4)*
4. Persistent negative trauma-related emotions (e.g., fear, horror, anger, guilt, or shame).	Do you often feel guilty about things you have or haven't done? *(scid2_40)* Feelings of Guilt: Do you often brood about past mistakes? (*HAMD_2)*
5. Markedly diminished interest in (pre-traumatic) significant activities.	Diminished interest in activities (*CAPS_ 9*)
6. Feeling alienated from others (e.g., detachment or estrangement).	Detachment & Estrangement (*CAPS_ 10*)
7. Constricted affect: persistent inability to experience positive emotions.	Restricted range of affect (*CAPS_11*)

Table 4. Items Selected for Arousal Variables

Physiological Reactivity	**Hyperarousal**
Phys. Reactivity on exposure cues (CAPS_B5)	Hypervigilance (CAPS_D4)
	Exaggerated startle response (CAPS_D5)
	Problems in concentration (CAPS_D3)

Table 5.. Items Selected for Depression Variables

Depression Diagnosis	**Additional Depressive S(x)**
Major Depression Diagnosis, Current (SCID-I)	Depressed Mood (HAMD_1)
	Loss of Appetite (HAMD_12)
	Loss of Weight (HAMD_16)
	Psychomotor Retardation (HAMD_8)

Table 6. ML Fit Indices for Model Comparison of Treatment Matching Model

Model	χ^2	χ^2 *sig*	*df*	RMSEA	CFI	AIC	BCC	SRMR
Hypothesized	460.374	<.001	311	.057	.815	648.374	691.878	.08
Simplistic	627.029	<.001	324	.079	.625	789.029	826.516	.09
DSM-IV	267.983	<.001	117	.093	.736	373.983	388.548	.10
DSM-5	628.203	<.001	267	.095	.520	794.203	829.292	.14

Note. df = degrees of freedom; RMSEA = Root Mean Square of Approximation; CFI = Comparative Fit Index; AIC = Akaike Information Criterion; BCC= Browne-Cudeck Criterion; SRMR= Standardized Root Mean Square Residual.

Table 7. GLS Fit Indices for Model Comparison of Treatment Matching Model

Model	χ^2	χ^2 *sig*	*df*	RMSEA	GFI	AIC	BCC	SRMR
Hypothesized	354.154	<.001	311	.031	.824	488.154	519.162	.11
Simplistic	421.066	<.001	324	.045	.791	529.066	554.058	.13
DSM-IV	204.894	<.001	119	.070	.828	823.044	881.574	.22
DSM-5	322.170	<.02	267	.037	.827	438.170	462.691	.18

Note. df = degrees of freedom; RMSEA = Root Mean Square of Approximation; CFI = Comparative Fit Index; AIC = Akaike Information Criterion; BCC= Browne-Cudeck Criterion; SRMR= Standardized Root Mean Square Residual

Table 8. ML Fit Indices for Models Examining PTSD and Depression

Model	χ^2	χ^2 *sig*	*df*	RMSEA	CFI	AIC	BCC	SRMR
Correlated Dep.	584.604	<.001	419	.052	.823	800.681	859.681	.08
Corr. Dep/Predict.Cog.	609.044	<.001	420	.055	.798	823.044	881.574	.09
Predict. Dep.	585.573	<.001	420	.051	.823	799.573	858.103	.08
Predict Dep./Predict. Cog.	609.269	<.001	421	.055	.799	821.269	879.252	.09

Note. df = degrees of freedom; RMSEA = Root Mean Square of Approximation; CFI = Comparative Fit Index; AIC = Akaike Information Criterion; BCC= Browne-Cudeck Criterion; SRMR= Standardized Root Mean Square Residual.

Table 9. GLS Fit Indices for Models Examining PTSD and Depression

Model	χ^2	χ^2 *sig*	*df*	RMSEA	GFI	AIC	BCC	SRMR
Correlated Dep.	452.359	.126	419	.023	.804	606.359	648.479	.11
Corr. Dep/Predict.Cog.	454.710	.117	420	.055	.798	606.710	648.283	.15
Predict. Dep.	452.657	.131	420	.023	.803	604.657	646.229	.14
Predict Dep./Predict. Cog.	456.477	.113	421	.024	.802	606.477	647.503	.16

Note. df = degrees of freedom; RMSEA = Root Mean Square of Approximation; CFI = Comparative Fit Index; AIC = Akaike Information Criterion; BCC=Browne-Cudeck Criterion; SRMR= Standardized Root Mean Square Residual.

