Behaviour Research and Therapy 102 (2018) 68–77

Contents lists available at ScienceDirect

Behaviour Research and Therapy

journal homepage: www.elsevier.com/locate/brat

Combat-related guilt and the mechanisms of exposure therapy

Benjamin Trachik[a,b,*], Clint Bowers[a], Sandra M. Neer[a], Vu Nguyen[a], B. Christopher Frueh[c], Deborah C. Beidel[a]

[a] UCF RESTORES and Department of Psychology, University of Central Florida, 4111 Pictor Lane, Orlando, FL 32816-1390, United States
[b] VA Puget Sound Health Care System, Seattle, 1660 S. Columbian Way, Seattle, WA 98108-1597, United States
[c] Department of Psychology, University of Hawaii, Hilo, HI, United States

ARTICLE INFO

Keywords:
Posttraumatic stress disorder
Combat
Exposure therapy
Guilt
Emotional processing

ABSTRACT

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 potential mechanisms underlying TMT treatment. TMT is an exposure based intervention that does not include an emotional processing component after the imaginal exposure session. A portion of the sample completed measures of guilt. As a result, sample size for these analyses ranged from 39 to 102 and varied by the domain and measure. Of the 102 individuals that completed the PTSD Checklist- Military Version, 42 completed the Trauma Related Guilt Inventory, and 39 completed the Clinician Administered PTSD Scale supplemental guilt items. Participants reported significant reductions in trauma-related guilt symptoms over the course of the TMT interventions. Greater reductions in avoidance and prior session general arousal predicted the reduction of guilt symptoms. Exposure therapy may be effective in reducing trauma-related guilt even in the absence of the emotional processing component of treatment.

1. Introduction

The treatment of posttraumatic stress disorder (PTSD) is a significant public health concern for the Department of Defense, 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). 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, 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., 2013).

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, Madden, Cohen, Bertenthal, & Seal, 2012; Rosenheck & 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.

Randomized-controlled trials have repeatedly demonstrated the efficacy of exposure therapy (EXP) for PTSD (Benish, Imel, & Wampold, 2008; Foa & Rauch, 2004; Foa, Keane, Friedman, & Cohen, 2008; Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010; Rothbaum et al., 2014), and although EXP is a well-supported intervention for PTSD, it is not associated with universal improvement, as a portion of individuals see minimal or no symptom reduction as a result of EXP (Bradley, Greene, Russ, Dutra, & Westen, 2005; Roberts, Kitchiner, Kenardy, & Bisson, 2009; Rothbaum et al., 2014). Further, the percentage of

Abbreviations: EPT, Emotional Processing Theory; EXP, Exposure Therapy; TRGI-GG, Trauma-related Guilt Inventory-Global Guilt

* Corresponding author. UCF RESTORES, Department of Psychology, University of Central Florida, 4000 Central Florida Blvd, Orlando, FL 32816, United States.
E-mail addresses: ben.trachik@knights.ucf.edu (B. Trachik), clint.bowers@knights.ucf.edu (C. Bowers), sandra.neer@ucf.edu (S.M. Neer), vu.nguyen@ucf.edu (V. Nguyen), frueh@hawaii.edu (B.C. Frueh), deborah.beidel@ucf.edu (D.C. Beidel).

https://doi.org/10.1016/j.brat.2017.11.006
Received 1 May 2017; Received in revised form 13 November 2017; Accepted 24 November 2017
Available online 27 November 2017
0005-7967/ Published by Elsevier Ltd.

treatment non-responders appears to be larger in military and veteran samples (Steenkamp, Litz, Hoge, & Marmar, 2015), a problem that is compounded by the significant dropout rates (17–52 percent) observed in this population (Gros, Yoder, Tuerk, Lozano, & Acierno, 2011; McLay et al., 2011; Reger et al., 2011; Strachan, Gros, Ruggiero, Lejuez, & Acierno, 2012; Tuerk, Yoder, Ruggiero, Gros, & Acierno, 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, & Ann Cox, 2008; Resick, Nishith, Weaver, Astin, & Feuer, 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, 1992) or Imagery Rescripting (Smucker & Dancu, 1999).

The theoretical underpinnings of EXP are largely based in animal research, and 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 (Hofmann, 2008; Lovibond, 2004). In a review of the cognitive processes during fear acquisition and extinction learning, Hofmann (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, & Langkaas, 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, 2013; Meiser-Stedman, Dalgleish, Glucksman, Yule, & Smith, 2009; Moser, Hajcak, Simons, & Foa, 2007). In addition, Litz et al. (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-5) 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 or shame.

Prior to the most recent DSM revision, PTSD had been classified as an anxiety disorder and was considered to represent maladaptive 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 (Harman & Lee, 2010). The association between guilt and PTSD is particularly strong among military and 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, Ray-Sannerud, Morrow, & Etienne, 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 (Haley, 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 combat veteran samples (Steenkamp et al., 2015).

Specifically, the emotional experience of guilt has been the subject of considerable debate regarding its relationship to maladaptive outcomes (Tilghman-Osborne, Cole, & Felton, 2010) and response to existing PTSD treatments (Rauch, Smith, Duax, & Tuerk, 2013; Smith, Duax, & Rauch, 2013; Steenkamp et al., 2013). In veteran populations, definitions of guilt consistent with the definition provided by Tilghman-Osborne et al. (2010) are associated with negative outcomes including depression and a higher risk of suicidal behavior (Bryan et al., 2015; Hendin & Haas, 1991; Henning & Frueh, 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, 1995; Pitman et al., 1991 as cited in; Pugh et al., 2015) resulting in avoidance and the reinforcement of trauma-related psychopathology (Held, Owens, Schumm, Chard, & Hansel, 2011; Street, Gibson, & Holohan, 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 et al., 2015). In a review of the literature concerning guilt and PTSD, Pugh et al. (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.

Efficacy studies of EXP for PTSD have also examined outcomes related to guilt. 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 et al., 1996) and depression (Rauch et al., 2013); however, the specific mechanisms by which these changes occur are unclear. Rauch et al. (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, Nixon, & Resick, 2005; Oktedalen, Hoffart, & Langkass, 2015; Resick et al., 2002; Zalta et al., 2014), others report limited improvement in guilt and shame symptoms (Arntz, Tiesema, & Kindt, 2007; Grunert, Smucker, Weis, & Rusch, 2003; &; Grunert, Weis, Smucker, & Christianson, 2007). Furthermore, studies attempting to augment PE with cognitive restructuring have either found no improvement over and above

traditional PE (Aderka, Gillihan, McLean, & Foa, 2013; Foa et al., 2005), or significantly worse outcomes (Moser, Cahill, & Foa, 2010). These findings suggest that additional treatment components explicitly targeting trauma-related cognitions may hinder the effects of PE or may not provide sufficient time for treatment elements to be implemented.

