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# Combat Related Guilt and the Mechanisms of Intensive Exposure Therapy

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# COMBAT RELATED GUILT AND THE MECHANISMS OF INTENSIVE EXPOSURE THERAPY

by

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# ABSTRACT

Posttraumatic stress disorder (PTSD) is highly prevalent in military populations, and is associated with significant medical costs. Due to these high costs and corresponding health infrastructure required to meet the needs of military service personnel, it is essential that the most effective and efficient treatments be implemented. Exposure therapy (EXP) is one of the most widely used and empirically supported treatments for PTSD; however, some researchers have questioned its efficacy with specific populations and in targeting specific symptoms. One such symptom, guilt, has garnered increased attention in the PTSD treatment literature, as it is associated with worse symptomatology and outcomes. The current study examined cognitive changes in guilt in response to Intensive (3-week) and standard (17-week) Trauma Management Therapy (TMT) and the impact of these cognitions on the mechanisms underlying TMT treatment. Sample size for these analyses varied by the measure being considered. 102 individuals completed the PCL-M, 42 individuals completed the TRGI, and 39 individuals completed the CAPS supplemental guilt items. Results suggest that a secondary benefit in guilt symptoms is achieved by targeting anxious-related distress with exposure therapy. Furthermore, in this sample guilt did not seem to inhibit the mechanisms or effectiveness of exposure therapy.

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| | |
|---|---|
| CAPS | Clinician Administered PTSD Scale |
| CPT | Cognitive Processing Therapy |
| DSM | Diagnostic and Statistical Manual of Mental Disorders |
| EPT | Emotional Processing Theory |
| EXP | Exposure Therapy |
| IED | Improvised Explosive Device |
| OEF | Operation Enduring Freedom |
| OIF | Operation Iraqi Freedom |
| PCL-M | PTSD Checklist- Military Version |
| PE | Prolonged Exposure |
| PTSD | Post-Traumatic Stress Disorder |
| SCID-I | Structured Clinical Interview for the DSM-IV, I |
| TBI | Traumatic Brain Injury |
| TMT | Trauma Management Therapy |
| TRGI | Trauma-related Guilt Inventory |
| TRGI-GG | Trauma-related Guilt Inventory-Global Guilt |

# CHAPTER ONE: INTRODUCTION

The treatment of posttraumatic stress disorder (PTSD) is a significant public health concern for veteran affairs organizations and national healthcare policy. Approximately 15 percent of all returning veterans will be diagnosed with PTSD at some point in their lives (Richardson, Frueh, & Acierno, 2010), and the USDVA (2014) reported that PTSD is the most common diagnosis among the more than 550,000 Operation Enduring Freedom/Operating Iraqi Freedom (OEF/OIF) veterans that received a psychiatric diagnosis at VA facilities. The cost of providing mental health services for these veterans is substantial, exceeding six billion dollars two years post-deployment when PTSD and comorbid depression are considered together (Tanielian & Jaycox, 2008, as cited in Gates et al., 2012). Furthermore, the median public health care cost for PTSD is approximately $12,000 per veteran annually (Watkins et al., 2011). This substantial cost is largely attributable to the significant health care utilization and lost work productivity associated with PTSD (Asnaani, Reddy, & Shea, 2014; Frayne et al., 2011; Tuerk et al., 2012).

When considering the high prevalence and significant cost associated with PTSD, the identification of efficacious, effective, and efficient interventions is crucial to alleviate the substantial strain on health care services. Furthermore, the effective utilization of health care providers and organization resources can help alleviate some of the burden from already overwhelmed facilities (Maguen et al., 2012; Roshenheck & Fontana, 2007). One way to achieve these goals is to ensure that healthcare providers are implementing the most empirically supported interventions and targeting the symptoms underlying the patient's distress. The

process of treatment and resource allocation can be greatly informed by a better understanding of mechanisms underlying improvement in therapy.

One of the most studied and empirically supported interventions for PTSD is exposure therapy (EXP). Recent studies have shown that EXP can be extremely efficient, as it remains efficacious in intensive short-duration treatment formats (Beidel et al., 2014) and with shorter individual sessions (Nacasch et al., 2015). EXP is based on Emotional Processing Theory (EPT), the foundations of which are rooted in early animal research theorizing that the acquisition of fear occurs through the repeated or intensive pairing of an unconditioned aversive stimulus and a neutral or conditioned stimulus (CS) (Mowrer, 1939). Inspired by this early animal research and information processing theories proposed by Lang (1979) and Rachman (1980), Foa and Kozak (1986) postulated that the extinction of pathological human fear can be achieved through repeated exposure to the fear/anxiety-provoking stimuli (CS) in the absence of aversive consequences. This repeated exposure allows for new learning to occur that overrides previous paired CS/US associations. Decreases in anxious responses that occur during an EXP session are termed within-session habituation, whereas anxiety reduction that occurs across EXP sessions is referred to as between-session habituation.

In addition to providing incompatible information to form new non-fear oriented associations in memory, EPT posits that the fear memory structure needs to be sufficiently activated (i.e. fear activation) for EXP to be effective. The importance of fear activation and the formation of new inhibitory associations in predicting treatment outcome has been supported by several studies that include physiological, neurological, and subjective measures of anxiety

(Beidel, Turner, & Dancu, 1985; Borkovec & Sides, 1979; Foa, Riggs, Massie, & Yarczower, 1995; Minnen & Hagenaars, 2002, Pitman et al., 1996).

Randomized-controlled trials have repeatedly demonstrated the efficacy of EXP for PTSD (Benish, Imel, & Wampold, 2008, Powers et al., 2010, Rothbaum et al., 2014) and previous research has supported the tenets of EPT by linking improvement in PTSD symptoms to habituation (Foa & Chambless, 1978; Jaycox, Foa, & Morral, 1998; Minnen & Hagenaars, 2002) and fear activation (Minnen & Hagenaars, 2002; Pitman, 1996). Habituation is most commonly defined as a significant reduction in Subjective Units of Distress (SUDS) ratings provided by the patient over the course of imaginal exposure therapy sessions. Researchers have demonstrated that greater reduction in peak SUDS ratings over the course of treatment is an indicator of treatment response and is associated with greater symptom improvement (Bluett et al., 2014; Gallagher & Resick, 2012; Rauch, Foa, Furr, & Filip 2004; Minnen & Foa, 2006).

Although EXP is a well-supported intervention for PTSD, EXP is not associated with universal improvement, as a portion of individuals see minimal or no symptom reduction as a result of EXP (Bradley et al., 2005; Roberts, Kitchiner, Kenardy, & Bisson, 2009; Rothbaum et al., 2014). Further, the percentage of treatment non-responders appears to be larger in military samples (Steenkamp, Litz, Hoge, & Marmar, 2015), a problem that is compounded by the significant dropout rates (17-52 percent) observed in this population (Gros et al., 2011; McLay et al., 2011; Reger et al., 2011; Strachan et al., 2012; Tuerk et al., 2010; Tuerk et al., 2011). Overall, meta-analytic studies have shown that EXP is associated with moderate effect sizes and some studies suggest that it may not adequately address all symptoms of PTSD (Owens, Chard, & Cox, 2008; Resick et al., 2002) or adequately target

all maladaptive psychological consequences of combat exposure (Litz et al., 2009). These results have led some to suggest that the mechanisms underlying exposure therapy are insufficient to address internalizing symptoms related to PTSD and propose alternative interventions such as Cognitive Processing Therapy (CPT) (Resick & Schnicke, 1993) and Imagery Rescripting (IR) (Smucker & Dancu, 1999).

Given that the theoretical underpinnings of EPT are largely based in animal research, it is generally assumed that improvement in EXP involves the exclusive recruitment of basic neural processes. This assumption is supported by some neurological research that links improvement in EXP to reduced amygdala and related medial prefrontal cortex activation (Ledoux, 1996; Phelps, Delgado, Nearing, & LeDoux, 2004; Repa et al., 2001). However, recent research suggests that extinction learning may involve more complex higher order cognitive processes that are essential to recovery (Hofman, 2008; Lovibond, 2004). In a review of the cognitive processes during fear acquisition and extinction learning, Hofman (2008) points to several studies that support the mediating role of higher order cognitive processes in extinction learning and in the pathogenesis of anxiety disorders such as social anxiety disorder and PTSD. Correspondingly, recent studies have demonstrated that changes in maladaptive trauma-related cognitions precede changes in other PTSD symptoms during EXP (Oktedalen, Hoffart, & Landkaas, 2015; Zalta et al., 2014).

In recent years, trauma-related cognitions associated with PTSD received increased empirical attention and numerous studies have identified a trauma-specific profile of maladaptive cognitions associated with greater functional impairment, symptom severity, and illness duration (Friedman et al., 2013; Meiser-Stedman et al., 2009; Moser et al., 2007). In addition, Litz and

colleagues (2009) have introduced the concept of moral injury (i.e., a violation of personal moral standards) specifically related to combat trauma and associated with negative outcomes and internalizing symptoms (e.g., guilt & shame). Because of the increased attention and support for the role of cognitive processes in PTSD, the latest revision to the Diagnostic and Statistical Manual of Mental Disorders (DSM) diagnostic criteria for PTSD included, among other changes, a subset of symptoms termed "negative alterations in cognitions and mood" (American Psychiatric Association, 2013). Three of these symptoms are entirely new to the DSM and reflect the presence of perceived internal threat such as guilt, shame, or general inferiority.

