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Improving human reactivity to trauma exposure using affect labeling

A dissertation submitted in partial satisfaction of the requirements for the degree Doctor of
Philosophy in Psychology

by

Lily Anna Brown

2016

ABSTRACT OF THE DISSERTATION

Improving human reactivity to trauma exposure using affect labeling

by

Lily Anna Brown

Doctor of Philosophy in Clinical Psychology and Learning and Behavior Psychology

University of California, Los Angeles, 2016

Professor Michelle G. Craske, Chair

Background: Posttraumatic Stress Disorder (PTSD) is of tremendous public health significance given high prevalence rates, chronicity, and resulting functional impairment. While a number of empirically-supported treatments have been developed, these treatments are not widely available, nor are they universally efficacious. The current studies translate a traditional assessment technique, Script-Driven Imagery, into a computerized training for individuals with elevated trauma reactivity. This imaginal-exposure based training was supplemented with Affect Labeling to determine whether inhibitory learning was enhanced with this augmentation strategy.

Methods: Participants ($n=107$) were college students and community members who were recruited for two studies. The first compared augmentation Task (Affect Labeling vs. a control task, Shape Labeling) and Order of Task (During vs. After labeling) on physiological and self-report outcomes at Pre- and Post-Training. The second compared Condition (Affect Labeling vs. a control active task, Distraction Labeling vs. a control inactive task, Exposure Only) on Pre- to Post-Training changes in physiological and self-report measures.

Results: The trainings provided in both studies were effective at reducing self-reported distress and physiological activation from Pre- to Post-Training, though there were no consistent

differences based on Condition. There was some evidence that Labeling (including Affect and Distract Labeling) conferred a benefit over No Labeling.

Conclusions: This study provides initial support for the acceptability and efficacy of this independently-operated computerized training for PTSD. Clinical implications and future directions are discussed.

The dissertation of Lily Anna Brown is approved.

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The Effect of Affect Labeling on Script-Driven Imagery for Trauma Exposure

Posttraumatic Stress Disorder (PTSD) is a chronic and impairing disorder that affects up to 12% of the general population (Breslau, Chilcoat, Kessler, & Davis, 1999), some of whom suffer with their symptoms for upwards of 50 years (Gold et al., 2000). Exposure therapy is an efficacious treatment for many individuals with PTSD, yet a substantial number of people remain symptomatic or experience a return of their symptoms after treatment (Bradley, Greene, Russ, Dutra, & Westen, 2005; Hofmann & Smits, 2008). The studies described herein examined the combined effect of Affect Labeling, or labeling ones emotions, and script-driven imagery (SDI), an imaginal exposure protocol, as a training for individuals who have been exposed to trauma. The first major goal of this research was to translate the Script-Driven Imagery (SDI) procedure from an assessment tool to a brief training for individuals with elevated reactivity following trauma exposure. The second goal was to investigate Affect Labeling as a supplement to the SDI training for trauma-exposed individuals. The outcome of these studies could have important implications for treatment development and dissemination for individuals with PTSD.

Human Reactivity to Trauma

Posttraumatic stress disorder (PTSD) can emerge following an individual's witnessing or experiencing a traumatic event. It is characterized by symptoms of avoidance, re-experiencing, negative cognitions, and hyperarousal (American Psychiatric Association, 2013). Sufferers with PTSD frequently report a "biphasic reliving and denial" (Davidson, 1997, p. 3) pattern of responding, driven by problematic shifts between hypervigilance and avoidance. Around 50% of those with PTSD report avoidance of both internal and external stimuli (see Foa, Zinbarg, & Rothbaum, 1992 for a review).

When compared to healthy controls, those with PTSD demonstrate elevated tonic physiological levels, including heart rate, blood pressure and startle response, and higher physiological, analgesic, and endocrine responding to trauma relevant stimuli (Butler et al., 1990; Foa et al., 1992). Chronic autonomic stimulation may contribute to reduced physiological responding to trauma triggers relative to non-PTSD groups that is sometimes reported (Cohen et al., 1998, 2000), though baseline differences in the studied samples make these comparisons challenging (Casada, Amdur, Larsen, & Liberzon, 1998). The majority of the available literature suggests that not only are those with PTSD operating at an elevated resting baseline physiology, but they also experience greater elevations in physiology to trauma cues, have difficulty with returning to baseline physiology upon encountering a trauma-relevant trigger (Beckham et al., 2000; Beckham et al., 2002; Kibler & Lyons, 2004), and demonstrate reduced physiological flexibility (as indexed by heart rate variability) to triggers (Keary, Hughes, & Palmien, 2009). These disturbances are directly proportional to disorder severity, an association that might be mediated by perceived coping ability (Kibler & Lyons, 2004). Thus, trauma exposure can impact both physiological and psychological health.

The prevalence of PTSD is estimated at 9-12% in the general population (Breslau et al., 1999; Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993) with a female to male ratio of 2:1 (Breslau et al., 1999; Frans, Rimmö, Åberg, & Fredrikson, 2005; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). In terms of trauma prevalence rates, the National Comorbidity Study found that 60.7% of men and 51.2% of women have experienced a traumatic event in their lifetime (Kessler et al., 1995). The most commonly reported traumatic events include witnessing someone badly injured or killed, involvement in a natural disaster, fire, life-threatening accident, or robbery, and sudden death of a loved one (Kessler et al., 1995; Norris, 1992; Resnick et al., 1993). While symptoms of PTSD are very common immediately following trauma exposure, a

reduction in symptoms typically occurs by three months post-trauma exposure (Riggs, Rothbaum, & Foa, 1995). However, up to 57% of rape victims and 27% of physical assault victims go on to develop PTSD and related functional impairment (Kilpatrick, Saunders, Veronen, Best, & Von, 1987).

Given the high rate of trauma exposure, it is important to delineate the factors that constitute the greatest risk of developing PTSD. Some studies have found that the severity of the traumatic event is a strong predictor of PTSD severity (see March, 1993 for a review), though this is not a universal finding (Shalev, Peri, Canetti, & Schreiber, 1996). Individuals exposed to combat, rape, or assault have some of the highest probabilities of developing PTSD (Breslau et al., 1999; Kessler et al., 1995), though disasters, accidents, loss, and non-malignant diseases are particularly risky events for women (Ditlevsen & Elklit, 2012). Furthermore, experiencing multiple traumatic events increases the likelihood of PTSD upon subsequent trauma exposure, particularly if the traumas are assaultive in nature and occur in childhood (Breslau et al., 1999). External secondary stressors may confer greater risk to excessive trauma reactivity (Pynoos, Steinberg, & Piacentini, 1999). In addition, uncontrollable and unpredictable events have a greater chance of resulting in PTSD, such that traumatic events occurring in a previously deemed safe environment could evoke a stronger reaction (Foa et al., 1992). Therefore, type of trauma, trauma severity, and the unpredictability of the trauma may be important risk factors.

Whereas early reports suggested that physical injury protected against PTSD development compared to trauma without physical injury (Merbaum & Hefez, 1975), particularly due to the validation and environmental support following physical injuries that are often absent following psychological stress, a robust empirical literature base counters this argument. Sustaining physical injury confers an eight-fold risk to the development of PTSD (Koren, Norman, Cohen, Berman, & Klein, 2005). Further, the impact of physical injury and

other complications resulting from trauma is greater in those with PTSD. The presence of PTSD contributes to worsened disease progression of human immunodeficiency virus (HIV), which is particularly relevant for survivors of sexual assault (see Ironson et al., 2013 for a review). PTSD status, as compared to injury severity, accounts for a greater proportion of variance in general health functioning at 6 months post-neurological injury (Michaels et al., 1999). Therefore, injury severity is a prospective predictor of PTSD status, and PTSD status is a prospective predictor of overall physical health and functioning.

In addition to the psychological and physical suffering associated with PTSD, the disorder also carries substantial economic burden. Those with PTSD are disproportionate consumers of the health care industry (Golding, Stein, Siegel, Burnam, & Sorenson, 1988; Kimerling & Calhoun, 1994), likely due in part to the chronicity of the disorder. Up to one-third of patients with PTSD continue to experience symptoms 10 (Kessler et al., 1995) to 50 years post-trauma exposure (Gold et al., 2000). Compared to women without trauma exposure, women with severe traumas commonly incur doubled health care expenses (Koss, Koss, & Woodruff, 1991). This is not reflective of an overreliance on only mental health services, as only 12% of recent crime victims receive therapy or psychopharmacology (Norris, Kaniasty, & Scheer, 1990). Therefore, there is a justification for investment in accessible and effective interventions to offset the psychological, physical, and financial impact of PTSD.

Fear Conditioning in PTSD

Fear conditioning models provide a useful understanding of the development and maintenance of PTSD (Michael, Ehlers, & Halligan, 2005; Pitman, 1989; Pitman, Orr, & Shalev, 1993). In short, non-biologically significant stimuli (conditioned stimuli, CS; including memories of the event) are paired with biologically threatening stimuli (unconditioned stimuli; US) that cause significant physical or emotional pain (unconditioned response; UR), forming an excitatory CS-US association. Subsequently, fear and avoidance of trauma triggers (conditioned responses; CRs) develop. Reductions in CR may be achieved through presentation of the CS in the absence of the US, the experimental analogue of exposure therapy, allowing for the formation of an inhibitory (CS-no US) association (Bouton, 2004; Craske, Liao, Brown, & Vervliet, 2012). Long-term PTSD maintenance is presumed due in part to continued avoidance of the CS, in particular trauma memories (Foa et al., 1992), preventing the formation of an inhibitory association.