Studies examining the temporal order of PTSD symptom change during PE have shown that changes in maladaptive cognitions (McLean, Yeh, Rosenfield, & Foa, 2015; Zalta et al., 2014) and guilt (Oktedalen et al., 2015) precede changes in other PTSD symptoms. However, these studies were not conducted with military or combat veterans that experience unique traumatic events (Hoge et al., 2004; Litz et al., 2009). Additionally, in one of these studies, Zalta et al. (2014) assessed trauma-related cognitions using the Post-Traumatic Cognitions Inventory (PTCI; Foa, Ehlers, Clark, Tolin, & Orsillo, 1999). 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 et al. (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 et al. (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 et al., 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 at the end of 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 et al., 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. TMT is designed to be delivered in a 17-week or intensive 3-week format. When conducted in an intensive 3-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. In the 17-week format all exposure therapy sessions are completed before group therapy begins. 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 overall treatment outcome. Furthermore, data from two treatment studies allows for the examination of the exposure therapy mechanisms associated with change in trauma-related guilt cognitions.

The present study examined guilt cognitions as time-varying predictors of treatment outcome and attempted to identify the potential mechanisms of TMT. Based on the PTSD treatment literature, the following hypotheses were tested:

1) Guilt cognitions as measured by the TRGI and guilt related to acts of "commission or omission" and "survivors guilt" as measured by the CAPS, will significantly improve from pre to post treatment.
2) Participants endorsing less subjective guilt will achieve greater PTSD symptom improvement as a result of exposure therapy.
3) The reduction of guilt will significantly predict subsequent changes in PTSD symptoms and general anxiety over the course of treatment.
4) The reduction of avoidance symptoms will significantly contribute to the reduction of guilt cognitions over the course of treatment.

2. Method

2.1. Study overview

Data was collected as part of two treatment studies funded by the Department of Defense. The Intensive TMT study evaluated the efficacy of a 3-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 3-week protocol, patients participated in daily EXP sessions and group therapy. Under the supervision of licensed clinical psychologists, doctoral students conducted all assessments and provided the treatment. Participants were compensated 50 dollars for completing pre-treatment and post-treatment assessments.

2.2. Participants

The sample consisted of treatment-seeking veterans as well as active-duty military personnel. Exclusion criteria were intentionally minimized to obtain a representative sample of individuals with combat trauma. 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 participants completed two measures related to guilt (See Table 1 for additional demographics).

2.3. Trauma management therapy protocols

Trauma Management Therapy (TMT) (Beidel, Stout, Neer, Frueh, & Lejuez, 2017; Frueh, Turner, Beidel, Mirabella, & Jones, 1996) is a

Table 1
3-week sample demographics.

	Sample with PCL-M (n = 102)	Sample with TRGI (n = 42)	Sample with Supplemental CAPS (n = 39)
	x̄ (s)	x̄ (s)	x̄ (s)
Age	37.1 (9.1)	37 (8.2)	37.8 (8.7)
	n (%)		
Gender			
Male	97 (95.1)	41 (97.6)	38 (97.4)
Female	5 (4.9)	1 (2.4)	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 (0.0)
Service Connected Disability			
Service Connected	51 (50.0)	18 (42.9)	16 (41.0)
None/Not Applicable	51 (50.0)	24 (57.1)	23 (59.0)
Average Disability %	74.5%	68.8%	72.67%

behavioral-based treatment specifically designed to address the needs of combat veterans diagnosed with PTSD. The current TMT protocol includes virtual-reality (VR) assisted imaginal exposure, in-vivo exposures, and group therapy sessions conducted over the course of 17-weeks. In the 17-week protocol imaginal exposure sessions are conducted one to three times per week depending on the participants availability. Participants in the 17-week treatment successfully complete all exposure therapy sessions prior to the initiation group therapy. 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, Turner, & Beidel, 1995; Stapleton, Taylor, & Asmundson, 2006). Because the current protocol did not appear to substantially affect sleep disturbance, going forward the number of social reintegration sessions will be decreased in order to add some sessions devoted to improving sleep.

The intensive TMT protocol (Beidel, Frueh, Neer, Bowers, & Rizzo, 2014) was conducted five days a week, over the course of three weeks (See Fig. 1 for CONSORT diagram). Each day, patients participated in imaginal exposure and group therapy sessions (15 individual/14 group sessions). The initial exposure session consistent of diagnostic interviews and pre-treatment data collection followed by psychoeducation and development of the imaginal exposure scene. Development of the exposure scene included “testing” the components of the virtual reality to align with the individuals description of their traumatic event. The imaginal scene was then written and approved by the primary investigators. Beginning in session 2, baseline level of distress was assessed and then the imaginal scene was presented. 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 to ten minutes, until the patient demonstrated a 50 percent reduction in SUDS ratings from that session's Peak SUDS rating, or demonstrated a return to that session's 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 3-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 doctoral clinicians and patients were provided with daily session-related assignments to be completed outside of group. Group therapy modules were presented in a staggered 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.

2.4. Data preparation

Data was obtained from both standard (17-week) and intensive (3-week) TMT treatment trials. 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 served as part of OIF/OEF/OND.

3. Measures

3.1. 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 was administered at pre-treatment and one-week post treatment.

3.2. 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) was calculated by summing the

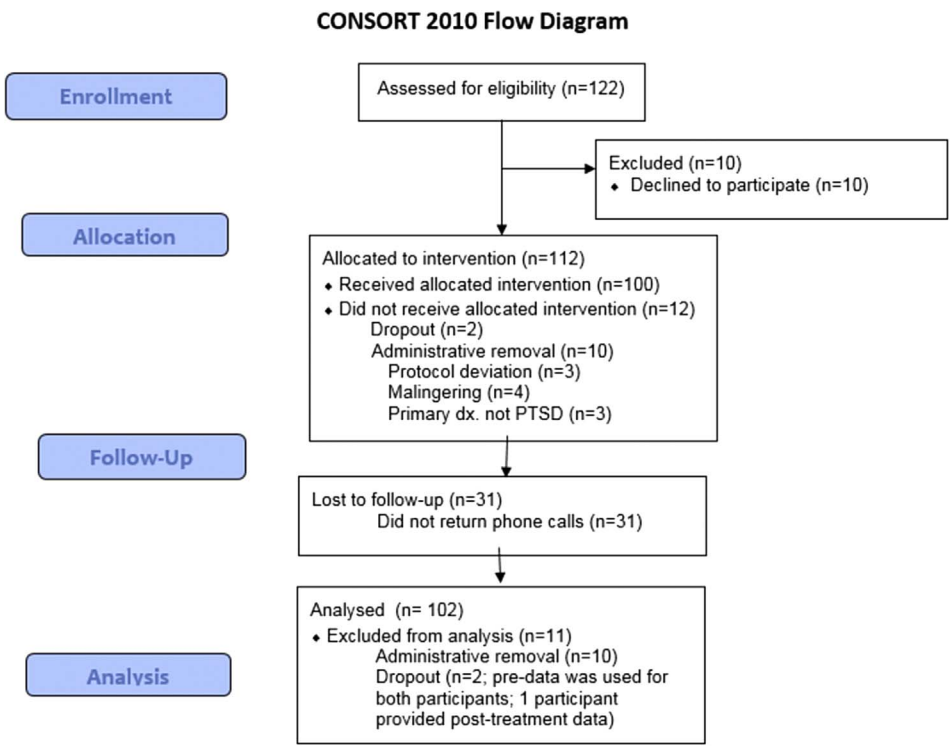

Fig. 1. 3-Week CONSORT Diagram.