Prior to the most recent DSM revision, PTSD had largely been considered an anxiety disorder that represented continued fear of external threat and perceived danger primarily maintained through the avoidance of anxiety-provoking stimuli. However, there is an emerging consensus in the literature that traumatic experiences can also elicit a diverse set of internalizing emotions such as guilt, shame, and anger (Litz et al., 2009; Power & Fyvie, 2013). Newer theories have broadened the conceptualization of PTSD to account for these emotions; positing that an internal threat to an individual's sense of self is a primary mechanism for maintaining PTSD (Harmen & Lee, 2010). The association between guilt and PTSD is particularly strong among veteran populations, which may be attributable to the unique types of traumatic events related to combat that can elicit both anxious and affective emotional responses (Pugh, Taylor, & Berry, 2015).

To this end, there is a burgeoning body of literature that acknowledges the diverse psychological harm that can occur as a result of combat exposure. This psychological harm can stem from traumatic events that involve intense fear and helplessness, as well as morally

injurious events involving perceived moral transgressions (Bryan et al., 2013; Steenkamp, Nash, Lebowitz, & Litz, 2013; Stein et al., 2012). Although guilt and shame have long been acknowledged as negative psychological consequences stemming from wartime violations of personal moral standards (Hayley, 1974; Rivers, 1918), specific treatment strategies to address these symptoms have been notably absent. Additionally, the tendency for existing treatments to emphasize the reduction of external threat may partially explain the higher rates of treatment non-responders in veteran samples (Steenkamp, Litz, Hoge, & Marmar, 2015).

Specifically, the emotional experience of guilt has been the subject of considerable debate regarding its relationship to maladaptive outcomes (Tilghman-Osbrone et al., 2010) and response to existing PTSD treatments (Rauch, Smith, & Duax, 2013; Smith, Duax & Rauch 2013; Steenkamp, Nash, Lebowitz, & Litz, 2014). Interestingly, guilt is not exclusively linked to negative outcomes. Some theorists highlight the adaptive nature of guilt, as it promotes reconciliation and maintains social relationships (Tanney et al., 2007). Tilghman-Osborne and colleagues (2010) emphasize the importance of clearly defining guilt and suggest that it is essential to differentiate guilt, which focuses on real or perceived misdeeds related to a specific behavior or set of behaviors, from shame. Shame is a more complex emotion as it is associated with persistent negative self-appraisal and perceived inferiority or judgment from others potentially leading to self-isolation and social withdrawal. Furthermore, guilt can be maladaptive if it co-occurs with shame or reinforces negative self-appraisals (Tanney et al., 2007).

In veteran populations, definitions of guilt consistent with the definition provided by Tilghman-Osborne and colleagues (2010) is associated with negative outcomes including depression and a higher risk of suicidal behavior (Bryan et al., 2014; Hendin, 1991; Hening et

al., 1997). Researchers have suggested that guilt may hinder natural emotional processing of traumatic events and inhibit the integration of perceived misdeeds into prior belief systems (Ehlers & Steil; Pitman et al., 1991 as cited in Pugh, Taylor, & Berry, 2015), resulting in avoidance and the reinforcement of trauma-related psychopathology (Held et al., 2011; Street et al., 2005). Specifically, guilt cognitions associated with a preventability, personal responsibility, and lack of justification were most strongly associated with intrusive PTSD symptoms, whereas preventability and personal responsibility were also related to avoidance (Pugh, Taylor, & Berry, 2015). In a review of the literature concerning guilt and PTSD, Pugh and colleagues (2015) cite evidence for the mediating role of avoidance between guilt and PTSD, suggesting that treatments such as EXP directly targeting avoidance may see a secondary benefit of reducing guilt cognitions.

Guilt has also been the subject of research regarding the effectiveness of EXP. A specific type of EXP, Prolonged Exposure (PE), is the most widely used form of EXP to treat PTSD. Studies have demonstrated that PE can effectively produce significant reductions in measures of trauma-related guilt (Trauma Related Guilt Inventory; TRGI; Kubany, 1996) and depression (Rauch, Smith, & Duax, 2013); however, the specific mechanisms by which these changes occur are unclear. Rauch and colleagues (2013) suggest that the standard PE protocol is meant to focus on any PTSD symptoms that are distressing for the patient and that habituation to a variety of emotions (e.g., sadness, guilt, disgust, anxiety) allows the patient to place the trauma in a broader emotional context and re-examine the meaning of the event. Further, these researchers state that mechanisms other than habituation that occur within other PE treatment elements may contribute to symptom improvement. Alternatively, some theorists have suggested that significant guilt

cognitions may interfere with habituation and may be a contraindication for EXP (Tarrier et al., 1999). Other researchers have suggested that since EXP fosters habituation through repeated exposure to present and future oriented fear, the retrospective nature of guilt may leave it largely immune to the effects of habituation and EXP (Dalgleish, 2004).

Direct empirical evaluations of guilt outcomes as a result of PE are rare and have reported mixed results. Although some studies report significant reductions in guilt as a result of PE (Nishith et al., 2005; Oktedalen, Hoffartm, &Langkass, 2015; Resick et al., 2002; Zalta et al., 2014), others report limited improvement in guilt and shame symptoms (Arntz et al., 2007; Grunert, Smucker, & Weis, 2003; & Grunert et al., 2007). Furthermore, studies attempting to augment PE with cognitive restructuring have either found no improvement over and above traditional PE (Foa et al., 2005) or significantly worse outcomes (Moser, Cahill & Foa, 2010). These findings suggest the potential for trauma-related cognitions to hinder the effects of PE or that traditional treatment formats do not provide sufficient time for independent treatment elements to be implemented sufficiently.

Studies examining the temporal order of PTSD symptom change during PE have shown that changes in maladaptive cognitions (Zalta et al., 2014) and guilt (Oktedalen, Hoffartm, & Langkass, 2015) precede changes in other PTSD symptoms. However, these studies were not conducted with military or combat veterans that experience unique (Hoge et al., 2004; Litz et al., 2009) and often multiple traumas (Kline et al., 2010). Additionally, in one of these studies, Zalta and colleagues (2014) assessed trauma-related cognitions using the Post-Traumatic Cognitions Inventory (PTCI; Foa et al., 2009). The PTCI assesses a variety of self-evaluative (e.g., the event

happened because of the way I acted) as well as present fear-oriented (e.g., the world is a dangerous place) cognitions, the latter of which may be more amenable to PE.

Certain guilt-related cognitions may respond differently to specific treatment modalities. Steenkamp and colleagues (2015) point out that the research supporting PE's effectiveness for guilt examines change in guilt cognitions in assault victims and not perpetrators of violence. Additionally, Resick and colleagues (2002) found that CPT demonstrated greater reductions than PE in cognitions related to hindsight bias and lack of justification. This finding is in line with existing research demonstrating that lack of justification is less related to avoidance than other guilt related cognitions (Pugh, 2015) and also consistent with the primary theory of trauma-related guilt (Kubany, & Watson, 2003). Kubany and Watson (2003) suggest that guilt cognitions that are associated with avoidance may be more amenable to EXP based techniques, therefore, guilt cognitions related to a lack of justification or hindsight bias may be better addressed by an alternative intervention. Collectively, these studies suggest that PE may not be equally effective for all trauma or guilt-related cognitions or may not sufficiently address these cognitions in all cases.

Although the development of PE was based on EPT and habituation, PE contains several treatment elements in addition to exposure, including psychoeducation and emotional processing. Psychoeducation is not unique to PE and occurs prior to the initiation of exposure techniques, whereas emotional processing occurs after each treatment session. Proponents of PE have suggested that although guilt stemming from morally injurious events can be acknowledged in each element of PE, it is most notably addressed during the processing element of treatment (Smith, Duax, & Rauch, 2013). Unfortunately, there is a clear absence of dismantling studies

involving PE, which limits the identification of the treatment elements responsible for reductions in overall symptomatology and specific symptoms such as guilt.