However, several factors lead to deficits in the development and retention of inhibitory learning following trauma exposure. Firstly, generalization, or the ability of a stimulus with similar elements to a CS (e.g., color, size, shape, location) to evoke a CR without its direct pairing with the US (Pearce, 1987), may play a key role (Pitman et al., 1993). External and internal stimuli that resemble but were not directly associated with the trauma may activate the excitatory association. For example, a news report of a sexual assault may trigger intrusive images of a rape victim's trauma, as may a community flier about preventing sexual harassment. When a CS is varied and complex, conditioning is facilitated (Mc Allister & Mc Allister, 1962) and the development of an inhibitory association to compound cues present during extinction is stunted (as is seen in appetitive conditioning, e.g. See, Grimm, Kruzich, & Rustay, 1999). Trauma in unpredictable contexts has a higher likelihood of generalization because of associative

learning to discrete rather than compound/complex cues (Foa et al., 1992). For instance, a caregiver who perpetrates abuse near the end of each month when finances are strained may invoke less generalized anxiety at other points in the month than a caregiver with erratic patterns of maltreatment. Further, inhibitory learning is context dependent, and trauma triggers may be ambiguous in their ability to evoke a CR based on the context in which the trigger is encountered (Bouton, 1984). While an intrusive memory may be manageable in a therapist office following imaginal exposure, it may cause extreme duress at home where imaginal exposure has not been rehearsed. In addition, deficits in extinction retention are found in PTSD compared to traumatized and non-trauma-exposed healthy controls, likely contributing to maintenance of the disorder (Blechert, Michael, Vriends, Margraf, & Wilhelm, 2007). Twin studies have demonstrated that combat-exposed individuals with PTSD have deficits in extinction retention whereas their non-exposed monozygotic twins do not (Milad et al., 2008). These PTSD-specific deficits in inhibitory learning are in addition to other factors that make long-term extinction retention difficult even in those without trauma exposure.

Inhibitory associations do not erase the original excitatory association, and continued competition for retrieval of both associations commonly results in return of fear (Bouton, 2004). Following exposure therapy for PTSD, the passage of time alone may result in a return of fear to trauma cues as occurs in spontaneous recovery (Pavlov, 1927; Quirk, 2002). Also, if exposure to trauma cues only occurs in limited contexts, as is the case in even well-planned exposure therapy, renewal of fear is common when CSs are encountered in novel contexts (Bouton, 2004). This is particularly problematic given that renewal occurs even following extensive extinction training (Bouton & Swartzentruber, 1989), though there are exceptions in extreme cases with 800 extinction trials following 8 conditioning trials (Denniston, Chang, & Miller, 2003). The

features that determine a novel context are varied and include both internal cues (such as mood states or influences of substances and medications; Bouton, Kenney, & Rosengard, 1990; Cunningham, 1979) and external environments. Finally, exposure to another painful stimulus may reinstate fear to the trauma cues that were previously extinguished (Pavlov, 1927; Rescorla & Heth, 1975). For example, military service members who complete exposure therapy following trauma, return to a combat zone, and are subsequently injured may experience reinstatement of fears to the original trauma cues. The limitations of reinstatement outside of the experimental laboratory are still being explored given that this effect may be due to the context in which the reinstating US is presented (Bouton & Bolles, 1979).

Cognitive Deficits Associated with PTSD

PTSD is associated with a variety of cognitive deficits that maintain distress and prevent the development of inhibitory learning. These include memory deficits, enhanced perceptual priming and poor stimulus discrimination, deficits in source monitoring, and attention deficits. Each of these domains are discussed in brief.

Memory deficits are observed across general, verbal, autobiographic, and working memory domains. Compared to controls, combat veterans with PTSD demonstrate reduced performance in the ability to recall words following interference from a distraction word list (Yehuda, Keefe, Harvey, & Levengood, 1995), and deficits in verbal memory are detected even if overall intelligence is preserved (Bremner et al., 1993). Similar deficits are present in children with PTSD, with 77.8% demonstrating impaired performance on general (i.e., non-trauma) memory tests compared to 13.6% of healthy controls (Moradi, Doost, Taghavi, Yule, & Dagleish, 1999). These deficits are detected in recall tests of both verbal and visual memory (LaGarde, Doyon, & Brunet, 2010). Animal models suggest that the relationship between stress

and memory impairment may be driven by neuronal damage in the hippocampus due to high glucocorticoid levels (Sapolsky, Packan, & Vale, 1988; Sapolsky, Uno, & Rebert, 1990).

Deficits in verbal memory are associated with reduced hippocampal volume (Bremner, Krystal, Southwick, & Charney, 1995), and these deficits are correlated with trauma abuse severity (Bremner, Randall, et al., 1995)

Autobiographical memory deficits are also reported in those with PTSD, both in terms of memory for details surrounding the trauma (Harvey, Bryant, & Dang, 1998) and non-trauma relevant memories (LaGarde et al., 2010; Wessel, Merceklbach, & Dekkers, 2002). Importantly, severity of trauma-relevant autobiographical memory deficits acutely following trauma is predictive of greater six-month PTSD severity (Harvey et al., 1998). Similarly to depression, PTSD is associated with reduced specificity of autobiographical memories compared to trauma-exposed controls (McNally, Litz, Prassas, Shin, & Weathers, 1994).

Deficits in working memory, or the ability to store and mentally manipulate information in short-term memory (Baddeley, 1992), is also impaired in PTSD. These deficits are associated with differences in brain activation in frontal and parietal brain regions compared to healthy controls (Weber et al., 2005). Similar deficits found in the dorsolateral prefrontal cortex have been linked to working memory difficulties in PTSD (Clark et al., 2003). Collective memory problems in PTSD likely contribute to the maintenance of the disorder via problems in encoding and storage of contextual details, elaboration of the memory, and integration of the memory with other memories (Ehlers & Clark, 2000).

Another domain of cognitive impairment in those with PTSD is related to enhanced perceptual priming toward trauma-relevant cues. This priming predisposes those with PTSD to a lower threshold of perceptual detection and enhanced processing of cues presented during

trauma (Ehlers & Clark, 2000), and greater generalization of learning to other cues post-trauma (Ehring & Ehlers, 2011). This threat priming is specifically toward cues associated with the trauma and not threatening cues more broadly (Michael et al., 2005). Enhanced priming for trauma-related cues is predictive of poor PTSD prognosis (Ehring & Ehlers, 2011). Relatedly, deficits in stimulus discrimination, or the ability to associate some cues with threat and others with safety (Jovanovic et al., 2010), is predictive of PTSD. Using conditional discrimination experimental designs, a variety of studies have demonstrated enhanced fear-potentiated startle responding to safety cues in PTSD compared to healthy controls even if explicit knowledge of the contingency (i.e., Cue-No US) remains intact (Grillon, Morgan, Davis, & Southwick, 1998; Jovanovic et al., 2010). Capacity for safety learning is linearly related to PTSD severity (Jovanovic et al., 2010; Jovanovic et al., 2009). Clearly, elevated responding to safe signals in PTSD is an important contributor to chronic distress.

Memory distortion occurs in PTSD, resulting in important changes to the memory that sometimes have major implications for the meaning of the event. Deficits in source monitoring, or the knowledge of the conditions under which a memory was encoded and related beliefs about those memories (Johnson, Hashtroudi, & Stephen, 1993), play a key role in the development and maintenance of PTSD. While source monitoring deficits were first explored in response to false memory claims in the 1990s, these deficits have important implications for distortion in important information for actual trauma memories. When categorizing words according to whether they were previously learned or not, those with PTSD make significantly more false detections and misses than trauma-exposed and healthy controls, and these false detections are detected with the same frequency as correct detections (Bremner, Shobe, & Kihlstrom, 2000). Trait anxiety and PTSD severity is related to degree of false recall (Zoellner, Foa, Brigidi, &

Przeworski, 2000). This tendency to falsely remember cues that were not present may underlie enhanced generalization of cues and deficits in safety learning, as those with PTSD may be more inclined to note absent associations between both trauma and safety cues and threat.

Finally, deficits in attention are related to the development and maintenance of PTSD. Sustained attention deficits are present in combat veterans with compared to without PTSD (Vasterling, Brailey, Constans, & Sutker, 1998; Vasterling et al., 2002). Further, 36% of combat veterans with PTSD provided retrospective reports of childhood attention deficit hyperactivity disorder (ADHD) compared to 9% of those with panic disorder (Adler, Kunz, Chua, Rotrosen, & Resnick, 2004). Similarly, in a study of maltreated children, 37% met criteria for ADHD (Famularo, Fenton, Kinscherff, & Augustyn, 1996). Attention bias toward threat is also detected in those with PTSD and is predictive of degree of fear conditioning and extinction learning (Fani et al., 2011). Collectively, these findings suggest that baseline impaired attention confers unique risk to PTSD, and that those with PTSD demonstrate preferential allocation of attention toward threat compared to those without PTSD.

Emotional Reactivity in PTSD

While PTSD is linked to elevated physiological and emotional responding in some cases, emotional distancing and numbing characterize other presentations (Roemer, Litz, Orsillo, & Wagner, 2001). Importantly, this tendency to avoid emotional experiencing and expression is associated with greater severity of diagnosis, poor treatment outcomes in even gold-standard treatments (Jaycox, Foa, & Morral, 1998), and includes numbing of both positively and negatively valenced emotions (Roemer et al., 2001). These numbing responses might reflect automatic responding to feared stimuli similar to freezing in rodents, possibly via catecholamine depletion or opioid-mediated analgesia, or they may reflect effortful avoidance through top-down

regulation strategies (see Roemer et al., 2001 for a review). Likely some combination of biological mediated and consciously motivated numbing result in reduced emotional arousal in a subset of patients with PTSD.

