Combat-related guilt and the mechanisms of exposure therapy

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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
treatment non-responders appears to be larger in military and veteran samples (Steenkamp, Litz, & 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; McLaey et al., 2011; Reger et al., 2011; Strachan, Gros, Ruggiero, Lejuez, & Acierno, 2012; Tuerk, Yoder, Ruggiero, Gros, & Acierno, 2016; 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, & Langkass, 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 treatments 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 (Terrriec 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 re-structuring have either found no improvement over and above
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