Trauma Management Therapy (TMT) is a multicomponent treatment that includes psychoeducation, imaginal exposure, in-vivo exposure, and group therapy. Trauma management group therapy focuses on addressing secondary features of combat-related PTSD that are addressed in three modules; social reintegration, anger management, and behavioral activation. When conducted in an intensive three-week format, individual and group components are conducted daily in two separate sessions. The anger module specifically addresses guilt during the eighth session by discussing distorted self-blame and making reparations. TMT is a unique treatment that achieves primary symptom reduction through EXP and targets secondary PTSD symptoms with group therapy. TMT is distinct from PE as exposure sessions primarily target fear and helplessness as other emotions are addressed in several additional group treatment modules. TMT also does not emphasize the role of emotional processing after the exposure session and post-session discussions are instead used to reinforce patient effort and positive treatment expectancy. The absence of guilt-based emotional processing after the EXP session provides the opportunity to assess the effects of guilt-related trauma cognitions on habituation and overall treatment outcome. The present study will examine habituation and guilt cognitions as time-varying predictors of treatment outcome to assess the effect of guilt cognitions on the underlying mechanisms of EXP. Based on the PTSD treatment literature, the following hypotheses were tested:

1) Guilt cognitions related to acts of "commission or omission" as measured by the CAPS will significantly improve from pre to post treatment.
2) Participants with fewer baseline self-reported guilt cognitions will achieve overall habituation in fewer sessions than individuals with greater baseline guilt cognitions.
3) The significant reduction of avoidance symptoms will significantly contribute to the prediction of guilt cognitions over the course of treatment.
4) Participants with greater self-reported guilt cognitions as measured by the TRGI will demonstrate lower fear activation over the course of treatment than participants with fewer self-reported guilt cognitions.
5) Participants with fewer self-reported guilt cognitions will demonstrate significantly greater reductions in Peak SUDs ratings over the course of treatment.
6) A model containing Peak SUDs ratings as an index of habituation, guilt cognitions as measured by the TRGI, and a time marker for the group guilt intervention will demonstrate the best fit to the data predicting PTSD symptoms over the course of treatment.

# CHAPTER TWO: METHOD

## Participants

Data was collected as part of two treatment studies funded by the Department of Defense. The Intensive TMT study evaluated the efficacy of a three-week exposure based treatment protocol for PTSD in combat veterans and active duty personnel of OEF, OIF, and OND. The standard 17-week study recruited a similar population and compared the efficacy of exposure therapy with TMT group therapy to exposure therapy with traditional psychoeducation group therapy. In the three-week protocol, patients participated in daily EXP sessions and group therapy. Under the supervision of licensed clinical psychologists, graduate students conducted all assessments and provided the treatment. Participants were compensated 50 dollars for completing pre-treatment and post-treatment assessments. The sample consisted of treatment-mandated and treatment-seeking veterans as well as active-duty military personnel. Exclusion criteria were intentionally minimized in order to obtain a representative veteran sample. Admission into the treatment protocol required a current clinician-determined diagnosis of combat-related PTSD confirmed by a supervising clinician. Due to the necessity for sustained physiological arousal in the early phases of treatment, patients were excluded if they had a history of significant cardiac symptoms. Patients were also excluded if they presented with an acute substance abuse disorder and were unable to demonstrate two weeks of abstinence, had a medication history that could not be stabilized for two weeks, or if the participant met criteria for antisocial personality disorder. Although screened for Traumatic Brain Injury (TBI), a TBI diagnosis did not exclude participants from participation in this treatment protocol as OEF, OIF,

OND veterans experience these injuries at high rates (Shively & Perl, 2012; Vasterling, Verfaellie, & Sullivan, 2009).

The final sample included 65 veterans and 37 active duty military personnel directly involved in OEF, OIF or OND between the ages of 23 and 63 years. Among the sample, 57 percent reported experiencing a blast injury and 49 percent reported a history of a TBI diagnosis. A subset of these veterans completed two measures related to guilt (See Table 1 for additional demographics).

## Intensive Trauma Management Therapy Protocol

Trauma Management Therapy (TMT) (Frueh, Turner, Beidel, Mirabella, & Jones, 1996) is a behavioral-based treatment specifically designed to address the needs of combat veterans diagnosed with PTSD. The original TMT protocol includes imaginal, in-vivo, and group therapy sessions conducted over the course of 17-weeks. The group component of treatment includes six Social Reintegration, four Anger Management, and four Behavioral Activation sessions. These interventions target secondary features commonly associated with PTSD, but are often not directly addressed in traditional EXP protocols (Frueh et al., 1995; Stapleton, Taylor, & Asmundson, 2006).

The intensive TMT protocol (Beidel, Frueh, Neer, Bowers, & Rizzo, 2014) was conducted five days a week, over the course of three weeks. Each day, patients participated in imaginal exposure and group therapy sessions (15 individual/14 group sessions). Imaginal exposure sessions were assisted by virtual reality (VR) equipment with visual, olfactory, auditory, and kinesthetic cues. All or some of these cues were utilized at the discretion of the clinician and were specific to the patient's traumatic event. The goal of this equipment is to

increase the patient's contact with the fear memory, which may promote greater fear activation and treatment generalization.

During imaginal exposure sessions, Subjective Units of Distress (SUDS) ratings were obtained approximately every five minutes, until the patient demonstrated a 50 percent reduction in SUDS ratings from that sessions Peak SUDS rating, or demonstrated a return to that sessions baseline SUDS rating (within-session habituation). If the patient demonstrated habituation to the imaginal scene (a 50 percent reduction in Peak SUDS ratings across sessions) before the end of the three-week protocol, the remainder of the sessions consisted of in-vivo exposure to patient-specific anxiety-provoking stimuli (e.g., large crowds). For the current analysis, only data from the imaginal exposure sessions were examined.

Group therapy modules were co-led by two graduate clinicians and patients were provided with daily session-related assignments to be completed outside of group. Group therapy modules were presented in a varied order to provide the patient with sufficient time to complete assignments and promote the integration of group content. The anger management module included a brief one-session intervention (session 8) targeting guilt symptoms designed to reduce distorted self-blame for a traumatic event and promote a healthy and more accurate diffusion of this responsibility.

## Measures

### Clinician-Administered PTSD Scale (CAPS)

The CAPS (Blake et al., 1990; Weathers & Litz, 1994) is a 25-item semistructured interview that assesses the DSM-IV criteria for PTSD. The CAPS includes dual (i.e., frequency and severity)

ratings of the 17 PTSD symptoms as well as questions assessing social and occupational impairment associated with PTSD. The CAPS interview is a clinician-assessed measure of PTSD symptoms, and provides a reliable evaluation of the patient's reported symptoms and functional impairment. A total severity score (range 0-136) was calculated by summing the patient's endorsements. Subscale scores were calculated based on the three factors (Re-experiencing, Avoidance, Hyperarousal) outlined in the DSM-IV. The CAPS also included two guilt-related questions that fall under "associated features," and assess the frequency and severity of "acts of commission or omission" and "survivor guilt." The CAPS interview is administered at pre-treatment and one-week post treatment.

### PTSD Checklist-Military Version (PCL-M)

The PCL-M (Weathers, Litz, Huska, & Keane, 1994) is a self-report measure assessing the 17 PTSD symptoms outlined in the DSM-IV with an emphasis on past military experiences. This measure instructed patients to rate how much they "have been bothered" by their symptoms on a Likert scale from 1 (not at all) to 5 (extremely) in the last week. A total severity score (range 0-85) were calculated by summing the patient's endorsements. Subscale scores were calculated based on the three factors (Re-experiencing, Avoidance, Hyperarousal) outlined in the DSM-IV. The PCL-M was administered at the beginning of each week over the course of treatment and at follow-up (one week, three months, and six months). For the purposes of this study, data was examined from the following collection points: pre-treatment, week one, week two, week three, and one-week post-treatment.

## Trauma-Related Guilt Inventory (TRGI)

The TRGI (Kubany, 1996) is a 32-item measure assessing three primary domains of guilt related cognitions (Global Guilt, Distress, and Guilt Cognitions). The TRGI also provides three additional scales (Hindsight Bias, Wrongdoing, and The Lack of Justification) comprised of smaller groupings of items. The TRGI is the most widely used measure of trauma-related guilt and is commonly used to assess change in cognitions over the course of PTSD treatment (Nishith, Nixon, Resick, 2005; Oktedalen, Hoffartm, & Langkass, 2015). The TRGI was administered at the beginning of each week over the course of treatment and at follow-up (one week, three months, and six months). For the purposes of this study, data was examined from the following collection points: pre-treatment, week one, week two, week three, and one-week post-treatment.

## Subjective Units of Distress (SUDS)

SUDS ratings are commonly used during exposure therapy to assess acute anxiety reactions to target stimuli. According to the TMT protocol, SUDS ratings (0, None to 8, Extreme) were obtained approximately every five minutes until the patient demonstrated within-session habituation. For the purposes of this protocol, within-session habituation was defined as a fifty percent reduction from the patient's peak SUDS rating or a return to the patient's baseline SUDS rating. In some circumstances, sessions were ended if the time exceeded 90 minutes.

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

The SCID-I (First et al, 1996) is a semi-structured diagnostic interview that assesses major psychiatric DSM-IV diagnoses. The SCID was administered to assess for comorbidities such as depression and to confirm a diagnosis of PTSD.

### Daily Behavior Rating Form (DBR)-Anxiety

The DBR-Anxiety is a measure designed for this study. From one week prior to treatment through post treatment, participants provided daily ratings of their subjective level of general anxiety on a Likert scale from 0 (None) to 10 (Severe).

### Overall Habituation

Overall habituation was defined as the number of sessions required for the patient to achieve a fifty percent reduction in overall peak SUDS rating.