Emerging research in the area of emotional numbing has warranted two main subtypes of PTSD based on differences in emotional experiencing and reactivity (Lanius, Brand, Vermetten, Frewen, & Spiegel, 2012). Those with chronic histories of childhood neglect or maltreatment are more likely to experience elevated dissociation (van der Kolk et al., 1996), or disconnection from thoughts, feelings, or physical sensations (Bernstein & Putnam, 1986b). Furthermore, PTSD is one of two disorders highly associated with alexithymia (Zlotnick, Mattia, & Zimmerman, 2001), or deficits in identification, interpretation, processing, and communication of affect (Krystal, 1988) whereas substance use, eating, and panic disorder were not. Degree of alexithymia is related to a greater degree of childhood emotional and physical neglect (Zlotnick et al., 2001). While dissociation and numbing are related constructs that both contribute to PTSD severity, they differ in that numbing is characterized by an inability to feel emotions whereas dissociation represents reduced consciousness of emotions and thoughts and alterations in memory that serve to avoid trauma triggers (Feeny, Zoellner, Fitzgibbons, & Foa, 2000). Importantly, emotional numbing acutely following trauma exposure confers elevated risk of PTSD diagnosis even after controlling for symptoms of both dissociation and depression (Feeny et al., 2000). Some data suggests that emotional numbing following trauma is a result of prolonged hyperarousal, resulting in depletion of emotional resources available for expression (Foa et al., 1992; Litz et al., 1997). Differing patterns of neural activation, both in terms of under- and over-modulation of emotional reactivity, in brain regions including the amygdala, insula, dorsal anterior cingulate

cortex, and medial prefrontal cortex, further support this distinction (Lanius, Vermetten, Loewenstein, et al., 2010)

Even when emotional expression abilities remain intact, they are frequently biased toward communication of less vulnerable emotions (Riggs, Dancu, Gershuny, Greenberg, & Foa, 1992). Diagnoses of PTSD are strongly related to anger and hostility across a variety of samples (Jakupcak et al., 2007; Orth & Wieland, 2006). While other anxiety disorders are also associated with anger, the effect size between PTSD and anger (compared to controls and anger) is at least twice as large (Cohen's $d=1.07$) as the comparison for panic disorder (Cohen's $d=.46$), obsessive compulsive disorder (OCD; Cohen's $d=.41$), or generalized anxiety disorder (GAD; Cohen's $d=.37$; Olatunji, Ciesielski, & Tolin, 2010). This relationship is particularly important because high dispositional and situational anger in PTSD has been related to poor treatment outcome and higher treatment dropout (Foa, Riggs, Massie, & Yarczower, 1995; Forbes, Creamer, Hawthorne, Allen, & McHugh, 2003; Forbes et al., 2008; Rizvi, Vogt, & Resick, 2009). Anger may be reinforced through disengagement with other more vulnerable emotions, such as fear and shame (Foa, Steketee, & Rothbaum, 1989; Jaycox & Foa, 1996) and may inhibit expression of fear (Riggs et al., 1992). Reduced fear arousal caused by anger expression may prevent the development of inhibitory learning to compete with the excitatory feared association, thereby maintaining PTSD and worsening treatment outcomes (Foa et al., 1995; Forbes et al., 2003; Pitman et al., 1991; Riggs et al., 1992).

Shame is also highly associated with self-directed anger in PTSD, and in a comparison between self- and other-directed anger (among other variables), shame was the only significant mediator of the relationship between childhood abuse and longitudinal prediction of PTSD (Andrews, Brewin, Rose, & Kirk, 2000). Therefore, while secondary anger is clearly related to

the maintenance of PTSD, this relationship might be driven by strong primary emotions like shame. The tendency to attempt anger suppression has been more strongly associated with PTSD than the tendency to express anger in some samples, though both "anger in" and "anger out" have significant relationships with PTSD (Orth & Wieland, 2006). Therefore, treatments that emphasize training toward healthy identification and expression of primary emotions may facilitate improved outcomes.

More broadly, general emotional expression following trauma exposure is linked to disorder severity. For instance, one study found that women were more emotionally expressive following trauma compared to men (Resick, 1986), which is consistent with the literature on non-traumatized gender differences in emotional expression (Polce-Lynch, Myers, Kilmartin, Forssmann-Falck, & Kliewer, 1998). Further, degree of emotional expressivity is predictive of subsequent PTSD diagnosis (Resick, 1986). However, a more parsimonious explanation for the relation between enhanced emotional expression and trauma exposure is that emotional expression is a byproduct of trauma reactivity, one symptom cluster of PTSD (American Psychological Association; 2013), rather than an independent risk factor for the diagnosis. Women with HIV and trauma benefit from expressive writing whereas men do not (Ironson et al., 2013), perhaps because of their increased proclivity for emotional expression. Theoretically, emotional expression during active recall of a trauma may lead to enhanced arousal and may result in improved memory of the traumatic experience, allowing for direct encounter with more trauma stimuli and more effective inhibitory learning (Keane, Zimering, & Caddell, 1985). However, this facilitation of inhibitory learning may only occur following expression of primary emotions, such as fear and shame, rather than secondary emotions, such as anger.

Empirically-Supported Treatments for PTSD

Several evidence-based cognitive behavioral therapy (CBT) treatment manuals have been developed for PTSD based on principles of fear conditioning, including Prolonged Exposure (PE; Foa et al., 1999; Foa & Kozak, 1986) and Cognitive Processing Therapy (CPT; Resick & Schnicke, 1992). These treatments have similar components with varying degrees of emphasis, though both include psychoeducation, exposure, and cognitive restructuring. Direct comparisons of these treatments have revealed few outcome differences on either PTSD or depression symptoms, though both outperform waitlist control groups (Resick & Schnicke, 1992).

Meta-analyses of effect-size estimates (according to Cohen's *d* standards; Cohen, 1988) revealed large effect sizes both for all treatments from pre- to post-treatment compared to wait list or usual care (Cohen's $d=1.43$; Bradley et al., 2005) and when just CBT was considered (Cohen's $d=1.70$, Bisson et al., 2007). The effect-sizes remain large in comparison to supportive control groups (Cohen's $d=.83$, Bradley et al., 2005). Despite these positive findings, many people fail to respond to treatment (Loerinc, Meuret, Rosenfield, & Craske, in press) or drop out early (Haby, Donnelly, Corry, & Vos, 2006a). In one meta-analysis, 46% of patients completing all treatments and 53% of those completing CBT did not meet the threshold for clinically significant improvement (Bradley et al., 2005). More recent estimates suggest that CBT for PTSD has medium effect sizes (Hedges' $g=.62$, 95% CI=.28-.96; Hofmann & Smits, 2008) on continuous self-report measures compared to placebo. Improvements in the available treatments are needed, and one promising area is in refinement of experimental analogs of anxiety treatments, in particular methods for enhancing extinction learning.

Treatment protocols have been developed to introduce skills for tolerating emotions (using affect labeling, mindfulness, distraction, and activity scheduling) prior to CBT in PTSD and have found added benefits compared to supplemental supportive therapy sessions plus CBT (Bryant et al., 2013). However, dismantling studies have not yet been conducted to establish the

active ingredients in emotion tolerance treatment for this population. Furthermore, differences in the number of completed sessions between the comparison groups in this study prevent thorough comparisons as the emotion tolerance training condition had significantly more sessions than the support condition. This study also had significant problems with attrition, perhaps suggesting that the intervention was not well-tolerated by some patients.

In addition to problems with response rates and attrition, a number of logistical barriers prevent the widespread dissemination and adoption of empirically supported treatments for PTSD. While gains have been made in this area following the mandate of empirically supported treatments in the United States Department of Veterans Affairs (VA) through programs like the PTSD Mentoring Program, PTSD Consultation Program, and the rollout of CPT (Resick & Schnicke, 1992) and PE (Foa, Keane, & Friedman, 2000; Karlin et al., 2010), many remain without adequate care even within the VA. For instance, service utilization intensity in the VA has decreased from 1997-2005, perhaps reflecting increasing demands on health service providers in the organization, resulting in fewer treatment sessions and the potential for less potent treatment effects (see Rosenhack & Fontana, 2007, for a review). Furthermore, a large proportion of trauma victims are not eligible for benefits through the VA and, with exceptions based on location (Clark et al., 2009), have few accessible treatment options particularly in impoverished locations (Chow, Jaffee, & Snowden, 2003). Further, most research on treatment dissemination of late has been within the confines of the VA, leaving gaps in assessment of treatment utilization for civilian trauma. Of the available research, estimates of treatment seeking for PTSD vary by ethnicity, with roughly half of white sufferers seeking treatment compared to a third or fewer of black or Asian sufferers (Roberts, Gilman, Breslau, & Koenen, 2011). These ethnic differences in treatment seeking are likely multiply determined and do not parse treatment receipt from desire for treatment, but they are consistent with disparity in

treatment access by ethnicity for other disorders (Wang, Olfson, Pincus, Wells, & Kessler, 2005). Technology-assisted or delivered options are becoming increasingly available and promising (Knaevelsrud & Maercker, 2007), though these options typically still require the presence of a supportive therapist. Clearly, there is a need for accessible and effective treatments that can be disseminated in a wide variety of contexts, justifying the further development of technology-administered treatment protocols.

Affect Labeling as a Supplement to Exposure Therapy for Trauma

The limitations described above indicate the need for further exploration of supplemental approaches for the treatment of PTSD. Affect labeling has gained support as an adjunctive component for non-trauma based exposure therapies and may be a promising avenue of inquiry. While Affect Labeling was initially used to describe any label of the emotional content of a stimulus (Lieberman, Inagaki, Tabibnia, & Crockett, 2011), it has been used in some clinical studies to describe one's emotional experience. One example of affect labeling is verbalizing an emotional response to a feared stimulus; for example, a person who is engaging in imaginal exposure to a sexual assault memory might verbalize "disgust," "shame," or "sad" during exposure trials. Research consistently demonstrates that verbalizing one's emotional experience results in an attenuation of negative affect (Pennebaker, 1997).