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.

3.3. Trauma-related guilt inventory (TRGI)

The TRGI (Kubany et al., 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 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 et al., 2005; Oktedalen et al., 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.

3.4. Structured clinical interview for DSM-IV axis I disorders (SCID-I)

The SCID-I (First, Gibbon, Spitzer, Williams, & Benjamin, 1997) 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.

3.5. 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).

3.6. Statistical strategy

Linear mixed-effects regression (LMER) was used to explore reductions in guilt symptoms over the course of the 3-week and 17-week treatment trials. 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, & Gramlich, 2015) 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. Repeated measures ANOVAs also 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 also accounts for both within (random effects) and between person (fixed effects) variance and has been shown to function well in smaller samples with multiple observations (Muth et al., 2016).

4. Results

4.1. 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 et al., 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 < 0.001) as did the CAPS item related to "survivors guilt" (Mdn_D = 4.5, V = 62, p < 0.01). This finding is corroborated by

Table 2
Pre-post guilt subscale differences.

Measure	Pre-Treatment			Post-Treatment		
	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.
$p < 0.05$*.
$p < 0.01$**.
$p < 0.001$***.

a more comprehensive guilt measure as the participants TRGI-GG scores also significantly decreased from pre to post treatment ($Mdn_D = 1.3$, $V = 117.5$, $p < 0.001$) (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 et al., 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 ($\beta = -0.296$, $SE = 0.051$, $p < 0.001$, $r^2 = 0.72$), Guilt Cognitions ($\beta = -0.141$, $SE = 0.034$, $p < 0.001$, $r^2 = 0.77$), and Distress ($\beta = -0.304$, $SE = 0.045$, $p < 0.001$, $r^2 = 0.60$) subscales; each score decreased over time.

To further explore the independent effects of exposure session on guilt symptoms, 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 ($t = -5.08$, $p < 0.001$, $r^2 = 0.76$) also significantly predicted the TRGI GG score and was associated with a similar cumulative reduction over time ($\beta_{3\text{-week}} = -1.2$; $\beta_{17\text{-week}} = -0.93$).

4.2. Session eight guilt intervention

Prior to considering the impact of guilt on treatment outcome, 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 the 3-week treatment. A guilt intervention variable was created that consisted of a 0 at each time point prior to the guilt intervention and a 1 for each time point after the guilt intervention. Although the reduction in guilt over the course of treatment remained significant over time ($t = 7.11$, $p < 0.01$), 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 ($t = 0.67$, $p = 0.51$) did not significantly predict the TRGI-GG subscale.

4.3. Effects of guilt on treatment outcome

To assess the effects of guilt on the overall treatment outcome a median split (CAPS- Acts of commission and omission 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 ($t = -0.286$, $p = 0.78$). 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, $p = 0.52$) or CAPS survivors guilt item (t (16) = 0.117, $p = 0.91$).

Table 3
LMER analyses: Guilt predicting PCL trajectory.

Measure	β	SE	t
PCL-M (n = 102)			
Random Effects	Estimate	SD	
σ^2	82.716	9.095	
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 (n = 42)			
Random Effects			
σ^2	45.83	6.77	
Fixed Effects			
Intercept	55.890	3.72	15.02***
Time	−5.423	0.830	−6.67***
TRGI-GG	4.423	1.04	4.247***
AIC	1024.373		
BIC	1038.711		
Marginal R^2	0.36		
Conditional R^2	0.58		
PCL-M (n = 42)			
Random Effects			
Time	7.50	2.74	
Fixed Effects			
Intercept	64.25	2.51	25.56***
Time	- 5.55	0.98	−5.65***
Guilt Group	11.20	4.01	2.79**
Guilt Group x Time	−4.09	1.56	−2.63**
AIC	1182.28		
BIC	1206.42		
Marginal R^2	0.19		
Conditional R^2	0.80		

P < 0.05*.
P < 0.01**.
P < 0.001***.

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 (See Table 3). 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 ($t = -16.059$, $p < 0.001$, $r^2 = 0.66$) 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 (See Table 3). Time ($t = -5.65$, $p < 0.001$), guilt group ($t = 2.79$. $p < 0.01$), and the interaction term ($t = -2.63$, $p < 0.01$) 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 ($\beta = 11.20$, $p < 0.001$) and improve faster over the course of treatment than individuals in the low guilt group ($\beta = -4.09$, $p < 0.001$).

4.4. Lagged regression models

Multiple LMER models examined 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

Table 4
3-week lagged regression results.

Measure	β	SE	t	p
TRGI-Global Guilt (n = 36)				
Random Effects	Estimate	SD		
σ^2	0.533	0.730		
Fixed Effects				
Intercept	0.124	0.538	0.231	0.818
Autocorrelation	0.670	0.066	10.078	< 0.001***
Time	−0.127	0.102	−1.247	0.218
Lagged PCL-M	0.012	0.007	1.829	0.072
AIC	231.50			
BIC	246.43			
Marginal R^2	0.617			
Conditional R^2	0.617			
PCL-M (n = 42)				
Random Effects	Estimate	SD		
σ^2	77.656	8.812		
Fixed Effects				
Intercept	36.587	8.052	4.544	< 0.001***
Autocorrelation	0.235	0.099	2.377	0.021*
Time	−2.199	1.322	−1.602	0.114
Lagged TRGI GG	1.756	1.431	1.431	0.158
AIC	802.029			
BIC	817.660			
Marginal R^2	0.180			
Conditional R^2	0.526			
TRGI- Global Guilt (n = 34)				
Random Effects	Estimate	SD		
σ^2	0.80	-0.898		
Fixed Effects				
Intercept	0.299	0.565	0.529	0.601
Autocorrelation	0.637	0.077	8.281	< 0.001***
Time	- 0.945	0.142	−0.664	0.512
Lagged Anxiety	0.094	0.042	2.209	0.036*
AIC	163.94			
BIC	176.60			
R^2	0.602			

P < 0.05*.
P < 0.01**.
P < 0.001***.

the PCL-M both time ($t = -6.67$, $p < 0.001$) and guilt ($t = 4.25$, $p < 0.001$) 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 3). 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 while controlling for the correlation between the two variables of interest.