### Fear Activation

Fear activation was extracted from the patient's session SUDS ratings and defined as the number of minutes until the patient reaches his PEAK SUDS rating for each session. Traditionally, fear activation is defined as the patient's baseline SUDS rating, however; the traditional index of fear activation (baseline SUDS rating) may not fully account for level of interference due to non-fear related cognitions. Traditional methods (Minnen & Hagenaars, 2002) for measuring fear activation were examined that included the participants baseline SUDS rating and a variable that is calculated by subtracting the baseline SUDS rating from the peak suds rating.

## Statistical Strategy

For hypothesis one and two, traditional mean-based statistics were used to determine the effects of imaginal exposure therapy on guilt-related cognitions and rate of habituation without accounting for the effects of time. Parametric and non-parametric tests were used to assess if guilt cognitions improve as a result of EXP and if individuals with fewer maladaptive guilt related cognitions achieve overall habituation in fewer sessions.

1) *Guilt cognitions related to acts of "commission or omission" as measured by the CAPS will significantly improve (decrease) from pre to post treatment.*
    - A Wilcoxon signed-rank test was conducted to assess significant guilt symptom reduction as a result of EXP.

2) *Participants with fewer baseline self-reported guilt cognitions, as measured by the TRGI, will achieve overall habituation in fewer sessions than individuals with greater baseline guilt cognitions.*
    - A dichotomous variable (0,1) was created based on the median of the TRGI global guilt subscale and an independent sample t-test was conducted to assess if individuals with fewer guilt symptoms prior to treatment achieve habituation in fewer sessions than individuals with greater guilt symptoms prior to treatment.

Hypotheses three through six will determine if levels of guilt in this sample interfere with the processes underlying exposure therapy and if changes in cognitive avoidance influence guilt cognitions over the course of treatment. Mixed-effects regression allowed for the examination of

changes in guilt and habituation over the course of treatment as well as determine if the session eight guilt intervention significantly contributed to predicting symptom improvement. Linear mixed-effects regression (LMER) provides many advantages over traditional ANOVA-based methods of assessing changes over time that are particularly advantageous given the limitations of treatment data.

Previous examinations of TMT and EXP data have revealed that the majority of symptom improvement likely occurs in the first two weeks of treatment (Munyan, Neer & Beidel, 2014) and that symptom severity influences the trajectory of treatment response (Currier, Holland, & Drescher, 2014). These findings cast doubt on the ability of EXP treatment data to meet the assumptions of ANOVA. Furthermore, repeated measures ANOVAs require complete data sets, often leading to the creation of artificial aggregate variables, the estimation of data points, or participant exclusion. Several factors have been shown to predict attrition during EXP (Minnen, Arntz, & Keijsers, 2002) which suggests that there may be an underlying pattern to missing data, violating a primary assumption of ANOVA based statistics and estimation methods. LMER accounts for both within (random effects) and between person (fixed effects) variance. Given the numerous advantages to LMER and the nature of treatment data, the following statistical strategy was implemented to evaluate the following hypotheses:

3) *The reduction of avoidance symptoms will significantly contribute to the prediction of guilt cognitions over the course of treatment.*
    - A mixed-effects regression was conducted to assess the effects of avoidance on the improvement in guilt symptoms. An LMER model of TRGI- Global

Guilt subscale was constructed that includes, time, avoidance from the PCL-M, and the interaction of these two terms.

4) *Participants with fewer self-reported guilt cognitions will demonstrate significantly greater reductions in Peak SUDs ratings over the course of treatment.*
    - A three level mixed-effects regression was conducted to assess the effects of guilt symptoms on habituation over the course of treatment. An LMER model of Peak SUDS was constructed that includes, time, the TRGI- Global Guilt subscale, and the interaction of these two terms.

5) *Participants with greater self-reported guilt symptoms as measured by the TRGI will demonstrate lower fear activation over the course of treatment than participants with fewer self-reported guilt symptoms.*
    - A three level mixed-effects regression was conducted to assess the effects of guilt symptoms on fear activation over the course of treatment. An LMER model of fear activation was constructed that includes, time, the TRGI- Global Guilt subscale, and the interaction of these two terms.

6) *A model containing Peak SUDs ratings as an index of habituation, guilt as measured by the TRGI, and a time marker for the group guilt intervention will demonstrate the best fit to the data predicting PTSD symptoms over the course of treatment.*

   - A five level mixed-effects regression was conducted to assess the effects of guilt symptoms, habituation, and the session eight guilt intervention on PTSD symptoms over the course of treatment.

## Data Preparation

Data was obtained from both standard (17-week) and intensive (3-week) TMT treatment trials. Although the majority of the proposed analyses were conducted with the three-week sample, the 17-week treatment trial was used as a point of comparison to reduce potential treatment confounds. Individuals in the 17-week protocol received group therapy only after completion of exposure therapy allowing the effects of exposure to be examined independently. The sample obtained for the purposes of this study was highly representative of the current veteran population as limited exclusion criteria were used and the active duty personnel and veterans recruited for the TMT project are largely veterans of the OIF/OEF conflicts. For hypotheses one and two, outliers were defined as ≥ three standard deviations from the mean of each guilt group (high/low). For hypotheses three through six, outliers and significant leverage cases were identified by examining individual participant plots. LMER does not require complete data and therefore no estimation method for missing data is required.

GPower 3.0.1(Faul, Erdfelder, Lang, & Buchner, 2007) was used to calculate the appropriate sample size for hypothesis one and two. Previous literature (Oktedalen, Hoffart, &

Lanngkaas, 2014; Stapelton, Taylor, & Asmundson, 2006) provided a sample size estimate of *d* = 0.71. With $\alpha = .05$, power $(1 - \beta) = .80$ and two groups, 66 participants (33 in each group) was needed to reliably reject the null hypothesis. Power estimates are not readily available for mixed-effects regression and therefore, the criterion of ten participants for each level of prediction was used. For adequate power, this would require 30 participants for hypothesis three through five and 50 participants for hypothesis six.

# CHAPTER THREE: RESULTS

## Preliminary Analysis

Prior to examining the linear and non-linear mixed model trends, preliminary analyses assessed if a significant change in guilt symptoms occurred over the course of the 3-week intervention. At pre-treatment, TRGI- Global Guilt (TRGI-GG) scores were similar ($t$ (40) = -1.47, $p$ = 0.15) to that of other treatment-seeking veterans reported in previous studies (Kubany, 1996). In testing hypothesis 1, the CAPS guilt item related to acts of commission or omission scores significantly decreased from pre-treatment to post-treatment ($Mdn_D$ = 5.5, V = 206.5, $p$ < .001) as did the CAPS item related to "survivors guilt" ($Mdn_D$ = 4.5, $V$ = 62, $p$ < .01). This finding is corroborated by a more comprehensive guilt measure as the participants TRGI-GG scores also significantly decreased from pre to post treatment ($Mdn_D$ = 1.3, $V$ = 117.5, $p$ < .001) (For $M$ and $SD$ see Table 2). Additionally, post-treatment TRGI-GG scores were similar ($t$ (27) = 1.10, $p$ = 0.28) to that of non-treatment seeking veterans (Kubany, 1996).

LMER analyses were conducted to further examine the change in guilt symptoms during treatment. A linear mixed-model was run that included 42 participants, 87 TRGI measurements, and assessed the effect of time (*exposure session*) on the TRGI subscales. Time significantly predicted the TRGI GG ($\beta$ = -.296, $SE$ = 0.051, $p$ < .001, $r^2$ = .72), Guilt Cognitions ($\beta$ = -.141, $SE$ = 0.034, $p$ < .001, $r^2$ = .77), and Distress ($\beta$ = -.304, $SE$ = 0.045, $p$ < .001, $r^2$ = .60) subscales that were all associated with a decrease over time. As a point of comparison, the same analysis was repeated for the 17-week participants that did not receive a group guilt intervention at any point during the exposure portion of the protocol. In the 17-week sample, time ($t$ = -5.08, $p$ <

.001, $r^2$= .76) also significantly predicted the TRGI GG score (See Table 3) and was associated with a similar cumulative reduction over time ($\beta_{3\text{-week}}$ = -1.2; $\beta_{17\text{-week}}$ = - .93).

To assess individual variation in the change of guilt symptoms over the course of treatment the slope of the TRGI-GG subscale was entered as a random effect. Time remained a significant predictor of the TRGI-GG score ($t$ = -5.281, $p$ <.001, $r^2$ = .85) and was associated with a decrease over the course of the 3-week intervention ($\beta$ = -.299, $SE$ = 0.057). The significance of the model indicates that individuals with a higher Global Guilt score at pretreatment demonstrated a greater rate of change than those with a lower Global Guilt score at pretreatment (See Figure 1). However, as this model was associated with similar fit as the model that did not include the TRGI-GG as a random effect ($AIC_{GG}$ = 366.861; $AIC_{GG+RE}$ = 367.889), the more parsimonious model was carried forward for additional analyses.