Expressive writing has been explored as a stand-alone intervention for PTSD with some encouraging results, both in terms of reduced trauma and depressive reactivity. Following 12 one-hour sessions of writing supplemented with supportive, non-directive therapy, Hedges' *g* effect size estimates for reduction in PTSD symptoms were .07 and 1.0 at post-treatment and six-month follow-up (Resick et al., 2008). However, at least three other studies have not replicated this finding (see Ironson et al., 2013 for a review). Similarly, trauma-exposed individuals living

with HIV experienced a reduction in CD4 + lymphocyte counts following expressive writing about their trauma (Petrie, Fontanilla, Thomas, Booth, & Pennebaker, 2004), though other studies of this population have not found a benefit from expressive writing in terms of reduced trauma reactivity (Wagner, Hilker, Hepworth, & Wallston, 2010). Therefore, while expressive writing may confer a benefit to trauma survivors, there is not enough evidence to support it as a stand-alone intervention. Further, other techniques that accomplish goals in line with expressive writing, such as affect labeling, may have more clinical utility due to their efficiency.

Affect labeling attenuates amygdala activation while increasing activation of the right ventrolateral prefrontal cortex (rVLPFC; Hariri, Bookheimer, & Mazziota, 2000; Lieberman et al., 2007). Extinction training decreases activation of the amygdala (Phelps, Delgado, Nearing, & LeDoux, 2004), and strategies that further downregulate amygdala activation (either in concert or occurring as a parallel process) may enhance extinction retention. This suggests the possibility that combining affect labeling, which reduces amygdala activation, with extinction training, which also reduces amygdala activation (either through the same or complementary pathways), may provide an enhanced treatment modality for individuals exposed to trauma.

Affect labeling may operate similarly to and have underlying shared neural pathways with cognitive reappraisal, a strategy that has been incorporated into CBT for PTSD. When reappraisal, an intentional regulation strategy, was compared to affect labeling, an implicit regulation strategy, during observation of emotionally-triggering stimuli in healthy controls, similar reductions in self-reported distress were reported relative to observation alone (Burklund, Creswell, Irwin, & Lieberman, 2014). In the same task, activations in RVL PFC were observed in both regulation strategies relative to observation-only, as were reductions in amygdala. Interestingly, affect labeling was associated with enhanced RVL PFC activation relative to

reappraisal, though there were no differences in amygdala activation between these groups. Other studies have reported similar results, though with some benefits in terms of self-reported distress for reappraisal over affect labeling (Lieberman et al., 2011).

Evocative images paired with affect labels generate attenuated amygdala activation compared to shape or gender labeling/matching or exposure only (Lieberman et al., 2007). Irrelevant labels generated attenuated autonomic activation (e.g. skin conductance responding and heart rate) compared to exposure alone or exposure and neutral or relevant labels in healthy participants (Tabibnia, Lieberman, & Craske, 2008). Similarly, negative labels reduced skin conductance responding to images of spiders in a phobic sample compared to exposure alone or exposure and non-affective labels (Tabibnia et al., 2008). Importantly, those in the affect labeling condition generalized their reductions in autonomic reactivity to novel images without any labels, whereas those previously trained in neutral labeling or exposure alone did not, suggesting that affect labeling may confer benefits above and beyond those observed from extinction training even in the absence of the labels in later trials.

Affect labeling has also been compared to other emotion regulation techniques, such as reappraisal or distraction, for clinical samples. Kircanski, Lieberman, and Craske (2012) exposed spider phobic participants to a tarantula and randomized them to one of four groups: exposure alone, affect labeling plus exposure, reappraisal plus exposure, and distraction plus exposure. In the affect labeling task participants verbalized one negative emotional response to the spider and one negative description of the spider for each exposure. The reappraisal group verbalized a description of the spider that was intended to make them feel less negatively about the spider. The distraction group verbalized a one sentence description of a room in their home and an object in that room. Affect labeling during exposure outperformed all of the other

conditions in terms of skin conductance responding and behavioral approach when participants with spider phobia are re-tested in a novel context. Participants who verbalized more words related to fear and anxiety experienced a significantly greater reduction in SCR.

In the context of social anxiety disorder, engaging in affect labeling results in a pattern of activation that differs from healthy controls. Specifically, whereas affect labeling increases RVL PFC activity and decreases amygdala activity in healthy controls, this task results in increased RVL PFC and amygdala activation in those with social anxiety disorder (Burklund, Craske, Taylor, & Lieberman, 2015). This enhanced amygdala activation appears driven by those with comorbid social anxiety and depression. When amygdala activation was directly compared between social anxiety and healthy controls, the socially anxious group had significantly greater amygdala activity, which was also driven by the comorbid depression group. One possible explanation for these findings is the possibility of abnormal connectivity between these brain regions in comorbid social anxiety and depression. These results provide evidence that the neural activation resulting from affect labeling differs between clinical and healthy comparison conditions, and therefore, the effect on psychopathology may be different.

The benefits of affect labeling have also been reported in public speaking anxiety. Those who engaged in an affect labeling task prior to speech performance had significant reductions in HR and the number of spontaneous skin conductance responses during recovery from the speech task compared to those in a shape matching control task (Niles, Craske, Lieberman, & Hur, 2015). Further, greater use of anxiety labels was associated with steeper declines in physiology during anticipation of exposure and greater reductions in self-reported distress overall. These results are moderated by baseline implicit emotion regulation ability, such that those with less

implicit ability performed better in affect labeling compared to the control condition during speech anticipation in terms of physiology and self-reported distress.

Due to the research on increased and spontaneous use of skills in managing emotions when they are practiced frequently (Mohlman, 2008), it is possible that teaching individuals to purposefully use affect labeling may increase their use of this skill when anxious. Neural plasticity research also suggests that learning a new skill results in structural or functional changes in the brain that increase the neural efficiency of engaging in that skill in the future (Harding, Paul, & Mendl, 2004; May et al., 2007). Affect labeling may result in neuronal changes that allow for more enhanced downregulation of amygdala activation by the PFC, and resulting reductions in physiological arousal and self-reported distress.

Script-Driven Imagery

One gold-standard method for retrieving trauma memories and assessing reactivity to trauma is the Script-Driven Imagery assessment (SDI-A; Lang, Levin, Miller, & Kozak, 1983) procedure. This procedure involves listening to a personalized audio-recorded description of an event for 30 s followed by imagining the event in detail for 30 s, and finally a recovery period for 60 s prior to the next script presentation (see Figure 1). SDI-A has been adapted for measuring reactivity to trauma memories, both in terms of physiology and brain imaging (Pitman, Orr, Forgue, de Jong, & Claiborn, 1987). Individuals with PTSD have a greater increase in heart rate and electromyography (EMG) responding in traumatic compared to neutral scripts in SDI-A (Shin et al., 2004). PTSD patients also have increased limbic activation while listening to a trauma memory compared to a neutral memory in SDI-A (Rauch et al., 1996). Therefore, there is support for the efficacy of the SDI-A in eliciting heightened responding for individuals with trauma exposure from both physiological and neuroimaging studies.

The SDI-A procedure has also been used as an outcome variable to determine the efficacy of treatments for PTSD. In a report of three cases studies of Israeli participants with PTSD who completed the SDI-A before and after treatment for systematic desensitization, all significant elevations (relative to neutral scripts) in heart rate, skin conductance, and frontalis EMG in pre-treatment responding were absent at post-treatment (Shalev, Orr, & Pitman, 1992). Interestingly, in cases of multiple trauma exposure, significant reductions in reactivity were observed to the trauma that received treatment but not untreated traumas. In a sample of 51 Vietnam veterans, exposure therapy resulted in pre- to post-changes in heart rate and skin conductance responding that was not present in a supportive comparison condition (Boudewyns & Hyer, 1990). Relative to a waitlist group, Brief Eclectic Therapy, including exposure and cognitive restructuring, demonstrated significant reductions in blood pressure and heart rate from pre- to post-treatment (Lindauer et al., 2006). Finally, significant reductions have been observed in heart rate responding to the SDI-A following propranolol treatment of PTSD relative to placebo (Hoge et al., 2012; Pitman et al., 2002). Therefore, there is clear justification for the use of SDI-A as a tool for assessing treatment outcome.

As would be expected based on the subtypes of PTSD described above (Lanius, Bluhm, Lanius, & Pain, 2006), responding to the SDI-A is heterogeneous. In one study, 30% of participants reported elevations in dissociation rather than subjective distress, and did not demonstrate elevated psychophysiology (Lanius et al., 2006). In contrast, 70% of participants reported strong experiences of reliving the trauma, and had corresponding elevations in heart rate.

While SDI-A is traditionally used as an assessment tool, it also acts as an abbreviated form of imaginal exposure to a traumatic memory. If a procedure such as SDI-A is effective at

reducing reactivity to a traumatic image, then it could potentially be a much more efficient form of imaginal exposure compared to 9-12 90 minute Prolonged Exposure sessions (Foa, Rothbaum, Riggs, & Murdock, 1991). It is also possible that employing affect labeling during an SDI Training (SDI-T) may have an additional benefit over traditional exposure therapy. Therefore, this study explored the feasibility of employing SDI-T as an exposure technique and aimed to determine if there are additional benefits of using affect labeling during SDI.

The two studies described herein provide two variations of a SDI-T that has been augmented with Affect Labeling. The pilot study compared Affect Labeling to a control condition, Shape Labeling, and included a forced choice between two labeling options in each condition. In addition, the pilot study evaluated the timing of labeling (e.g., During or After trauma imagination) to determine the effect on long-term fear reduction. The main study uses the timing information provided in the pilot study and included three experimental conditions informed by Kircanski and colleagues (2012): Affect Labeling, Distraction Labeling, and Exposure Only. The active labeling conditions in the main study allowed participants to freely choose their Distraction or Affect Labels, providing a standardized labeling test that more closely paralleled the potential clinical use of this supplemental exposure strategy.

Specific Aims

Pilot study.

Specific aim 1. This study aimed to demonstrate that a brief imaginal exposure Training using the SDI-T procedure would result in significant reductions in trauma symptoms from Pre- to Post-Training. We hypothesized that there would be significant reductions in physiology and self-report symptoms over Time.