To evaluate hypothesis 3, lagged regression analyses were conducted to examine the relationship between symptoms of PTSD and guilt over the course of treatment (See Table 4). 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 did not significantly predict the subsequent TRGI score ($t = 1.83$, $p = 0.07$) and time was no longer a significant predictor ($t = -1.25$, $p = 0.22$). For the inverse model, the lagged TRGI-GG score ($t = 1.43$, $p = 0.16$) and time ($t = -1.60$, $p = 0.11$) also did not account for the subsequent PCL-M score. These results were replicated in the 17-week data (See Table 5).

5. Treatment mechanism

5.1. General arousal

To further investigate hypothesis 3, an additional model examined if a lagged measure of general anxiety corresponded to changes in the TRGI-GG subscale over time. In this model, the lagged anxiety variable

Table 5
17-week treatment lagged regression results.

Measure	*β*	SE	t	p
TRGI-Global Guilt (n = 17)				
Random Effects	Estimate	SD		
σ^2	0.282	0.531		
Fixed Effects				
Intercept	0.037	0.358	0.103	0.919
Autocorrelation	0.637	0.067	13.038	0.000
Time	0.023	0.042	0.553	0.582
Lagged PCL	0.000	0.006	0.062	0.951
AIC	163.94			
BIC	176.60			
R^2	0.75			
PCL-M (n = 20)				
Random Effects	Estimate	SD		
σ^2	93.123	9.650		
Fixed Effects				
Intercept	18.021	5.721	3.145	0.003
Time	−1.118	0.655	−1.706	0.093
Lagged TRGI GG	1.714	1.012	1.681	0.098
AIC	600.56			
BIC	614.78			
R^2	0.56			

P < 0.05*.
P < 0.01**.
P < 0.001***.

($t = 2.21$, $p = 0.04$) significantly predicted the TRGI-GG score at the next session ($r^2 = 0.60$) and time ($t = -0.66$, $p = 0.51$) was no longer a significant predictor (See Table 4).

5.2. Avoidance

In testing hypothesis 4, an LMER model with 38 participants and 130 observations included time and an avoidance change (Pre_{PCLAvd} - $Post_{PCLAvd}$) score (See Table 6). Although the avoidance change score ($t = 0.177$, $p = 0.860$) was not a significant predictor of the TRGI-GG subscale, time and the interaction of time and avoidance change ($t = -1.988$, $p = 0.049$) significantly predicted guilt scores. In support of hypothesis 4, this analysis indicated that individuals with higher avoidance change scores experienced faster changes in guilt over the course of treatment.

6. Discussion

The purpose of this study was to examine changes in guilt symptoms over the course of 3-week and 17-week Trauma Management Therapy and identify potential mechanisms of therapeutic action. Both the structure of TMT and the design of the treatment trials provided a unique opportunity to examine changes in guilt symptoms over the course of EXP without additional confounds such as emotional

Table 6
3-week mechanisms model.

Measure	*β*	SE	t
TRGI-GG (n = 38)			
Random Effects	Estimate	SD	
σ^2	0.630	0.396	
Fixed Effects			
Intercept	2.481	0.260	9.543***
Time	−0.197	0.067	−2.935**
Avoidance Change	0.011	0.064	0.177
Avoidance Change x time	−0.034	0.017	−1.988*
AIC	357.67		
BIC	374.69		
Marginal R^2	0.084		
Conditional R^2	0.743		

processing or cognitive restructuring. TMT is based directly on flooding principles and the underlying mechanisms of exposure therapy. Unlike PE, TMT does not teach temporary coping mechanisms (e.g., breathing retraining) or conduct emotional processing after each individual session. Additionally, the TMT group modules that emphasize skill building (i.e., social reintegration, anger management, and behavioral activation) were not delivered following exposure sessions in the 17-week sample proving the opportunity to isolate the effects of exposure therapy on guilt symptoms.

Over the course of the 3-week and 17-week TMT intervention, symptoms of guilt significantly decreased from pre to post treatment. This difference appears to be clinically meaningful as our samples pre-treatment symptom guilt levels were similar to Vietnam veterans whereas post-treatment levels were similar to non-treatment seeking veterans (Kubany et al., 1996). LMER analyses provided a 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 composed of both individual and group therapy, we also examined the individual contributions of each treatment component in the 3-week sample. Although the effects of exposure therapy were confounded with the group intervention that occurred simultaneously during the 3-week treatment, results were also replicated in the 17-week data that did not include a co-occurring group intervention. These analyses revealed that there was no detectable unique effect of the one session group 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 et al., 2015; Resick et al., 2002; Zalta et al., 2014). Furthermore, when taken together the 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.

To further examine the relationship between symptoms of PTSD and guilt, lagged regression analyses were conducted. The results of these analyses suggest that symptoms of PTSD and guilt varied together rather than one set of symptoms predicting the other. This is somewhat at odds with the findings of Zalta et al. (2014) and Oktedalen et al. (2015) who found that trauma-related cognitions predicted changes in PTSD symptoms. These discrepant findings could potentially be explained by the absence of emotional processing in our treatment and that TMT focuses primarily on anxious distress in individual exposure sessions (e.g., targeting “hot spots” in session 1). This interpretation is supported by lagged regression analyses that 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. This interpretation is consistent with the extensive literature on cognitive-arousal theory (Schachter & Singer, 1962; for review see; Reisenzein, 2017) that emphasizes the cognitive interpretation of physiological arousal as essential in determining the perception of an emotional experience. Additionally, directly targeting anxious arousal or general distress may allow for greater and more efficient treatment gains (Beidel, Frueh, Neer, & Lejuez, 2017; Beidel et al., 2017) that extend beyond directly targeted mechanisms. This may also partially explain previous findings reporting reductions in symptoms like depression in exposure therapy trials (Minnen, Harned, Zoellner, & Mills. 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 et al., 2015; Zalta et al., 2014), a larger sample would allow for greater generalization to diverse trauma types and symptom presentations. Furthermore, as the sample was comprised of active duty military personnel and combat Veterans, future research may wish to explore the generalization of these findings to purely Veteran or active duty military samples.

Due to the expanding criteria for PTSD, the use of the DSM-IV criteria may have also 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 (Tangney, Stuewig, & Mashek, 2007), and did not examine fear oriented cognitions that may change differently than self-evaluative 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 effectiveness of 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.