## Effects of Guilt on Treatment Outcome

To assess the effects of guilt on the overall treatment outcome a median split (CAPS median = 6, TRGI-GG median = 3) was performed to create a dichotomous high and low guilt group variable. No significant difference in the total score on the post PCL-M was found between participants with high and low guilt based on the CAPS "acts of commission or omission" ($W$ = 102.5, $p$ = 0.84) or the TRGI-GG ($t$ = -0.286, $p$ = 0.78) (See Table 4). Post Guilt scores were also not significantly different between high (≥ 50 percent CAPS reduction) and average (< 50 percent CAPS reduction) treatment responders regardless of whether guilt was

measured by the TRGI-GG ($t$ (6) = -0.55, $p$ = 0.60), CAPS acts of commission or omission item ($t$ (11) = -0.66, $p$ = .52) or CAPS survivors guilt item ($t$ (16) = 0.117, $p$ = .91).

Prior to examining the relationships between guilt and PTSD symptoms, a baseline model of the change in PTSD symptoms over the course of the 3-week treatment was constructed. The PCL-M included 102 participants, and 388 measurements at four time points (pre-treatment, session 6, session 11, and post-treatment). Time significantly predicted the PCL-M ($t$ = -16.059, $p$ <.001, $r^2$ = .66) and was associated with a decrease from pre to post-treatment ($\beta$ = -6.727, $SE$ = 0.419). To assess the impact of guilt on the trajectory of PTSD symptoms, the dichotomous high and low TRGI-GG group variable was entered into an LMER model that included time and the interaction between time and the guilt group variable. Time ($t$ = 25.56, $p$ < .001), guilt group ($t$ = 2.79. $p$ < .01), and the interaction term ($t$ = -2.63, $p$ < .01) all significantly predicted the PCL-M. The model indicated that individuals in the high guilt group tended to begin treatment with higher PCL-M scores and improve faster ($\beta$ = -9.7) over the course of treatment than individuals in the low guilt group ($\beta$ = -5.6)

Multiple LMER models assessed the effects of guilt on PTSD symptom trajectory in addition to the effects of PTSD symptoms on guilt trajectory over the course of treatment. Traditional LMER models compare the relationships between variables at the same time point. For example, in a LMER model including the TRGI-GG subscale predicting the PCL-M both time ($t$ = -6.67, $p$ <.001) and guilt ($t$ = 4.25, $p$ <.001) were significant. This model demonstrates that these two symptoms vary together and that each change in the PCL-M is associated with a corresponding change in the TRGI-GG (See Table 5). An alternative approach to establish the role of each variable in predicting the other over the course of treatment is to lag one

measurement behind by one time point so that the model represents variable x at one time point predicting variable y at the next time point.

Lagged regression analyses were conducted to examine the causal relationship between symptoms of PTSD and guilt over the course of treatment (See Table 6). In the first model, time and the PCL-M score from the previous session were used as predictors of the TRGI-GG subscale. The lagged PCL-M variable significantly predicted the subsequent TRGI score ($t$ = 2.88, $p < .01$); however, time was no longer a significant predictor ($t$ = -1.526, $p$ =.13). For the inverse model, the lagged TRGI-GG score ($t$ = 2.41, $p < .02$) significantly predicted the subsequent PCL-M score and time ($t$ = -3.86, $p < .001$) remained a significant predictor in the model. These results were replicated in the 17-week data (See Table 7).

To determine if these results were consistent for individuals with significant guilt symptoms, the dichotomous high and low guilt group variables based on the TRGI-GG subscale and CAPS guilt item were assessed with LMER models. The resulting groups for the TRGI-GG included 25 participants in the high group and 16 participants in the low group and for the CAPS 16 in the high guilt group and 18 in the low guilt group. In the high guilt group, the results were similar as described above (See Table 4). However, for the low group, neither the lagged PCL-M ($t$ = 0.222, $p$ = .83) nor the lagged Global Guilt ($t$ = 1.104, $p$ = .28) models significantly accounted for change in the other variable in the subsequent session. Despite both the high ($\beta$ = -8.67, $SE$ = 1.018, $p < .001$, $r^2$ = .61) and low ($\beta$ = -5.5, $SE$ = 0.051, $p < .001$, $r^2$ = .60) groups demonstrating change over time in PTSD symptoms and guilt.

## Treatment Mechanism

### Session Eight Guilt Intervention

Prior to considering the impact of guilt on specific treatment mechanisms, a discontinuity analysis was conducted to examine the effects of the session eight guilt intervention on the trajectory of guilt symptoms over the course of treatment. Although the reduction in guilt over the course of treatment remained significant over time ($t = 7.11$, $p < .01$), the trajectory of guilt was unchanged by the inclusion of the session eight-guilt intervention ($\beta = -0.10$, $SE = 0.252$, p = 0.69). Additionally, the interaction of time and the session eight variable ($t = 0.67$, $p = .51$) did not significantly predict the TRGI-GG subscale.

### Avoidance

In testing hypothesis 3, an LMER model with 33 participants and 109 observations included time and an avoidance change ($Pre_{PCLAvd} - Post_{PCLAvd}$) score (See Table 8). Although the avoidance change score ($t = 0.387$, $p = .70$) was not a significant predictor of the TRGI-GG subscale, time and the interaction of time and avoidance change ($t = - 2.07$, $p = .04$) significantly predicted guilt scores. In support of hypothesis 3, this analysis indicated that individuals with higher avoidance change scores experienced faster changes in guilt over the course of treatment.

## Fear Activation and Habituation

To examine the treatment mechanisms of exposure therapy, LMER analyses assessed fear activation and between–session habituation and the role of these variables in predicting treatment outcome. On average participants received 9.6 imaginal exposure therapy sessions and 65.6 percent of participants achieved between-session habituation (50 percent reduction in Peak SUDS rating). Over the course of treatment, time significantly predicted peak SUDS ($t$ = -28.54, $p$ < .001) and fear activation ($t$ = -4.12, $p$ <.001) that were both associated with a decrease over time ($\beta_{PS}$ = -0.32, $\beta_{FA}$ = -1.14). Additionally, when LMER models were run that also included peak SUDS and fear activation as random effects, the peak SUDS model demonstrated significantly better fit with the random effect ($AIC_{PS}$ = 3182.12; $AIC_{PS+RE}$ = 2890.29), whereas the fear activation model did not demonstrate notably better fit with the random effect ($AIC_{FA}$ = 3270.80; $AIC_{FA+RE}$ = 3227.00). The best fitting and most parsimonious model was carried forward for additional analyses (See Tables 8 & 9). Peak SUDS ($t$ = 3.36, $p$ <. 001) and fear activation ($t$ = 2.26, $p$ <.05) also predicted the PCL-M score over the course of treatment and both variables were associated with reductions in PTSD symptoms ($\beta_{PS}$ = 0.97, $SE$ = .29; $\beta_{FA}$ = 0.41, $SE$ = .18) (See Table 10).

With regard to guilt, 81 percent achieved habituation in the high guilt group and 60 percent achieved habituation in the low guilt group. This group proportion difference was not significant ($\chi^2(1) = 1.17$, $p$ = .28). In testing hypothesis 2, the high and low group did not differ in the number of sessions required to achieve between-session habituation based on the TRGI-GG ($t$ (25) = -1.9, $p$ = .06), CAPS acts of commission or omission ($t$ (14) = - 1.68, $p$ = .11) or

CAPS survivors guilt ($t$ (18) = 0.28, $p$ =.78) scores. Hypothesis 4 and 5 were also not supported as time, guilt, and the interaction were not significant predictors of fear activation or peak SUDS ratings (See Tables 8 & 9).

In the evaluation of hypothesis 6, an LMER model was constructed that included time, peak SUDS and the TRGI-GG subscale predicting the PCL-M over the course of treatment. The session eight guilt variable was omitted from this analysis as prior analyses demonstrated that it did not affect guilt trajectory. Although time ($t$ = -2.19, $p$ < .05) and guilt ($t$ = 2.82, $p$ <.02) remained significant predictors of the PCL-M, peak SUDS ($t$ = 0.894, $p$ = .388) did not predict the PCL-M over in this model.

## Arousal

In the lagged PCL-M models described earlier, time no longer accounted for variability in the TRGI-GG subscale when the lagged PCL-M score was included in the model. To investigate further if reductions in anxious arousal predicted guilt over the course of treatment, an additional model examined if a lagged measure of general anxiety corresponded to changes in the TRGI-GG subscale over time. In this model, time ($t$ = -2.17, $p$ <.05) and the lagged anxiety variable ($t$ = 2.41, $p$ < .05) significantly predicted the TRGI-GG score at the next session ($r^2$ = 0.96) (See Table 6).