Specific aim 2. The study aimed to evaluate the degree to which type of labeling (“Task”) reduces physiological and self-report distress. Therefore, Affect Labeling (“AL”) was compared to a control condition, Shape Labeling (“SL”), from Pre-to Post-Training. We hypothesized that the AL group would demonstrate greater reductions in physiological and self-report responding to trauma cues compared to the SL group.

Specific aim 3. Within both AL and SL, the study compared the timing of labeling (“Order”; During vs. After the imagination phase of the SDI-T), as a predictor of outcome. We hypothesized that across both labeling conditions, labeling After imagination would result in greater reductions in physiological and self-report responding to trauma cues compared to the labeling During groups due to reduced distraction during the exposure, and hence increased ability to fully engage in the exposure.

Specific aim 4. This study compared the interaction of Task (AL vs. SL), Order (During vs. After Imagination), and Time (Pre vs. Post-Training) on physiological responding. We hypothesized that the AL-After group would benefit the most for reasons described in the prior two aims.

Specific aim 5. Finally, the pilot study compared choice of negative emotion versus neutral emotion words as a predictor of improved outcome. This was an exploratory aim, but we hypothesized that greater overall percentage of negative emotion word choices would result in greater reductions in physiology and self-reported distress compared to neutral emotion word choices.

Main Study.

Specific aim 1. We aimed to replicate the effects of Time found in the pilot study in a larger sample recruiting more heavily from community members. We hypothesized that there would be significant main effects of Time on psychophysiological and self-reported measures of trauma distress.

Specific aim 2. In addition, another goal was to determine whether AL enhances the effect of the training compared to either DL or EO. We hypothesized that AL would result in lower overall levels of physiological activation during the training and at Post-Training.

Specific aim 3. We aimed to determine whether there was an interaction between Time X Condition on physiological and self-report measures of symptom distress. We hypothesized that the reduction in physiological and self-report distress would be greatest in the AL condition.

Specific aim 4. In addition, we aimed to determine whether trauma severity moderated the relationship between Time, Condition, and outcome variables. We hypothesized that those with more severe trauma severity and functional impairment would receive the greatest benefit.

Specific aim 5. We also aimed to use negative emotion word choice throughout AL as a predictor of physiological and trauma severity outcome. We hypothesized that greater use of anxiety affect labels would result in improvements in physiological responding and trauma severity, whereas greater use of anger affect labels would result in less improvement.

Specific aim 6. Finally, we aimed to determine the trajectory of symptom change throughout each trial of the training. We hypothesized that the pattern of change would be non-linear due to return of fear between phases of the experiment (Plendl, 2010).

Pilot Study Methods

Study Design

The pilot study used a 2 (Pre- vs. Post-Training, Aim 1) X 2 (AL vs. SL; Aim 2) x 2 (During vs After; Aim 3) design. All participants completed the SDI assessment (SDI-A) as the primary psychophysiological outcome assessment, and a self-report trauma questionnaire as the primary subjective outcome at Pre and Post-Training (SDI-T). The SDI-T included 12 trials on two separate days (24 trials total) using the trauma script.

Participants

Participants ($n=41$, 32 completers) were adult college undergraduates and community members. They were 19.7 ($SD=3.2$) years old on average, 85.4% female, 26.8% Asian/Asian American, and 22% Caucasian. See Table 1 below for full demographic details. Seven participants did not complete the study for unspecified reasons, one did not complete for scheduling reasons, and one did not complete due to equipment problems.

Table 1. Demographic Details for Pilot Study.

	AL- During	AL-After	SL- During	SL-After	Total	Order (During vs. After)	Task (Affect Vs. Shape)	Order x Task
Age	18.6 (SD=.9)	19.3 (SD=1.8)	18.8 (SD=.9)	22.0 (SD=5.6)	19.7 (SD=3.2)	$F(1,38)=4$.804, $p=.05$, partial $^2=.097$	$F(1,38)=2.2$ 20, $p=.145$, partial $^2=.055$	$F(1,37)=$ 1.855, $p=.181$, partial $^2=.048$
Ethnicity						Fisher's exact $p=.44$	Fisher's exact $p=.58$	Fisher's exact $p=.10$
Asian	18.2%	50%	30.0%	0%	22.0%			
Caucasian	27.3%	12.50%	20.0%	30.0%	26.8%			
African American	0%	25%	20.0%	0%	9.8%			
Latino	18.2%	0%	10.0%	20.0%	12.2%			
Other	36.4%	12.50%	20.0%	20.00%	29.2%			

Percent Female	100%	80%	90.9%	90.0%	85.4%	Fisher's Exact=.52	Fisher's Exact=.16	Fisher's exact: .73
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Undergraduates were recruited from the Psychology Department mass testing subject

pool. Students who scored in the clinical range on the PTSD Checklist, a screening measure, were invited via email and phone call to participate in the study. Per the recommendations from Cook, Schnurr, & Foa (2004) on contraindications for exposure therapy, participants were deemed ineligible if they were currently experiencing psychosis, mania, hypomania, suicidality, or self-injury. In addition, individuals with severe dissociation were excluded because they may represent a different subtype of PTSD with significant deviations in affect modulation and brain activation (Lanius, Vermetten, Lowenstein, et al., 2010). Participants were also ineligible if they had severe depression. This decision was informed by findings that participants with depression respond differentially to SDI-A than participants without depression (Lanius et al., 2007) and because of evidence suggesting distinct physiological patterns in depressed individuals (Desmond & Walter, 1969; Frith, Stevens, Johnstone, & Crow, 1982). For completing the study, participants were offered 5 course credits or \$40 as compensation.

Measures

Script-Driven Imagery assessment (SDI-A). The SDI-A was conducted at baseline (Day 1) and one week after the SDI-T was completed (Day 15; see Appendix I for study diagram). Two written narratives based on a traumatic event and a neutral event were generated for this procedure. In order to generate the trauma script, the following was read to participants:

"We would like you to write a description of a traumatic event that you have experienced. Include in your description the bodily sensations you were aware of at the time. We will interview you in more detail about this experience later. Sometimes it is difficult to think

of something to write 'on the spot.' It may help to close your eyes and imagine yourself back in the situation. Try to generate the same sensations and feelings that you experienced at the time. While the image is vivid in your memory, jot down the details of the scene and the sensations you experienced. Describe the trauma situation. Please include such details as who was there, what you were doing, where you were, how things looked, what you heard, etc. Continue on the reverse side if necessary. On the second page are lists of body sensations and emotions that you may have had during that experience. Circle any of them that you remember.”

The experimenter directed the participants to focus on the most distressing portion of the traumatic event and to write about that specific portion of the event in detail. Participants selected up to 5 responses from a list of bodily responses and emotions that accompanied the experience. The written responses were reviewed, clarified, and expanded upon when necessary. The writing samples were translated into audio-recordings that were made in the first person and present tense. These recordings were approximately 30-seconds in duration and incorporated five of the selected bodily responses, or as many as the participant selected, whichever was less. The script also included a description of at least two senses (sight, hearing, touch, smell, taste).

Then a similar procedure was undertaken for generating a script describing a neutral event. The following was read to participants:

"We would like you to write a description of a neutral event that you have experienced. It is important that this experience happened within a year of the trauma (before or after) you have just written about. Include in your description the bodily sensations you were aware of at the time. We will interview you in more detail about this experience later. Sometimes it is difficult to think of something to write 'on the spot.' It may help to close

your eyes and imagine yourself back in the situation. Try to generate the same sensations and feelings that you experienced at the time. While the image is vivid in your memory, jot down the details of the scene and the sensations you experienced. Describe the neutral situation. Please include such details as who was there, what you were doing, where you were, how things looked, what you heard, etc. Continue on the reverse side if necessary."

The SDI-A included two trials of the trauma script and two of the neutral script, presented in counterbalanced order. Each trial included 30 s of listening to the script, 30 s of imagining the context of the script, and 60 s of recovery (see Figure 1).

Figure 1. Script Driven Imagery Assessment (SDI-A).

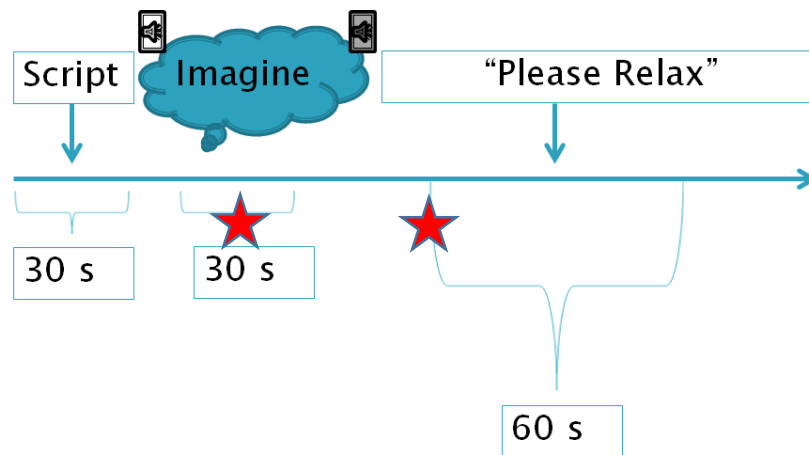


Figure 1 Note: The speaker indicates the presentation of a tone. Each trial included two startle probes presented during the imagination portion of the trial, represented by the stars in the diagram. Each trial included the presentation of either the trauma script or the neutral script.

Prior to the SDI-A, participants completed a 2-minute baseline to collect resting physiological levels. A practice trial with a standardized test script was presented at the beginning of SDI-A to orient participants to the different phases of the trial. Two startle probes were presented during the imagination portion for each trial.