In conclusion, the current study is the first to provide evidence that trauma-related guilt symptoms can be reduced through exposure therapy in the absence of emotional processing. 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. Although a specific component of PE is referred to as emotional processing, the resolution of trauma related emotions and cognitions is a dynamic process that likely occurs during all components of exposure therapy. It is possible that much of the therapeutic work conducted during the emotional processing component of PE occurs naturally as cognitive resources are made available in the absence of aversive physiological arousal. This study identifies the reduction of general anxiety as a potential mechanism for reducing symptoms of guilt. The reduction of this subjective aversive arousal through exposure therapy may alleviate general distress that is no longer attributed to guilt cognitions.

Funding

This study was supported by the U.S. Army Medical Research & Materiel Command-Military Operational Medicine Research Program (USAMRMC-MOMRP; contract W81XWH-11-2-0038) to the fifth and sixth author. The study had both MRCM HRPO and local IRB approval and does not necessarily reflect the policy/position of the government. The funding source had no involvement with the design, collection,

analysis or interpretation of the data, or the writing of the report or the decision to submit the article for publication.

Conflicts of interest

The authors have no conflicts of interest to disclose.

Acknowledgments

The authors greatly appreciate the effort of the entire UCF RESTORES staff including many hardworking and diligent undergraduate psychology students. Special thanks to Rachel Ruffin, Ph.D., Ashley Arens, Ph.D., Katherine Ragsdale, Ph.D., Franklin Mesa, Ph.D., Maryann Owens, Ph.D., Jeremy Stout, Benson Munyanm, Madeline Marks, Thien-An Le, Michael Gramlich, Emily Kitsmiller, Krystal Morrison, and Jennifer Scheurich for serving as individual or group therapists.

References

Aderka, I. M., Gillihan, S. J., McLean, C. P., & Foa, E. B. (2013). The relationship between posttraumatic and depressive symptoms during prolonged exposure with and without cognitive restructuring for the treatment of posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology, 81*(3), 375. http://dx.doi.org/10.1037/a0031523.

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.

Arntz, A., Tiesema, M., & Kindt, M. (2007). Treatment of PTSD: A comparison of imaginal exposure with and without imagery rescripting. *Journal of Behavior Therapy and Experimental Psychiatry, 38*(4), 345–370. http://dx.doi.org/10.1016/j.jbtep.2007.10.006.

Asnaani, A., Reddy, M. K., & Shea, M. T. (2014). The impact of PTSD symptoms on physical and mental health functioning in returning veterans. *Journal of Anxiety Disorders, 28*(3), 310–317. http://dx.doi.org/10.1016/j.janxdis.2014.01.005.

Beidel, D. C., Frueh, B. C., Neer, S. M., Bowers, C., & Rizzo, A. (2014b). Using virtual reality as part of an intensive treatment program for PTSD. *Paper presentation at interservice/industry training, simulation, and education conference (I/ITSEC). Orlando, FL*.

Beidel, D. C., Frueh, B. C., Neer, S. M., Bowers, C. A., Trachik, B., Uhde, T. W., et al. (2017). Trauma management therapy with virtual-reality augmented exposure therapy for combat-related PTSD: A randomized controlled trial. *Journal of Anxiety Disorders*.

Beidel, D. C., Frueh, B. C., Neer, S. M., & Lejuez, C. W. (2017). The efficacy of trauma management therapy: A controlled pilot investigation of a three-week intensive outpatient program for combat-related PTSD. *Journal of Anxiety Disorders, 50*, 23–32. http://dx.doi.org/10.1016/j.janxdis.2017.05.001.

Benish, S. G., Imel, Z. E., & Wampold, B. E. (2008). The relative efficacy of bona fide psychotherapies for treating post-traumatic stress disorder: A meta-analysis of direct comparisons. *Clinical Psychology Review, 28*(5), 746–758.

Blake, D., Weathers, F., Nagy, L., Kaloupek, D., Klauminzer, G., Charney, D., et al. (1990). Clinician-administered PTSD scale (CAPS). *Boston (Mass), 7*.

Bradley, R., Greene, J., Russ, E., Dutra, L., & Westen, D. (2005). A multidimensional meta-analysis of psychotherapy for PTSD. *American Journal of Psychiatry, 162*, 214–227. http://dx.doi.org/10.1176/appi.ajp.162.2.214.

Bryan, C. J., Griffith, J. H., Pace, B. T., Hinkson, K., Bryan, A. O., Clemans, T. a., et al. (2015). Combat exposure and risk for suicidal thoughts and behaviors among military personnel and veterans: A systematic review and meta-analysis. *Suicide and Life-threatening Behavior*. http://dx.doi.org/10.1111/sltb.12163.

Bryan, C. J., Ray-Sannerud, B., Morrow, C. E., & Etienne, N. (2013). Guilt is more strongly associated with suicidal ideation among military personnel with direct combat exposure. *Journal of Affective Disorders, 148*(1), 37–41. http://dx.doi.org/10.1016/j.jad.2012.11.044.

Currier, J. M., Holland, J. M., & Drescher, K. D. (2014). Residential treatment for combat-related posttraumatic stress disorder: Identifying trajectories of change and predictors of treatment Response. *PLoS ONE, 9*(7), e101741. http://dx.doi.org/10.1371/journal.pone.0101741.

Dalgleish, T. (2004). Cognitive approaches to posttraumatic stress disorder: The evolution of multirepresentational theorizing. *Psychological Bulletin, 130*(2), 228–260. http://dx.doi.org/10.1037/0033-2909.130.2.228.

Ehlers, A., & Steil, R. (1995). Maintenance of intrusive memories in posttraumatic stress disorder: A cognitive approach. *Behavioural Cognitive Psychotherapy, 23*(3), 217–249. http://dx.doi.org/10.1017/S135246580001585X.

First, M. B., Gibbon, M., Spitzer, R. L., Williams, J. B. W., & Benjamin, L. S. (1997). *User's guide for the structured clinical interview for DSM-IV Axis II personality disorders: SCID-II*.

Foa, E. B., Ehlers, A., Clark, D. M., Tolin, D. F., & Orsillo, S. M. (1999). The posttraumatic cognitions inventory (PTCI): Development and validation. *Psychological Assessment, 11*(3), 303–314. http://dx.doi.org/10.1037/1040-3590.11.3.303.

Foa, E. B., Hembree, E. A., Cahill, S. P., Rauch, S. A., Riggs, D. S., Feeny, N. C., et al. (2005). Randomized trial of prolonged exposure for posttraumatic stress disorder with and without cognitive restructuring: Outcome at academic and community clinics. *Journal of Consulting and Clinical Psychology, 73*(5), 953.

Foa, E. B., Keane, T. M., Friedman, M. J., & Cohen, J. A. (2008). *Effective treatments for PTSD: Practice guidelines from the international society for traumatic stress studies*. Guilford Press.

Foa, E. B., & Rauch, S. M. (2004). Cognitive changes during prolonged exposure versus prolonged exposure plus cognitive restructuring in female assault survivors with posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology, 72*(5), 879–884. http://dx.doi.org/10.1037/0022-006X.72.5.879.