# CHAPTER FOUR: DISCUSSION

The purpose of this study was to examine the changes in guilt symptoms over the course of a three-week intensive Trauma Management Therapy Program and investigate the impact of these symptoms on theorized mechanisms of therapeutic action. TMT is based in exposure therapy, and unlike PE, does not teach temporary coping mechanisms (e.g., breathing retraining) or conduct emotional processing after each session. TMT also includes several group modules that emphasize skill building in areas (i.e., social reintegration, anger, and depression) not adequately addressed by PE or the direct mechanisms of exposure therapy (Beidel et al., 2014; Frueh, Turner, Beidel, Mirabella, & Jones, 1996). By basing treatment delivery directly on flooding principles and the underlying mechanisms of exposure therapy, TMT provided a unique opportunity to examine changes in guilt symptoms over the course of EXP without additional confounds such as emotional processing or cognitive restructuring.

Over the course of the three-week and 17-week TMT intervention, symptoms of guilt significantly decreased from a pre-treatment average similar to Vietnam veterans to a score similar to non-treatment seeking veterans (Kubany, 1996). LMER analyses provided a more thorough examination of guilt reduction over the course of treatment and revealed that the intervention was equally effective for individuals with high and low guilt. Guilt symptoms also did not negatively affect treatment outcome as participants with high and low guilt demonstrated no difference in PTSD symptom reduction.

As TMT is comprised of both individual and group therapy, we examined the individual contributions of each treatment component. Although the effects of exposure therapy were confounded with the group intervention that occurred simultaneously during the three-week

treatment, results were also replicated in the 17-week protocol that did not include a co-occurring group intervention. These analyses revealed that there was no detectable unique effect of the one session guilt intervention and that the reduction in guilt symptoms was primarily due to exposure therapy.

These findings are in agreement with previous literature reporting significant changes in guilt symptoms as a result of therapeutic interventions that include exposure therapy (Nishith et al., 2005; Oktedalen, Hoffartm, & Langkass, 2015; Resick et al., 2002; Zalta et al., 2014). Furthermore, when taken togetherthe results of this study suggest that the reductions in guilt reported in previous studies may be primarily due to the exposure component of PTSD treatment and not to emotional processing or additional added treatment components.

In this study, guilt also did not inhibit the underlying mechanisms of exposure therapy. Although both between-session habituation and fear activation were significantly related to overall PTSD symptom reduction, guilt did not affect fear activation. Furthermore, higher guilt scores were related to an increased rate of habituation. This finding is in the opposite direction of the stated hypotheses and may be secondary to the greater severity of PTSD symptomatology in the high guilt group. Participants also did not differ in the percentage that achieved between-session habituation or in those who were classified as high treatment responders.

To further examine the relationship between symptoms of PTSD and guilt, lagged regression analyses were conducted. The results of these analyses suggest that change in PTSD symptoms predict change in guilt and that change in guilt symptoms predict change in PTSD symptoms. However, in the LMER model that included lagged PTSD symptoms predicting guilt, the effect of time (# of sessions) was not significant. This was in contrast to the lagged guilt

model where time remained a significant predictor. Although multicollinearity cannot be ruled out, further lagged regression analyses revealed that over the course of treatment, a participant's arousal (i.e., general anxiety) significantly predicted changes in guilt scores. Furthermore, greater reductions in avoidance over the course of treatment were associated with a more rapid improvement in guilt symptoms. Interestingly, the exposure component of treatment only targeted avoidance of distress directly related to anxiety. These findings suggest that a reduction in the participant's anxiety-related distress has the secondary benefit of altering guilt attributions associated with aversive physiological arousal. In the absence of this aversive physiological reactivity, guilt attributions may be subjectively experienced as less distressing and less meaningful. Additionally, directly targeting anxious arousal or general distress may allow for greater and more efficient treatment gains (Beidel et al., 2016) that extend beyond directly targeted mechanisms. This may also partially explain previous findings reporting reductions in symptoms like depression in exposure therapy trials (Minnen et al., 2012; Powers et al., 2010).

This study provided a detailed examination of change processes during exposure therapy; however, there are some limitations. Perhaps the largest limitation is due to the size of the sample. Although, the sample size is comparable to that of other studies examining changes in cognitions during exposure therapy (Oktedalen, Hoffartm, & Langkass, 2015; Zalta et al., 2014), a larger sample would allow for greater generalization to diverse trauma types and symptom presentations. Due to the expanding criteria for PTSD, the use of the DSM-IV criteria may have biased the sample selection toward a more anxious symptom presentation. Directly targeting anxious distress may not be possible or effective in more guilt or cognitive symptom presentations based on newer, broader conceptualizations of PTSD. Furthermore, the study did

not differentiate between shame and guilt, which may be theoretically distinct (Tanney et al., 2007), and did not examine anxious related cognitions that may change differently than non-anxious cognitions over the course of therapy. Finally, the study also relied exclusively on subjective ratings of both anxious and guilt related distress. Participants may have found it difficult to differentiate between these two mechanisms.

The general arousal hypothesis described above may be more directly assessed with physiological measurements. Future research should explore more concrete methods for assessing arousal reduction during exposure therapy and the influence of this reduction on the specific trajectories of trauma related attributions and cognitions. Future research should also continue to explore the mechanisms of exposure therapy responsible for secondary benefits in other non-anxiety related symptoms like shame, anhedonia, and maladaptive cognitions. For example, a prospective dismantling study of exposure therapy would provide insight into additional treatment mechanisms and uncover methods to increase the efficiency of efficacy trauma focused therapies. A more thorough understanding of the mechanisms associated with different treatment components could potentially lead to strategies to match patients with differing treatment presentations to specific treatments. Future research should continue to look beyond treatment outcome and explore how modifications in exposure therapy (e.g., intensive approaches, the addition of cognitive restructuring) may alter specific symptom trajectories.

In conclusion, the current study is the first to provide evidence that guilt symptoms do not inhibit the mechanisms of exposure therapy. Additionally, the findings of this study suggest that the reductions in guilt symptoms reported in previous studies may be attributable to the exposure component of treatment and subsequent reductions in avoidance rather than emotional

processing. This study also identifies a potential exposure mechanism for a secondary benefit in guilt symptoms as reductions in general anxiety lessen subjective aversive arousal that the patient may no longer attribute to guilt cognitions.