Physiological responding during these assessments was used as the primary outcome measure, and was captured using Variotest software Version 1.89 (Mutz, 2012). These included the following:

Skin Conductance Level (SCL). SCL was measured with two electrodes that were affixed to the palm of the participants' non-dominant hand (see Appendix II Part A). Electrode gel was used to increase conductivity. A sampling rate of 64 Hz was used and data were visually inspected for noise, and filtered using a .05 Hz low-pass butterworth filter. Average SCL while listening to the script and imagining the contents of the script was averaged across trials for both the trauma script and the neutral script and was calculated as a difference between responding during the portion of interest and the baseline. SCL is used in lieu of skin conductance response (SCR) when trial length is longer than a few seconds (Dawson, Schell, & Filion, 2000).

Fear-potentiated startle electromyography (EMG). Electromyography (EMG) response following a startle probe differentiates fear evoking stimuli and neutral or safe stimuli (Traupe & Kaernbach, 2011). This suggests that startle modulation can reflect a priming of a fear response. Therefore, this study measured startle responding to probes presented during the assessment phases. Startle probes were presented during the imagination portion of trials during the SDI assessment. The area under the participants' right eye was cleaned with an alcohol swab and two Ag-AgCl electrodes were affixed beneath the eye (8 mm below the eye, 1 cm apart; see Appendix II part B). Participants wore headphones which delivered a .5 MS burst of white noise. An infinite impulse response (IIR) 55 Hz notch filter was applied to reduce noise in the data. EMG amplitude was calculated as the difference between the mean response in the 200 ms prior to startle probe onset and the maximum in the 20-200 ms after probe onset, and the integral was calculated as the area under the curve within the same window. Responses including

excessive noise or blinks in the 50 ms baseline window were excluded from analysis. These values were then log-transformed for normality.

Frontalis electromyography (F-EMG). The area above each participants' right eyebrow was cleaned with an alcohol swab and two Ag-AgCl electrodes were affixed above the right eyebrow to measure the frontalis muscle (see Appendix II Part C). F-EMG can be analyzed across longer phases of measurement rather than in response to discrete stimuli (see for example Delmonte, 1979; Leboeuf & Lodge, 1980). Therefore, in the current study F-EMG responding was averaged during the imagination portion of the neutral and trauma script trials of the assessments and was calculated as the difference from the phase of interest and baseline. A 55 Hz infinite impulse response notch filter was used on F-EMG data.

Heart rate (HR). Heart rate was measured with three electrodes: one under the participant's right collarbone, another just below the last rib on their left side, and a ground electrode under the left collarbone. HR was recorded at 16 Hz and data were filtered using an infinite impulse response notch filter. Beats per minute were averaged across the imagination portion of the neutral and trauma script trials during the assessments and was calculated as the difference between the phase of interest and the baseline.

Self-report measures.

PTSD Checklist (PCL; Blanchard, Jones-Alexander, Buckley, & Forneris, 1996; Weathers et al., 1996). The PCL includes 17 PTSD symptoms that participants rated on a 1 ("not at all") to 5 ("extremely") scale according to how bothered they were by each symptom in the past month. Individuals who scored 34 or greater on the measure during the psychology mass

pre-testing were invited to participate. The PCL has demonstrated good validity and reliability (Blanchard et al., 1996).

Mini-International Neuropsychiatric Interview (MINI; Sheehan et al., 1998). The MINI is a clinician-rated brief interview that was used to diagnose anxiety, mood, psychotic, substance, and eating disorders. It also measures suicidality both in the context of depression and outside of depression screening. The MINI is a valid and reliable measure and is widely used in treatment-outcome studies (Sheehan et al., 1998).

Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986a). A shortened 8-item version of the DES was used in the current study that is rated on a 0 (“never”) to 5 (“at least once a week”) point Likert scale. An example item is “I find that I did things that I do not remember doing.” The full version of the scale is a valid and reliable measure of dissociation (van Ijzenboorn & Schuengel, 1996).

Inventory of Statements about Self-Injury (ISAS; Klonsky & Glenn, 2009). The ISAS is a comprehensive measure of non-suicidal self-injurious behaviors and related symptoms. The current study included only the list of self-injurious behaviors. Participants were asked to indicate whether they have ever engaged in these behaviors, the frequency of the behaviors, and whether the behaviors occurred in the prior month. Any episode of self-injurious behavior in the prior month resulted in exclusion from participation. The full version of the ISAS is a valid and reliable measure of self-injury (Klonsky & Glenn, 2009).

Beck Depression Inventory-II (BDI; Beck, Steer, & Brown, 1996; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). The BDI is a widely used 21-item questionnaire that broadly assesses symptoms of depression and negative mood. A meta-analytic review of

psychometric properties of the instrument revealed a Cronbach's $\alpha = .81$ for internal consistency and a correlation with the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960), another commonly used depression instrument, of $r = .74$ for non-psychiatric patients (Beck, Steer, & Carbin, 1988). Participants who scored 29 or greater on the BDI or endorsed moderate to severe suicidal ideation on the BDI (by indicating either "I would like to kill myself" or "I would kill myself if I had the chance") were deemed ineligible.

Posttraumatic Diagnostic Scale. (PDS; Foa, Cashman, Jaycox, & Perry, 1997) The PDS is a measure of trauma exposure and reactivity to trauma. It includes 49 items and four sections: 1) trauma checklist, 2) selection of the most upsetting trauma, 3) ratings of the 17 PTSD symptom levels on a 0 (not at all) to 3 (almost always) scale, and 4) functional impairment resulting from symptoms. The measure provides a total severity score ranging from 0-51 for each of the 17 PTSD symptoms and has demonstrated high reliability and sensitivity (Foa et al., 1997). The 9 impairment items are dichotomous (i.e., impairment present or absent) across number of functional domains (range of scores from 0-9). This measure was only administered at baseline in the Pilot Study.

Impact of Events Scale (IES; Horowitz, Wilner, & Alvarez, 1979). The IES is a 15 item measure of responding to stressful events that gauges symptoms to a traumatic event on a zero (not at all) to three (often) Likert scale. The measure asks about responding in the prior seven days. Example items include "I thought about [the event] when I didn't mean to," and "I stayed away from reminders of it." It has demonstrated high test-retest reliability (.87 for the total score, .89 for intrusion, .79 for avoidance) and validity as a measure of responding to stressful situations (Horowitz et al., 1979). In the beginning of the pilot study, two participants

mistakenly completed this measure about a trauma unrelated to the trauma that they rated as the most distressing, and therefore their data on this measure was discarded.

Client Satisfaction Questionnaire (CSQ; Attkisson & Zwick, 1982). The CSQ is an 8 item measure of client satisfaction rated on a 1-4 point Likert scale that was administered at the end of the study to gauge the acceptability and face-validity of the Training. This measure asks about the quality of services, the extent to which the program met participant needs, likelihood of recommending the program to others, and willingness to seek help in the same program should a similar problem arise in the future. The CSQ has high internal consistency and validity (Attkisson & Zwick, 1982).

SDI-T

Each experimental condition included 24 total trials across two SDI-T phases, with 12 trials in each SDI-T. All SDI-T sessions began with a practice trial to orient participants to the format. In all trials, participants were given 5 s to make a labeling decision. If a response was not selected within this window, the options were removed and the experiment continued.

Affect Labeling After Exposure (AL-After; $n=10$, $n=8$ completers). The trial order for this condition was as follows: exposure to the trauma script (30 s), imagination of the contents of the trauma script (30 s), followed by three affect labeling decision points (each lasting up to 5 s for a total of 15 s), and recovery (60 s; see Figure 2 below).

Figure 2. Labeling After imagination example.

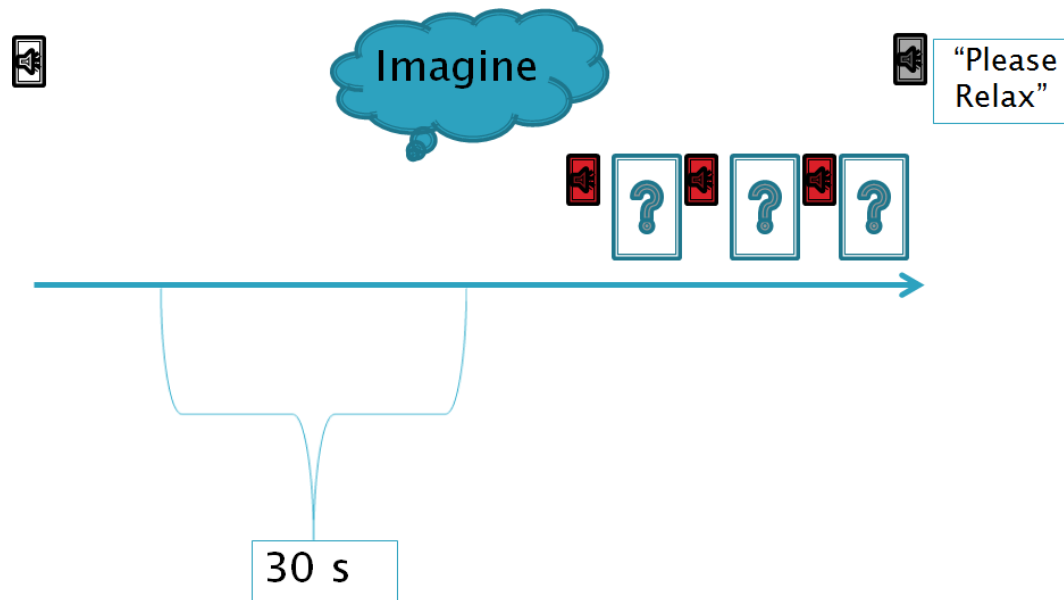


Figure 2 Note: The question mark (?) indicates the presentation of a labeling decision point.

These AL decision points were prompted by an audio tone and the presentation of a neutral image with two emotion labels beneath it (see Figure 3 below).

Figure 3. Example of AL decision point.