Frayne, S. M., Chiu, V. Y., Iqbal, S., Berg, E. A., Laungani, K. J., Cronkite, R. C., et al. (2011). Medical care needs of returning veterans with PTSD: Their other burden. *Journal of General Internal Medicine, 26*(1), 33–39. http://dx.doi.org/10.1007/s11606-010-1497-4.

Friedman, M. J. (2013). Finalizing PTSD in DSM-5: Getting here from there and where to go next. *Journal of Traumatic Stress, 26*(5), 548–556. http://dx.doi.org/10.1002/jts.21840.

Frueh, B. C., Turner, S. M., & Beidel, D. C. (1995). Exposure therapy for combat-related PTSD: A critical review. *Clinical Psychology Review, 15*, 799–817. http://dx.doi.org/10.1016/0272-7358(95)00049-6.

Frueh, B. C., Turner, S. M., Beidel, D. C., Mirabella, R. F., & Jones, W. J. (1996). Trauma management therapy: A preliminary evaluation of a multicomponent behavioral treatment for chronic combat-related PTSD. *Behaviour Research and Therapy, 34*(7), 533–543.

Gates, M. A., Holowka, D. W., Vasterling, J. J., Keane, T. M., Marx, B. P., & Rosen, R. C. (2012). Posttraumatic stress disorder in veterans and military personnel: Epidemiology, screening, and case recognition. *Psychological Services, 9*(4), 361–382. http://dx.doi.org/10.1037/a0027649.

Gros, D. F., Yoder, M., Tuerk, P. W., Lozano, B. E., & Acierno, R. (2011). Exposure therapy for PTSD delivered to veterans via telehealth: Predictors of treatment completion and outcome and comparison to treatment delivered in person. *Behavior Therapy, 42*(2), 276–283. http://dx.doi.org/10.1016/j.beth.2010.07.005.

Grunert, B. K., Smucker, M. R., Weis, J. M., & Rusch, M. D. (2003). When prolonged exposure fails: Adding an imagery-based cognitive restructuring component in the treatment of industrial accident victims suffering from PTSD. *Cognitive and Behavioral Practice, 10*(4), 333–346. http://dx.doi.org/10.1016/S1077-7229(03)80051-2.

Grunert, B. K., Weis, J. M., Smucker, M. R., & Christianson, H. F. (2007). Imagery rescripting and reprocessing therapy after failed prolonged exposure for post-traumatic stress disorder following industrial injury. *Journal of Behavior Therapy and Experimental Psychiatry, 38*(4), 317–328. http://dx.doi.org/10.1016/j.jbtep.2007.10.005.

Haley, S. A. (1974). When the patient reports atrocities: Specific treatment considerations of the Vietnam veteran. *Archives of General Psychiatry, 30*(2), 191–196.

Harman, R., & Lee, D. (2010). The role of shame and self-critical thinking in the development and maintenance of current threat in post-traumatic stress disorder. *Clinical Psychology & Psychotherapy, 17*(1), 13–24. http://dx.doi.org/10.1002/cpp.636.

Held, P., Owens, G. P., Schumm, J. A., Chard, K. M., & Hansel, J. E. (2011). Disengagement coping as a mediator between trauma-related guilt and PTSD severity. *Journal of Traumatic Stress, 24*(6), 708–715. http://dx.doi.org/10.1002/jts.20689.

Hendin, H., & Haas, A. P. (1991). Suicide and guilt as manifestations of PTSD in Vietnam combat veterans. *The American Journal of Psychiatry, 148*(5), 586–591.

Henning, K. R., & Frueh, B. C. (1997). Combat guilt and its relationship to PTSD symptoms. *Journal of Clinical Psychology, 53*, 801–808.

Hofmann, S. G. (2008). Cognitive processes during fear acquisition and extinction in animals and humans: Implications for exposure therapy of anxiety disorders. *Clinical Psychology Review, 28*(2), 199–210. http://dx.doi.org/10.1016/j.cpr.2007.04.009.

Hoge, C. W., Castro, C. A., Messer, S. C., McGurk, D., Cotting, D. I., & Koffman, R. L. (2004). Combat duty in Iraq and Afghanistan, mental health problems, and barriers to care. *New England Journal of Medicine, 351*(1), 13–22.

Kubany, E. S., Haynes, S. N., Abueg, F. R., Manke, F. P., Brennan, J. M., & Stahura, C. (1996). Development and validation of the trauma-related guilt inventory (TRGI). *Psychological Assessment, 8*(4), 428–444.

Kubany, E. S., & Watson, S. B. (2003). Guilt: Elaboration of a multidimensional model. *The Psychological Record, 53*(1), 51.

LeDoux, J. (1996). Emotional networks and motor control: A fearful view. *Progress in Brain Research, 107*, 437–446.

Litz, B. T., Stein, N., Delaney, E., Lebowitz, L., Nash, W. P., Silva, C., et al. (2009). Moral injury and moral repair in war veterans: A preliminary model and intervention strategy. *Clinical Psychology Review, 29*(8), 695–706. http://dx.doi.org/10.1016/j.cpr.2009.07.003.

Lovibond, P. F. (2004). Cognitive processes in extinction. *Learning & Memory, 11*(5), 495–500. http://dx.doi.org/10.1101/lm.79604.

Maguen, S., Madden, E., Cohen, B. E., Bertenthal, D., & Seal, K. H. (2012). Time to treatment among veterans of conflicts in Iraq and Afghanistan with psychiatric diagnoses. *Psychiatric Services, 63*(12), 17–24. http://dx.doi.org/10.1176/appi/ps.201200051.

McLay, R. N., Wood, D. P., Webb-Murphy, J. A., Spira, J. L., Wiederhold, M. D., Pyne, J. M., et al. (2011). A randomized, controlled trial of virtual reality-graded exposure therapy for post-traumatic stress disorder in active duty service members with combat-related post-traumatic stress disorder. *Cyberpsychology, Behavior, and Social Networking, 14*(4), 223–229. http://dx.doi.org/10.1089/cyber.2011.0003.

McLean, C. P., Yeh, R., Rosenfield, D., & Foa, E. B. (2015). Changes in negative cognitions mediate PTSD symptom reductions during client-centered therapy and prolonged exposure for adolescents. *Behaviour Research and Therapy, 68*, 64–69. http://dx.doi.org/10.106/j.brat2015.03.008.