# APPENDIX A: FIGURES

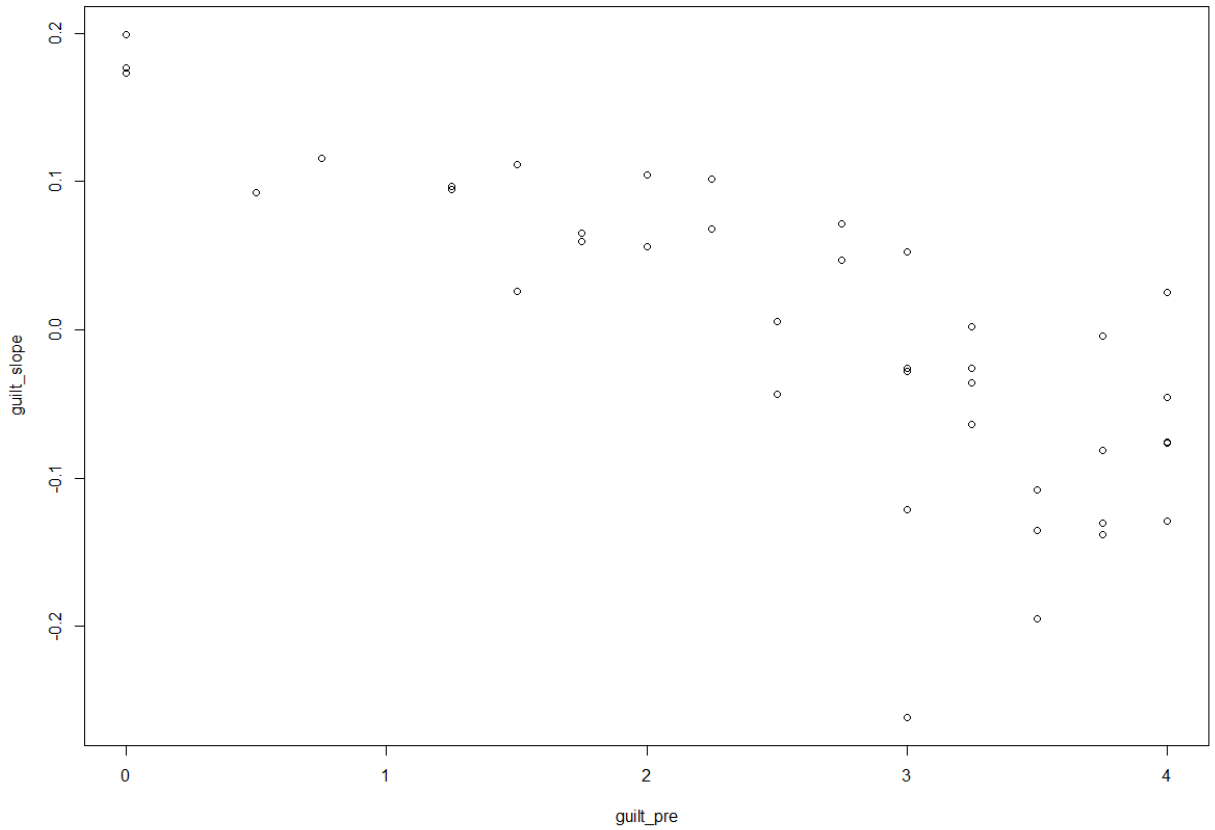


Figure 1. Rate of Change and Pre-Guilt

# APPENDIX B: TABLES

Table 1. Sample demographics

| | Sample with PCL-M | Sample with TRGI | Sample with Supplemental CAPS |
|---|---|---|---|
| | $\bar{x}$ (s) | $\bar{x}$ (s) | $\bar{x}$ (s) |
| Age | 37.1 (9.1) | 37 (8.2) | 37.8 (8.7) |
| | N (%) | | |
| Gender | | | |
| Male | 97 (95) | 41 (98) | 38 (97.4) |
| Female | 5 (5) | 1 (2) | 1 (2.6) |
| Race | | | |
| Caucasian | 67 (65.7) | 30 (71.4) | 27 (69.2) |
| Hispanic | 15 (14.7) | 6 (14.3) | 6 (15.4) |
| Black | 12 (11.8) | 2 (4.8) | 2 (5.1) |
| Other | 8 (7.8) | 4 (9.5) | 4 (10.3) |
| Education | | | |
| High School Diploma | 17 (16.7) | 8 (19.1) | 8 (20.5) |
| Some College | 61 (59.8) | 27 (64.3) | 24 (61.5) |
| Bachelors | 16 (15.7) | 4 (9.5) | 4 (10.3) |
| Graduate | 8 (7.8) | 3 (7.1) | 3 (7.7) |
| Marital Status | | | |
| Single | 17 (16.7) | 5 (11.9) | 5 (12.8) |
| Married | 55 (53.9) | 22 (52.4) | 20 (51.3) |
| Separated | 10 (9.8) | 5 (11.9) | 9 (23.1) |
| Divorced | 20 (19.6) | 10 (23.8) | 5 (12.8) |
| Military Branch | | | |
| Army | 74 (72.5) | 27 (64.3) | 25 (64.1) |
| Marines | 11 (10.8) | 5 (11.9) | 4 (10.3) |
| Navy | 7 (6.9) | 6 (14.3) | 6 (15.4) |
| Airforce | 9 (8.8) | 4 (9.5) | 4 (10.3) |
| Coast Guard | 1 (≈1) | 0 (0) | 0 (0.0) |
| Service Connected Disability | | | |
| Service Connected | 51 (50) | 18 (42.9) | 16 (41.0) |
| None/Not Applicable | 51 (50) | 24 (57.1) | 23 (59.0) |
| Average Disability % | 74.5% | 68.8% | 72.67% |
| Total | 102 (100%) | 42 (100%) | 39 (100%) |

Table 2. Pre-Post Subscale Differences

| | *Pre-Treatment* | | | *Post-Treatment* | | |
|---|---|---|---|---|---|---|
| *Measure* | *n* | *M* | *SD* | *n* | *M* | *SD* |
| TRGI | | | | | | |
| Global Guilt** | 40 | 2.57 | 1.20 | 28 | 1.66 | 1.16 |
| Guilt Cognitions** | 41 | 1.63 | 0.90 | 29 | 1.17 | 0.93 |
| Distress*** | 41 | 3.17 | 0.61 | 29 | 2.28 | 0.97 |
| Hindsight Bias** | 41 | 1.65 | 1.16 | 29 | 1.06 | 1.14 |
| Lack of Justification* | 41 | 2.03 | 1.18 | 29 | 1.58 | 1.17 |
| Wrongdoing** | 41 | 1.68 | 0.98 | 29 | 1.21 | 1.17 |
| CAPS | | | | | | |
| Item 26: Co/Omission** | 34 | 4.68 | 2.99 | 30 | 1.03 | 1.83 |
| Item 27: Survivor's Guilt** | 31 | 3.48 | 3.25 | 27 | 1.22 | 2.28 |

*Reflects significance for parametric/nonparametric tests.

P<.05*

P<.01**

P<.001***

Table 3. Linear Mixed Effect Regression TRGI
*Measure*

| | β | *SE* | *t* |
|---|---|---|---|
| **TRGI-Global Guilt** | | | |
| *Random Effects* | Estimate | SD | |
| $\sigma^2$ | 0.413 | 0.643 | |
| *Fixed Effects* | | | |
| *Intercept* | 2.867 | 0.198 | 14.484*** |
| *Time* | -0.296 | 0.051 | -5.765*** |
| *AIC* | 366.861 | | |
| *BIC* | 378.482 | | |
| *Marginal* $R^2$ | 0.076 | | |
| *Conditional* $R^2$ | 0.715 | | |
| **TRGI-Global Guilt with slope parameter as RE** | | | |
| *Random Effects* | Estimate | SD | |
| $\sigma^2$ | 0.352 | 0.593 | |
| *Fixed Effects* | | | |
| *Intercept* | 2.877 | 0.222 | 12.939*** |
| *Time* | -0.299 | 0.057 | -5.281*** |
| *AIC* | 367.889 | | |
| *BIC* | 385.32 | | |
| *Marginal* $R^2$ | 0.047 | | |
| *Conditional* $R^2$ | 0.851 | | |
| **TRGI-Guilt Cognitions** | | | |
| *Random Effects* | | | |
| $\sigma^2$ | 0.188 | 0.434 | |
| *Fixed Effects* | | | |
| *Intercept* | 1.749 | 0.148 | 11.768*** |
| *Time* | -1.141 | 0.034 | -4.106*** |
| *AIC* | 280.603 | | |
| *BIC* | 292.312 | | |
| *Marginal* $R^2$ | 0.03 | | |
| *Conditional* $R^2$ | 0.774 | | |
| **TRGI-Distress** | | | |
| *Random Effects* | | | |
| $\sigma^2$ | 0.33 | 0.574 | |
| *Intercept* | 3.391 | 0.148 | 22.869*** |
| *Time* | -0.304 | 0.045 | -6.773*** |
| *AIC* | 320.326 | | |
| *BIC* | 332.035 | | |
| *Marginal* $R^2$ | 0.141 | | |
| *Conditional* $R^2$ | 0.599 | | |
| | | | |
| **TRGI Global Guilt 17-week** | | | |
| *Random Effects* | | | |
| $\sigma^2$ | 0.79 | 0.890 | |

| | | | |
|---|---|---|---|
| *Fixed Effects* | | | |
| *Intercept* | 2.47 | 0.210 | 11.724*** |
| *Time* | -0.133 | 0.026 | -5.08*** |
| *AIC* | 249.110 | | |
| *BIC* | 259.985 | | |
| *Marginal* $R^2$ | .06 | | |
| *Conditional* $R^2$ | .76 | | |

P<.05*
P<.01**
P<.001***

Table 4. High and Low Guilt Means and SDs for Treatment Completers.

| | *Pre-PCL-M*** | | | *Post-PCL-M*** | | |
|---|---|---|---|---|---|---|
| *Measure* | *n* | *M* | *SD* | *n* | *M* | *SD* |
| TRGI- Global Guilt | | | | | | |
| High | 16 | 69.13 | 9.76 | 14 | 40.86 | 12.11 |
| Low | 25 | 60.44 | 10.70 | 21 | 43.62 | 14.81 |
| CAPS | | | | | | |
| Item 26: Co/Omission | | | | | | |
| High | 16 | 67.19 | 11.41 | 13 | 44.38 | 16.90 |
| Low | 18 | 60.50 | 9.38 | 15 | 40.07 | 10.36 |

P<.05*
P<.01**
P<.001***

Table 5. PCL-M and TRGI-GG

| *Measure* | *β* | SE | t |
|---|---|---|---|
| **PCL-M** | | | |
| *Random Effects* | Estimate | SD | |
| $\sigma^2$ | 9.095 | 82.716 | |
| *Fixed Effects* | | | |
| *Intercept* | 68.034 | 1.512 | 45.008*** |
| *Time* | -6.727 | 0.419 | -16.059*** |
| *AIC* | 2997.639 | | |
| *BIC* | 3013.462 | | |
| *Marginal* $R^2$ | 0.232 | | |
| *Conditional* $R^2$ | 0.662 | | |
| **PCL-M** | | | |
| *Random Effects* | | | |
| $\sigma^2$ | 45.83 | 6.77 | |
| *Fixed Effects* | | | |
| *Intercept* | 55.890 | 3.72 | 15.02*** |
| *Time* | -5.423 | .830 | -6.67*** |
| *TRGI-GG* | 4.423 | 1.04 | 4.247*** |
| *AIC* | 1030.106 | | |
| *BIC* | 0.587 | | |
| *Marginal* $R^2$ | .36 | | |
| *Conditional* $R^2$ | .58 | | |
| **PCL-M** | | | |
| *Random Effects* | | | |
| *Time* | 7.50 | 2.74 | |
| *Fixed Effects* | | | |
| *Intercept* | 64.25 | 2.51 | |
| *Time* | - 5.55 | 0.98 | |
| *Guilt Group* | 11.20 | 4.01 | |
| *Guilt Group x Time* | -4.09 | 1.56 | |
| *AIC* | 1182.28 | | |
| *BIC* | 1206.42 | | |
| *Marginal* $R^2$ | .19 | | |
| *Conditional* $R^2$ | .80 | | |