Table 10. Treatment Matching Model: Physiological Symptom Loadings

	β	Pvalue
Re-Experiencing Symptoms		
Intrusive Recollections (CAPS_1)	.597	<.001
Distressing Dreams (CAPS_2)	.400	<.001
Acting/Feeling Recurring (CAPS_3)	.488	<.001
Psychological Reactivity (CAPS_4)	.650	<.001
Anxious Arousal Symptoms		
Inability to Recall (CAPS_8)	.597	.035
Difficulty Sleeping(CAPS_13)	.301	.003
Irritability/Anger (CAPS_14)	.450	<.001
Difficulty Concentrating (CAPS_15)	.582	<.001
Hyper-Arousal Symptoms		
Physiological Reactivity (CAPS_5)	.654	<.001
Hypervigilance (CAPS_16)	.434	<.001
Exaggerated Startle Response(CAPS_17)	.454	-
Avoidance Physiological Symptoms		
Avoidance Thoughts (CAPS_6)	.416	..077
Avoidance Places/Activities (CAPS_7)	.190	.173

Table 11. Treatment Matching Model: Cognitive Symptom Loadings

	β	Pvalue
Negative Belief Symptoms		
Anhedonia (CAPS_9)	.613	.002
Detachment (CAPS_10)	.511	.001
Restricted Affect (CAPS_11)	.312	.010
Sense of Foreshortened Future(CAPS_12)	.555	<.001
Inadequate Person (SCID II_34)	.445	.002
Often Put Self Down (SCID II_35)	.266	.020
Preocc. with Past/Future Neg Events (SCID II_36)	.306	.011
Judge Others Harshly (SCID II_37)	.108	.260
Most people No Good (SCID II_38)	.141	.154
Expec. Neg. Outcomes (SCID II_39)	.315	.009
Distorted Blame Symptoms		
Can't Forgive Others (SCID II_46)	.133	.174
Satisfaction with Self-Esteem (QOLI4_4)	-.560	<.001
Guilt Symptoms		
Often Feeling Guilty (SCID II_40)	.314	.010
Guilt for Past Mistakes (HAMD_2)	.339	-
Avoidance: Cognitive Symptoms		
Avoidance Thoughts (CAPS_6)	.149	.308
Avoidance Places/Activities (CAPS_7)	.418	.091

APPENDIX C: IRB APPROVAL LETTER

University of Central Florida Institutional Review Board
Office of Research & Commercialization
12201 Research Parkway, Suite 501
Orlando, Florida 32826-3246
Telephone: 407-823-2901 or 407-882-2276
www.research.ucf.edu/compliance/irb.html

Approval of Human Research

From: **UCF Institutional Review Board #1**
FWA00000351, IRB00001138

To: **Deborah Casamassa Beidel**

Date: **September 18, 2014**

Dear Researcher:

On 9/18/2014 the IRB approved the following human participant research until 8/26/2015 inclusive:

Type of Review:	Submission Response for IRB Continuing Review Application Form
Project Title:	Trauma Management Therapy for OEF and OIF Combat Veterans
Investigator:	Deborah Casamassa Beidel
IRB Number:	SBE-10-07066
Funding Agency:	DOD/Army
Grant Title:	
Research ID:	1048785

The scientific merit of the research was considered during the IRB review. The Continuing Review Application must be submitted 30days prior to the expiration date for studies that were previously expedited, and 60 days prior to the expiration date for research that was previously reviewed at a convened meeting. Do not make changes to the study (i.e., protocol, methodology, consent form, personnel, site, etc.) before obtaining IRB approval. A Modification Form **cannot** be used to extend the approval period of a study. All forms may be completed and submitted online at https://iris.research.ucf.edu .

If continuing review approval is not granted before the expiration date of 8/26/2015, approval of this research expires on that date. When you have completed your research, please submit a Study Closure request in iRIS so that IRB records will be accurate.

Use of the approved, stamped consent document(s) is required. The new form supersedes all previous versions, which are now invalid for further use. Only approved investigators (or other approved key study personnel) may solicit consent for research participation. Participants or their representatives must receive a signed and dated copy of the consent form(s).

All data, including signed consent forms if applicable, must be retained and secured per protocol for a minimum of five years (six if HIPAA applies) past the completion of this research. Any links to the identification of participants should be maintained and secured per protocol. Additional requirements may be imposed by your funding agency, your department, or other entities. Access to data is limited to authorized individuals listed as key study personnel.

In the conduct of this research, you are responsible to follow the requirements of the Investigator Manual.

On behalf of Sophia Dziegielewski, Ph.D., L.C.S.W., UCF IRB Chair, this letter is signed by:

Signature applied by Patria Davis on 09/18/2014 04:54:52 PM EDT

IRB Coordinator

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