Meiser-Stedman, R., Dalgleish, T., Glucksman, E., Yule, W., & Smith, P. (2009). Maladaptive cognitive appraisals mediate the evolution of posttraumatic stress reactions: A 6-month follow-up of child and adolescent assault and motor vehicle accident survivors. *Journal of Abnormal Psychology, 118*(4), 778–787. http://dx.doi.org/

10.1037/a0016945.
Minnen, A. V., Arntz, A., & Keijsers, G. P. J. (2002). Prolonged exposure in patients with chronic PTSD: Predictors of treatment outcome and dropout. *Behaviour Research and Therapy, 40*, 439–457.
Minnen, A. V., Harned, M. S., Zoellner, L., & Mills, K. (2012). Examining potential contraindications for prolonged exposure therapy for PTSD. *European Journal of Psychotraumatology, 3*, 1–14. http://dx.doi.org/10.3402/ejpt.v3i0.18805.
Moser, J. S., Cahill, S. P., & Foa, E. B. (2010). Evidence for poorer outcome in patients with severe negative trauma-related cognitions receiving prolonged exposure plus cognitive restructuring: Implications for treatment matching in posttraumatic stress disorder. *The Journal of Nervous and Mental Disease, 198*(1), 72–75. http://dx.doi.org/10.1097/NMD.0b013e3181c81fac.
Moser, J. S., Hajcak, G., Simons, R. F., & Foa, E. B. (2007). Posttraumatic stress disorder symptoms in trauma-exposed college students: The role of trauma-related cognitions, gender, and negative affect. *Journal of Anxiety Disorders, 21*(8), 1039–1049. http://dx.doi.org/10.1016/j.janxdis.2006.10.009.
Munyan, B. G., Neer, S. M., Beidel, D. C., & Gramlich, M. A. (April, 2015). How many sessions are Enough? Exposure therapy with OEF/OIF veterans with combat-related posttraumatic stress disorder. *Poster presented at the meeting of the anxiety and depression association of America, Miami, Florida*.
Muth, C., Bales, K. L., Hinde, K., Maninger, N., Mendoza, S. P., & Ferrer, E. (2016). Alternative models for small samples in psychological research: Applying linear mixed effects models and generalized estimating equations to repeated measures data. *Educational and Psychological Measurement, 76*(1), 64–87. http://dx.doi.org/10.1177/0013164415580432.
Nishith, P., Nixon, R. D. V., & Resick, P. A. (2005). Resolution of trauma-related guilt following treatment of PTSD in female rape victims: A result of cognitive processing therapy targeting comorbid depression? *Journal of Affective Disorders, 86*(2–3), 259–265. http://dx.doi.org/10.1016/j.jad.2005.02.013.
Oktedalen, T., Hoffart, A., & Langkaas, T. F. (2015). Trauma-related shame and guilt as time-varying predictors of posttraumatic stress disorder symptoms during imagery exposure and imagery rescripting-A randomized controlled trial. *Psychotherapy Research: Journal of the Society for Psychotherapy Research, 25*(5), 1–15. http://dx.doi.org/10.1080/10503307.2014.917217.
Owens, G. P., Chard, K. M., & Ann Cox, T. (2008). The relationship between maladaptive cognitions, anger expression, and posttraumatic stress disorder among veterans in residential treatment. *Journal of Aggression, Maltreatment & Trauma, 17*(4), 439–452. http://dx.doi.org/10.1080/10926770802473908.
Phelps, E. A., Delgado, M. R., Nearing, K. I., & LeDoux, J. E. (2004). Extinction learning in humans: Role of the amygdala and vmPFC. *Neuron, 43*(6), 897–905. http://dx.doi.org/10.1016/j.neuron.2004.08.042.
Power, M. J., & Fyvie, C. (2013). The role of emotion in PTSD: Two preliminary studies. *Behavioural and Cognitive Psychotherapy, 41*(2), 162–172. http://dx.doi.org/10.1017/S1352465812000148.
Powers, M. B., Halpern, J. M., Ferenschak, M. P., Gillihan, S. J., & Foa, E. B. (2010). A meta-analytic review of prolonged exposure for posttraumatic stress disorder. *Clinical Psychology Review, 30*(6), 635–641. http://dx.doi.org/10.1016/j.cpr.2010.04.007.
Pugh, L. R., Taylor, P. J., & Berry, K. (2015). The role of guilt in the development of posttraumatic stress disorder: A systematic review. *Journal of Affective Disorders, 182*, 138–150. http://dx.doi.org/10.1016/j.jad.2015.04.026.
Rauch, S. M., Smith, E., Duax, J., & Tuerk, P. (2013). A data-driven perspective: Response to commentaries by Maguen and Burkman (2013) and Steenkamp et al. (2013). *Cognitive and Behavioral Practice, 20*(4), 480–484. http://dx.doi.org/10.1016/j.cbpra.2013.07.002.
Reger, G. M., Holloway, K. M., Candy, C., Rothbaum, B. O., Difede, J., Rizzo, A. A., et al. (2011). Effectiveness of virtual reality exposure therapy for active duty soldiers in a military mental health clinic. *Journal of Traumatic Stress, 24*(1), 93–96. http://dx.doi.org/10.1002/jts.20574.
Reisenzein, R. (2017). The legacy of cognition-arousal Theory: Introduction to a special section of emotion review. *Emotion Review, 9*(1), 3–6. http://dx.doi.org/10.1177/175407391666255.
Repa, J. C., Muller, J., Apergis, J., Desrochers, T. M., Zhou, Y., & LeDoux, J. E. (2001). Two different lateral amygdala cell populations contribute to the initiation and storage of memory. *Nature Neuroscience, 4*(7), 724–731. http://dx.doi.org/10.1038/89512.
Resick, P. A., Nishith, P., Weaver, T. L., Astin, M. C., & Feuer, C. A. (2002). A comparison of cognitive-processing therapy with prolonged exposure and a waiting condition for the treatment of chronic posttraumatic stress disorder in female rape victims. *Journal of Consulting and Clinical Psychology, 70*(4), 867–879. http://dx.doi.org/10.1037/0022-006x.70.4.867.
Resick, P. A., & Schnicke, M. K. (1992). Cognitive processing therapy for sexual assault victims. *Journal of Consulting and Clinical Psychology, 60*, 748–756.
Richardson, L. K., Frueh, B. C., & Acierno, R. (2010). Prevalence estimates of combat-related posttraumatic stress disorder: Critical review. *Australian and New Zealand Journal of Psychiatry, 44*, 4–19. http://dx.doi.org/10.3109/00048670903393597.
Rivers, W. H. (1918). The repression of war experience. *Proceedings of the Royal Society of Medicine, 11*(Sect Psych), 1–20.
Roberts, N. P., Kitchiner, N. J., Kenardy, J., & Bisson, J. I. (2009). Systematic review and meta-analysis of multiple-session early interventions following traumatic events. *The American Journal of Psychiatry, 166*(3), 293–301. http://dx.doi.org/10.1176/appi.ajp.2008.08040590.