The neutral image was presented to control for the presence of a visual stimulus in the shape labeling task. The emotion labels were randomly chosen from a list of negative and neutral words and the participants were instructed to choose the word that most accurately describes their current emotion (see Table 2 below). Choices were indicated by a button press on a keyboard.

Table 2. List of neutral and negative words.

Neutral Words	Negative Words
Relaxed	Sad
At ease	Angry
Calm	Disgusted
Bored	Afraid
Nonplussed	Downhearted

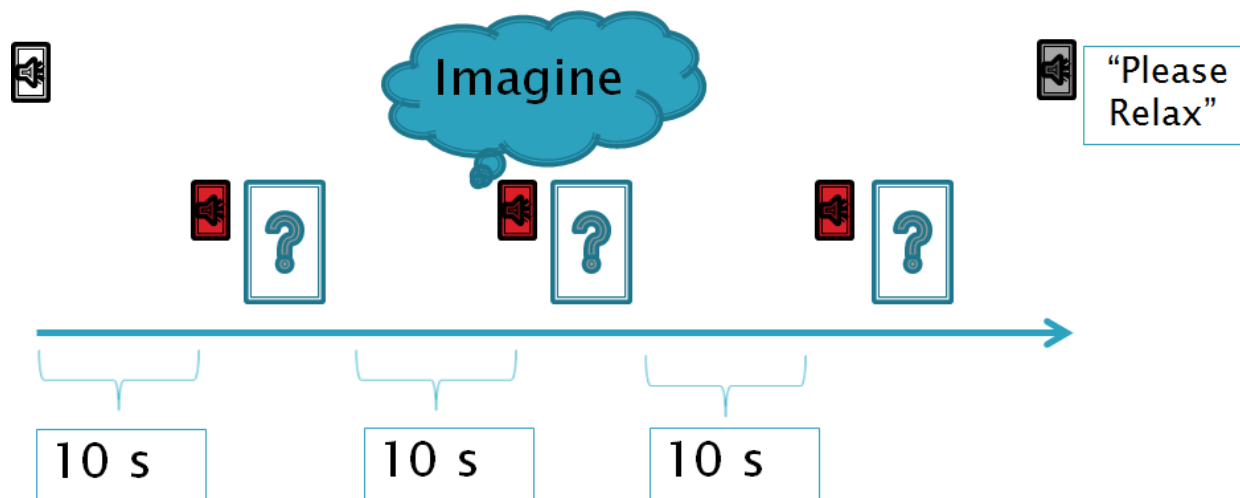
Uninterested	Nervous
Unconcerned	Lonely
Uninvolved	Distressed
Indifferent	Blameworthy
Impartial	Guilty
Neutral	Hostile
Balanced	Frightened
Disengaged	Scornful
Collected	Scared
Cool-headed	Irritable
Restful	Upset
Unemotional	Loathing
Untroubled	Ashamed
Composed	Hopeless

This procedure was designed to parallel prior Affect Labeling research (see Tabibnia et al., 2008, for an example) that included a forced-choice between two words of different emotional content. In the AL-After group, each trial included labeling choice points presented in immediate succession (i.e., without a break between labeling decisions). Each trial included

three combinations of negative and neutral-valenced word options: 1) two negative affect options, 2) two neutral affect options, and 3) one negative and one neutral affect options. Immediately following the third labeling choice on a trial, participants were prompted to relax for a 60 s break.

Affect Labeling During Exposure (AL-During; $n=11$, $n=9$ completers). The trial order for this condition was as follows: exposure to the trauma script (30 s), imagination of the contents of the trauma script (10 s), one AL choice (5 s), continued imagination of the contents of the trauma script (10 s), a second AL choice (5 s), further imagination of the contents of the trauma script (10 s), a third AL choice (5 s), and finally recovery (60 s; see Figure 4 below).

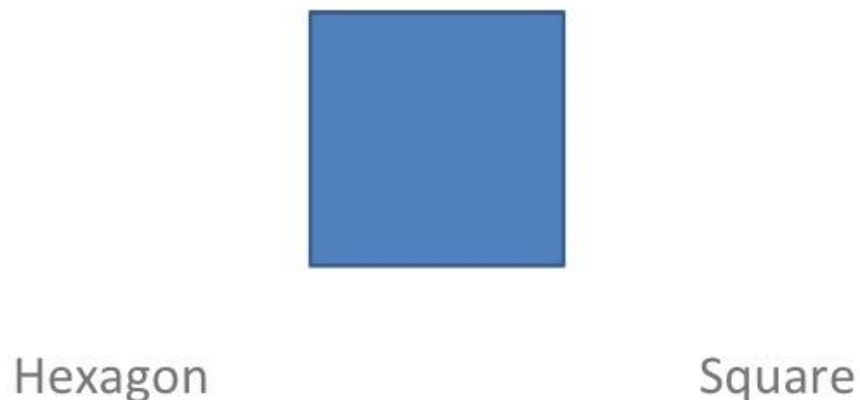
Figure 4. Labeling During imagination example.



The transition to imagination and labeling portions of the phase was prompted by two different tones presented through the headphones. As with the AL-After condition, the emotion labels were randomly chosen from the list of negative and neutral words, and the combination of negatively- and neutrally-valenced affect options was identical to the AL-After condition.

Shape Label After Exposure (SL-After; $n=10$, $n=7$ completers). The format of the SL-After Training was identical to the AL-After (see Figure 2), except for the content of the labeling task. These trials included the following: exposure to the trauma script (30 s), imagination of the contents of the trauma script (30 s), three SL decision points each lasting up to 5 s for a total of 15 s, and recovery (60 s). The SL groups were presented with a shape in the middle of the screen and two shape words at the bottom of the screen. Participants were prompted to choose the word from the two provided at the bottom that described the shape image in the middle of the screen (see Figure 5).

Figure 5. Example of SL decision.



The shapes presented to participants were randomly chosen from the list indicated in Table 3.

Table 3. Shape choices for SL task.

Shape Choices	
Circle	Star

Square	Trapezoid
Rectangle	Hexagon
Triangle	Diamond
Oval	Parallelogram

Shape Labeling During Exposure (SL-During; $n=10$, $n=8$ completers). The SL-During trials were identical to the AL-During trials (see Figure 4), except for the content of the labeling task. The SL-During trials included the following: exposure to the trauma script (30 s), imagination of the contents of the trauma script (10 s), one SL choice (5 s), continued imagination of the contents of the trauma script (10 s), a second SL choice (5 s), further imagination of the contents of the trauma script (10 s), a third SL choice (5 s), and finally recovery (60 s).

Procedure

Participants were invited to participate in the study via email and phone call. Interested participants presented to the laboratory to complete the informed consent and an initial assessment, which included a demographics form, an experimenter-administered MINI, and study questionnaires.

Following the initial assessment, participants generated a written narrative describing their trauma in detail. Participants who experienced repeat traumas or multiple types of trauma were asked to focus on the trauma that was the most bothersome in the prior month. A standard script preparation form was used for script preparation. The experimenters included research

assistants who had at least a B.A. and were trained in clinical psychology. They were trained in providing cognitive behavioral therapy for anxiety disorders and were closely supervised by two licensed clinical psychologists (Michelle Craske, Ph.D., and Raphael Rose, Ph.D.). They also received in-depth training by Michelle Craske, Ph.D. to perform the procedures outlined. The research assistants completed several "mock" experiments to ensure protocol adherence.

Following completion of script generation, the participant took a brief break while the scripts were organized and audio recorded. The scripts were all 30 s-40 s in duration. Randomization to experimental condition occurred after script recording to reduce experimenter bias in the recording of the scripts. Following script recording, physiological electrodes were affixed to the participant.

Next, participants completed the baseline SDI-A. Upon completion of this assessment, participants began their respective SDI-T. These include 1) AL-During, 2) AL-After, 3) SL-After, and 4) SL-During. Then, the physiological electrodes were removed and Day 1 was complete.

Participants returned to the laboratory one week later for an additional 12 trials of their respective SDI-T. This break was included to allow for consolidation of inhibitory learning. Physiological responding was recorded throughout the Day 8 Training.

One week later, participants returned to the lab for their Post-Training assessment on Day 15. The assessment include the Post-SDI-A with two presentations of the trauma script and two presentations of the neutral script in counterbalanced order. SCL, heart rate, and EMG were collected during the SDI-A. As on the first assessment, two startle probes were presented during the imagery portion for half of the scripts of the SDI-A. Participants also completed a Post-

Training IES to measure changes in trauma levels, and a Post-Training CSQ to measure satisfaction.

Data Analytic Strategy. As stated in the introduction, the specific aims were as follows.

Specific aim 1. This study aimed to demonstrate that a brief imaginal exposure Training using the SDI-T procedure would result in significant reductions in trauma symptoms from Pre- to Post-Training. We hypothesized that there would be significant reductions in physiology and self-report symptoms over Time.

Specific aim 2. The study aimed to evaluate the degree to which labeling Task (Affect vs. Shape Labeling) reduces physiological and self-report distress. Therefore, AL was compared to a control condition, SL, from Pre- to Post-Training. We hypothesized that the AL group would demonstrate greater reductions in physiological and self-report responding to trauma cues compared to the SL group.

Specific aim 3. Within both AL and SL, the study compared Order of labeling (During vs. After the imagination phase of the SDI-T), as a predictor of outcome. We hypothesized that across both labeling conditions, labeling After imagination would result in greater reductions in physiological and self-report responding to trauma cues compared to the labeling During groups due to reduced distraction during the exposure, and hence increased ability to fully engage in the exposure.

Specific aim 4: This study compared the interaction of Task (Affect vs. Shape Labeling), Order (During vs. After imagination), and Time (Pre vs. Post-Training) on physiological responding. We hypothesized that the AL-After group would benefit the most for reasons described in the prior two aims.