P<.05*
P<.01**
P<.001***

Table 6. Three-Week Treatment Lagged Regression Results

| *Measure* | *β* | SE | t | p |
|---|---|---|---|---|
| **TRGI-Global Guilt** | | | | |
| *Random Effects* | Estimate | SD | | |
| $\sigma^2$ | 0.463 | 0.681 | | |
| *Fixed Effects* | | | | |
| *Intercept* | 1.269 | 0.660 | 1.925 | 0.060 |
| *Time* | -0.16 | 0.108 | -1.526 | 0.133 |
| *Lagged PCL-M* | 0.023 | 0.008 | 2.880 | 0.006 |
| *AIC* | 268.75 | | | |
| *BIC* | 281.25 | | | |
| $R^2$ | .64 | | | |
| **PCL-M** | | | | |
| *Random Effects* | Estimate | SD | | |
| $\sigma^2$ | 60.773 | 7.796 | | |
| *Fixed Effects* | | | | |
| *Intercept* | 52.500 | 4.798 | 10.943 | <.001 |
| *Time* | -4.01 | 1.040 | -3.856 | <.001 |
| *Lagged TRGI GG* | 2.827 | 1.170 | 2.414 | 0.0191 |
| *AIC* | 752.65 | | | |
| *BIC* | 765.473 | | | |
| $R^2$ | .67 | | | |
| **TRGI- Global Guilt** | | | | |
| *Random Effects* | Estimate | SD | | |
| $\sigma^2$ | .80 | -.898 | | |
| *Fixed Effects* | | | | |
| *Intercept* | 2.08 | .599 | 3.47 | < .001 |
| *Time* | - 0.29 | .14 | -2.17 | < .05 |
| *Lagged Anxiety* | 2.827 | 1.170 | 2.414 | < .05 |
| *AIC* | 159.04 | | | |
| *BIC* | 172.42 | | | |
| $R^2$ | .96 | | | |

P<.05*
P<.01**
P<.001***

Table 7. Lagged Regression Results 17-Week

| *Measure* | *β* | SE | t | p |
|---|---|---|---|---|
| **TRGI-Global Guilt** | | | | |
| *Random Effects* | Estimate | SD | | |
| $\sigma^2$ | 0.255 | 0.505 | | |
| *Fixed Effects* | | | | |
| *Intercept* | 0.900 | 0.514 | 1.750 | 0.085 |
| *Time* | -0.027 | 0.045 | -0.586 | 0.560 |
| *Lagged PCL-M* | 0.022 | 0.007 | 3.325 | 0.002 |
| *AIC* | 195.07 | | | |
| *BIC* | 207.17 | | | |
| *Marginal* $R^2$ | 0.135 | | | |
| *Conditional* $R^2$ | 0.749 | | | |
| **PCL-M** | | | | |
| *Random Effects* | Estimate | SD | | |
| $\sigma^2$ | 77.656 | 8.812 | | |
| *Fixed Effects* | | | | |
| *Intercept* | 50.263 | 4.749 | 10.583 | <.001 |
| *Time* | -3.199 | 0.643 | -4.973 | <.001 |
| *Lagged TRGI GG* | 5.450 | 1.367 | 3.986 | <.001 |
| *AIC* | 752.650 | | | |
| *BIC* | 765.473 | | | |
| *Marginal* $R^2$ | 0.407 | | | |
| *Conditional* $R^2$ | 0.605 | | | |

P<.05*
P<.01**
P<.001***

Table 8. Peak SUDS

| *Measure* | *β* | SE | t |
|---|---|---|---|
| **Peak SUDS** | | | |
| *Random Effects* | Estimate | SD | |
| $\sigma^2$ | 1.14 | 1.07 | |
| *Fixed Effects* | | | |
| *Intercept* | 7.95 | .13 | 59.367*** |
| *Time* | -0.32 | 0.01 | -28.54*** |
| *AIC* | 3174.89 | | |
| *BIC* | 3194.31 | | |
| *Marginal* $R^2$ | 0.45 | | |
| *Conditional* $R^2$ | 0.70 | | |
| **Peak SUDS and RE** | | | |
| *Random Effects* | Estimate | SD | |
| *Time* | .05 | .240 | |
| *Fixed Effects* | | | |
| *Intercept* | 8.41 | 0.11 | 75.48*** |
| *Time* | -0.43 | 0.03 | -15.77*** |
| *AIC* | 2884.11 | | |
| *BIC* | 2913.23 | | |
| *Marginal* $R^2$ | 0.34 | | |
| *Conditional* $R^2$ | 0.92 | | |
| **Peak SUDS and RE** | | | |
| *Random Effects* | Estimate | SD | |
| *Time* | .54 | .74 | |
| *Fixed Effects* | | | |
| *Intercept* | 7.67 | 1.52 | 5.05*** |
| *Time* | -1.07 | .66 | -1.60 |
| *Guilt* | -0.17 | .66 | -0.26 |
| *Guilt x Time* | 0.11 | 0.29 | 0.38 |
| *AIC* | 212.79 | | |
| *BIC* | 223.76 | | |
| *Marginal* $R^2$ | .22 | | |
| *Conditional* $R^2$ | .37 | | |

P<.05*
P<.01**
P<.001***

Table 9. Fear Activation

| Measure | $\beta$ | SE | t |
|---|---|---|---|
| **Fear Activation** | | | |
| *Random Effects* | | | |
| $\sigma^2$ | 1.12 | 1.12 | |
| *Fixed Effects* | | | |
| *Intercept* | 3.36 | 0.14 | 23.45*** |
| *Time* | -0.13 | 0.012 | -10.42*** |
| *AIC* | 3270.80 | | |
| *BIC* | 3290.17 | | |
| *Marginal* $R^2$ | .10 | | |
| *Conditional* $R^2$ | .51 | | |
| **Fear Activation and RE** | | | |
| *Random Effects* | Estimate | SD | |
| *Time* | 1.32 | 1.15 | |
| *Fixed Effects* | | | |
| *Intercept* | 3.44 | 0.17 | 19.92*** |
| *Time* | -0.15 | 0.02 | -7.52*** |
| *AIC* | 3227.0 | | |
| *BIC* | 3256.06 | | |
| *Marginal* $R^2$ | .07 | | |
| *Conditional* $R^2$ | .60 | | |
| **Fear Activation** | | | |
| *Random Effects* | Estimate | SD | |
| $\sigma^2$ | 2.8 | 1.68 | |
| *Intercept* | 2.917 | 1.012 | 0.007 |
| *Time* | -0.267 | 0.231 | 0.27 |
| *Guilt* | -0.058 | 0.39 | 0.99 |
| *Guilt x Time* | | | |
| *AIC* | 199.98 | | |
| *BIC* | 209.02 | | |
| *Marginal* $R^2$ | .03 | | |
| *Conditional* $R^2$ | .13 | | |

P<.05*
P<.01**
P<.001***

Table 10. Mechanisms Model

| *Measure* | $\beta$ | SE | t |
|---|---|---|---|
| **TRGI-GG** | | | |
| *Random Effects* | Estimate | SD | |
| $\sigma^2$ | 1.10 | 1.047 | |
| *Fixed Effects* | | | |
| *Intercept* | 2.66 | 0.32 | 8.29*** |
| *Time* | -0.180 | 0.08 | -2.36* |
| *Avoidance Change* | 0.080 | 0.08 | 1.01 |
| *Avoidance Change x time* | -4.09 | 1.56 | -2.07* |
| *AIC* | | | |
| *BIC* | | | |
| *Marginal* $R^2$ | | | |
| *Conditional* $R^2$ | | | |
| **PCL-M** | | | |
| *Random Effects* | | | |
| *Peak SUDS* $\sigma^2$ | 4.48 | 2.117 | |
| | | | |
| *Fixed Effects* | | | |
| *Intercept* | 59.56 | 2.390 | 24.914*** |
| *Time* | -1.291 | 0.841 | -15.346*** |
| *Peak SUDS* | 0.975 | 0.290 | 3.358*** |
| *AIC* | 320.326 | | |
| *BIC* | 332.035 | | |
| *Marginal* $R^2$ | 0.141 | | |
| *Conditional* $R^2$ | 0.599 | | |
| **PCL-M** | | | |
| *Random Effects* | | | |
| $\sigma^2$ | 105.5 | 10.27 | |
| | | | |
| *Fixed Effects* | | | |
| *Intercept* | 66.134 | 1.289 | 51.28*** |
| *Time* | -1.595 | 0.071 | -22.37*** |
| *Fear Activation* | .410 | .181 | 2.259*** |
| *AIC* | 6453.18 | | |
| *BIC* | 6477.34 | | |
| *Marginal* $R^2$ | 0.18 | | |
| *Conditional* $R^2$ | 0.76 | | |

P<.05*
P<.01**
P<.001***

# 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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