Rosenheck, R. A., & Fontana, A. F. (2007). Recent trends in VA treatment of post-traumatic stress disorder and other mental disorders. *Health Affairs, 26*(6), 1720–1727. http://dx.doi.org/10.1377/hlthaff.26.6.1720.
Rothbaum, B. O., Price, M., Jovanovic, T., Norrholm, S. D., Gerardi, M., Dunlop, B., et al. (2014). A Randomized, double-blind evaluation of d-cycloserine or alprazolam combined with virtual reality exposure therapy for posttraumatic stress disorder in Iraq and Afghanistan war veterans. *American Journal of Psychiatry, 171*(6), 640–648. http://dx.doi.org/10.1176/appi.ajp.2014.13121625.
Schachter, S., & Singer, J. (1962). Cognitive, social, and physiological determinants of emotional state. *Psychological Review, 69*(5), 379. http://dx.doi.org/10.1037/h0046234.
Shively, S. B., & Perl, D. P. (2012). Traumatic brain injury, shell shock, and posttraumatic stress disorder in the military—past, present, and future. *The Journal of Head Trauma Rehabilitation, 27*(3), 234–239. http://dx.doi.org/10.1097/HTR.0b013e318250e9dd.
Smith, E. R., Duax, J. M., & Rauch, S. M. (2013). Perceived perpetration during traumatic events: Clinical suggestions from experts in prolonged exposure therapy. *Cognitive and Behavioral Practice, 20*(4), 461–470. http://dx.doi.org/10.1016/j.cbpra.2012.12.002.
Smucker, M. R., & Dancu, C. V. (1999). *Cognitive-behavioral treatment for adult survivors of childhood trauma: Imagery rescripting and reprocessing.* Lanhan, MD, US: Jason Aronson.
Stapleton, J. A., Taylor, S., & Asmundson, G. J. G. (2006). Effects of three PTSD treatments on anger and guilt: Exposure therapy, eye movement desensitization and reprocessing, and relaxation training. *Journal of Traumatic Stress, 19*(1), 19–28. http://dx.doi.org/10.1002/jts.20095.
Steenkamp, M. M., Litz, B. T., Hoge, C. W., & Marmar, C. R. (2015). Psychotherapy for military-related PTSD. *JAMA, 314*(5), 489–500. http://dx.doi.org/10.1001/jama.2015.8370.
Steenkamp, M. M., Nash, W. P., Lebowitz, L., & Litz, B. T. (2013). How best to treat deployment-related guilt and shame: Commentary on Smith, Duax, and Rauch (2013). *Cognitive and Behavioral Practice, 20*, 471–475. http://dx.doi.org/10.1016/j.cbpra.2013.05.002.
Stein, N. R., Mills, M. A., Arditte, K., Mendoza, C., Borah, A. M., Resick, P. A., et al. (2012). A scheme for categorizing traumatic military events. *Behavior Modification, 36*(6), 787–807. http://dx.doi.org/10.1177/0145445512446945.
Strachan, M., Gros, D. F., Ruggiero, K. J., Lejuez, C. W., & Acierno, R. (2012). An integrated approach to delivering exposure-based treatment for symptoms of PTSD and depression in OIF/OEF veterans: Preliminary findings. *Behavior Therapy, 43*(3), 560–569. http://dx.doi.org/10.1016/j.beth.2011.03.003.
Street, A. E., Gibson, L. E., & Holohan, D. R. (2005). Impact of childhood traumatic events, trauma-related guilt, and avoidant coping strategies on PTSD symptoms in female survivors of domestic violence. *Journal of Traumatic Stress, 18*(3), 245–252. http://dx.doi.org/10.1002/jts.20026.
Tangney, J. P., Stuewig, J., & Mashek, D. J. (2007). Moral emotions and moral behavior. *Annual Review of Psychology, 58*, 345–372. http://dx.doi.org/10.1146/annurev.psych.56.091103.070145.
Tanielian, T. L. (2008). *Invisible wounds of war: Summary and recommendations for addressing psychological and cognitive injuries.* Santa Monica, CA: RAND, Center for Military Health Policy Research.
Tarrier, N., Pilgrim, H., Sommerfield, C., Faragher, B., Reynolds, M., Graham, E., et al. (1999). A randomized trial of cognitive therapy and imaginal exposure in the treatment of chronic posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology, 67*(1), 13–18.
Tilghman-Osborne, C., Cole, D. A., & Felton, J. W. (2010). Definition and measurement of guilt: Implications for clinical research and practice. *Clinical Psychology Review, 30*(5), 536–546. http://dx.doi.org/10.1016/j.cpr.2010.03.007.
Tuerk, P. W., Wangelin, B., Rauch, S. A., Dismuke, C. E., Yoder, M., Myrick, H., et al. (2013). Health service utilization before and after evidence-based treatment for PTSD. *Psychological Services, 10*(4), 401. http://dx.doi.org/10.1037/a0030549.
Tuerk, P. W., Yoder, M., Grubaugh, A., Myrick, H., Hamner, M., & Acierno, R. (2011). Prolonged exposure therapy for combat-related posttraumatic stress disorder: An examination of treatment effectiveness for veterans of the wars in Afghanistan and Iraq. *Journal of Anxiety Disorders, 25*(3), 397–403. http://dx.doi.org/10.1016/j.janxdis.2010.11.002.
Tuerk, P. W., Yoder, M., Ruggiero, K. J., Gros, D. F., & Acierno, R. (2010). A pilot study of prolonged exposure therapy for posttraumatic stress disorder delivered via telehealth technology. *Journal of Traumatic Stress, 23*(1), 116–123. http://dx.doi.org/10.1002/jts.20494.
Vasterling, J. J., Verfaellie, M., & Sullivan, K. D. (2009). Mild traumatic brain injury and posttraumatic stress disorder in returning veterans: Perspectives from cognitive neuroscience. *Clinical Psychology Review, 29*(8), 674–684. http://dx.doi.org/10.1016/j.cpr.2009.08.004.
Watkins, K. E., Pincus, H. A., Paddock, S., Smith, B., Woodroffe, A., Farmer, C., et al. (2011). Care for veterans with mental and substance use disorders: Good performance, but room to improve on many measures. *Health Affairs,* 10–1377. http://dx.doi.org/10.1377/hlthaff.2011.0509.
Weathers, F. W., & Litz, B. (1994). Psychometric properties of the clinician-administered PTSD scale, CAPS-1. *PTSD Research Quarterly, 5*, 2–6.
Weathers, F., Litz, B., Huska, J., & Keane, T. (1994). PTSD Checklist-Military version. In P. T. S. D. NCf (Ed.). Boston: Behavioral sciences division 1994.
Zalta, A. K., Gillihan, S. J., Fisher, A. J., Mintz, J., McLean, C. P., Yehuda, R., et al. (2014). Change in negative cognitions associated with PTSD predicts symptom reduction in prolonged exposure. *Journal of Consulting and Clinical Psychology, 1*, 171–175. http://dx.doi.org/10.1037/a0034735.