To address specific aims 1-4, a series of analysis of variance (ANOVA) were run to test whether there were differences by experimental condition in baseline demographic, self-report, and physiology variables by Order, Task, and Order X Task. Non-significant interaction effects were removed, and main effects were reported in these instances. In the beginning of the pilot study, participants had a one-week break between Pre-SDI-A and the first Training. To reduce participant burden, these were combined into one visit. To confirm that there were no effects of study duration on outcome, a series of analysis of covariance (ANCOVA) were run using outcome variables on self-report and physiological indices as dependent variables, duration (3 vs 4 Days) as the independent variable, and baseline scores and Condition (Order X Task) as covariates.

In instances where there were baseline differences on an outcome variable, a series of ANCOVAs were run using Post-Training scores as the dependent variable, baseline scores as a covariate, and effects of Order, Task, and Order X Task as independent variables. As in the prior analyses, non-significant interaction terms were dropped from the model and rerun using only main effects.

For outcome variables where there were not significant baseline differences, a series of mixed multilevel models were run. Two level growth curve models were calculated using the mixed command in Stata 13 (StataCorp, 2013) using maximum likelihood to account for the autocorrelation among observations (see Appendix III for Intraclass Correlation, ICC). Level 1, Time (Pre- and Post-Training) was modeled as a continuous linear predictor. Level 2 included experimental condition. Random effects of intercepts were included in all models using homogeneous variance structures, but random slopes were not included as they were not significant. The model for Specific Aims 1-4 was as follows:

$$\begin{aligned} \tilde{z}_{it} &= \beta_0 + \beta_1 t + \beta_2 \text{Task} + \beta_3 \text{Order} + \beta_4 \text{Task} \times \text{Order} \\ &+ \beta_5 \text{Time} + \beta_6 \text{Time} \times \text{Task} + \beta_7 \text{Time} \times \text{Order} + \beta_8 \text{Time} \times \text{Task} \times \text{Order} \\ &+ \beta_9 \text{Task} \times \text{Order} + \beta_{10} \text{Task} \times \text{Time} + \beta_{11} \text{Task} \times \text{Time} \times \text{Order} \\ &+ \beta_{12} \text{Order} \times \text{Time} + \beta_{13} \text{Order} \times \text{Time} \times \text{Task} + \beta_{14} \text{Order} \times \text{Time} \times \text{Task} \times \text{Order} \\ &+ \beta_{15} \text{Task} \times \text{Time} \times \text{Order} \times \text{Task} + \beta_{16} \text{Task} \times \text{Time} \times \text{Order} \times \text{Task} \times \text{Order} \\ &+ \beta_{17} \text{Order} \times \text{Time} \times \text{Task} \times \text{Order} \times \text{Task} + \beta_{18} \text{Order} \times \text{Time} \times \text{Task} \times \text{Order} \times \text{Task} \times \text{Order} \end{aligned}$$

Where \tilde{z}_{it} represents the dependent variable for time t nested within individual i , β_0 represents time for an individual i , β_1 is the intercept of the dependent variable for individual i , and β_2 is the growth parameters for an individual i , β_3 is the Task (Shape =0 vs. Affect Labeling =1), β_4 is the Order (After =1 vs. During =0), β_5 is the Task X Order interaction, β_6 is an overall intercept (i.e., overall score on the dependent variable at Pre-SDI-A in SL-During), β_7 is the mean difference in the dependent variable between SL and AL at Pre-SDI-A for the During Condition, β_8 is the mean difference in the dependent variable between During and After at Pre-SDI-A for SL, β_9 is the mean effect of Order on the relationship between Task and the dependent variable at Pre-SDI-A, β_{10} is the mean effect of Time for SL-During, β_{11} is the mean difference in the Time effect for AL vs SL for the During condition, β_{12} is the mean difference in the Time effect for After vs. During in SL, β_{13} is the additional change in the Time effect related to the Task effect for After (i.e., the additional change in the slope of time when for SL to AL, or from During to After), β_{14} is the within-person error variance for person i at time t , and β_{15} is each person's deviation from the overall intercept controlling for Order and Task.

When there were not significant Time x Task X Order interaction, this parameter was removed from the model, as were any other non-significant interactions. Consistent with prior research, psychophysiological data collected during SDI-A was averaged across both trials for each script and calculated as the difference between the baseline phase and the script or imagination phase, respectively for all measures except fear potentiated startle EMG response,

which did not have a baseline (Shin et al., 2000). Intent to treat analyses generally offer a more conservative analysis compared to analyses of only treatment completers (Armijo-Olivo, Warren, & Magee, 2009; Gupta, 2011) and therefore all participants were including in analyses regardless of attrition status. Cohen's d (Cohen, 1992) and Hedges' g (Hedges, 1981, which takes sample size into account) effect size estimates were calculated as differences from Pre- to Post-SDI-A for measures where there were not baseline differences.

Specific aim 5. Finally, the pilot study compared choice of negative emotion versus neutral emotion words as a predictor of improved outcome. This was an exploratory aim, but we hypothesized that greater overall percentage of negative emotion word choices would result in greater reductions in physiology and self-reported distress compared to neutral emotion word choices.

Average emotion word choice across both days of experimentation was entered as a predictor of difference scores in self-report and physiology outcomes, alongside Order and the Order X Percent Emotion Words interaction, using hierarchical regression analyses.

Pilot Study Results

Baseline Characteristics

As demonstrated in Table 1, there were no significant differences on Gender or Ethnicity based on Order or Task. However, there was a marginally significant difference in Age based on Order ($F(1,38)=4.804, p=.05, \eta^2=.097$), as the After condition (mean=20.7, SD=4.3) was older than the During condition (mean=18.7, SD=.9). Therefore, Age was used as a covariate in all remaining analyses. There were significant differences on IES by the Order X Task interaction ($F(1,35)=5.034, p<.05$, see Appendix IV). While none of the pairwise comparisons

were significant, SL-During (mean=26.5, SD=15.6) and AL-After (mean=21.3, SD=12.0) were higher than SL-After (mean=13.7, SD=9.71) and AL-During (mean=16.3, SD=11.6). Therefore, baseline IES was also used as a covariate in all analyses of Pre- to Post-SDI-T changes. Similarly, baseline differences emerged in the same direction for PDS. There were no baseline differences on the BDI or DES (see Table 4).

Table 4. Baseline Differences in Self-Report Distress.

	Mean (SD)				Order (During v. After)	Task (Affect v. Shape)	Order X Task
	AL-During	SL-During	AL-After	SL-After			
DES	13.0 (8.6)	12.6 (8.0)	18.2 (10.6)	13.0 (8.6)	$F(1,38)=1.696$, $p=.201$, partial $\eta^2=.043$	$F(1,38)=.494$, $p=.486$, partial $\eta^2=.013$	$F(1,37)=.322$, $p=.574$, partial $\eta^2=.009$
BDI	8.5 (5.3)	11.6 (7.7)	9.3 (6.4)	10.7 (6.1)	$F(1,37)=.000$, $p=.994$, partial $\eta^2=.000$	$F(1,37)=1.314$, $p=.259$, partial $\eta^2=.000$	$F(1,36)=.192$, $p=.664$, partial $\eta^2=.005$
IES	16.3 (11.6)	26.3 (15.6)	21.3 (12.0)	13.7 (9.71)	$F(1,36)=.537$, $p=.469$, partial $\eta^2=.015$	$F(1,36)=.041$, $p=.841$, partial $\eta^2=.001$	$F(1,35)=5.034$, $p<.05$, partial $\eta^2=.126$
PDS	12.4 (9.8)	18.2 (9.3)	20.5 (10.8)	12.9 (9.3)	$F(1,37)=.285$, $p=.596$, partial $\eta^2=.008$	$F(1,37)=.076$, $p=.784$, partial $\eta^2=.002$	$F(1,36)=4.65$, $p<.05$, partial $\eta^2=.114$

In terms of physiological measures, there were no significant baseline differences by Condition in any of the following measures: SCL, HR during the trauma script, F-EMG during the trauma script, imagination, and neutral script, and EMG during neutral imagination. There were baseline differences in HR during trauma imagination, neutral script, and neutral imagination. Whereas none of the post-hoc tests were significant, AL-During and SL-After were significant higher on all HR indices (see Table 6 below). There were also significant differences in F-EMG during neutral imagination and EMG during trauma imagination with both During

conditions lower than After conditions (though none of the pairwise comparisons were significant).

Table 6. Baseline Physiology	AL-During	SL-During	AL-After	SL-After	Order (During v. After)	Task (Affect v. Shape)	Order X Task
HR Trauma Script	4.3 (3.6)	3.4 (5.2)	1.3 (2.9)	4.3 (3.0)	$F(1,35)=.668, p=.419,$ partial $\eta^2=.019$	$F(1,35)=.714, p=.404,$ partial $\eta^2=.020$	$F(1,34)=2.596, p=.116,$ partial $\eta^2=.071$
HR Trauma Imagine	4.1 (4.6)	1.0 (3.8)	.8 (2.7)	3.5 (3.3)	$F(1,34)=.151, p=.700,$ partial $\eta^2=.004$	$F(1,34)=.033, p=.858,$ partial $\eta^2=.001$	$F(1,34)=5.862, p<.05,$ partial $\eta^2=.147$
HR Neutral Script	3.9 (3.2)	1.2 (3.5)	.6 (2.2)	4.5 (3.3)	$F(1,34)=.000, p=.999,$ partial $\eta^2=.000$	$F(1,34)=.376, p=.544,$ partial $\eta^2=.011$	$F(1,34)=10.797, p<.01,$ partial $\eta^2=.241$
HR Neutral Imagine	2.8 (5.6)	-0.8(1.9)	-8 (3.0)	1.1 (2.5)	$F(1,34)=.63, p=.433,$ partial $\eta^2=.018$	$F(1,34)=.559, p=.460,$ partial $\eta^2=.016$	$F(1,34)=6.033, p<.05,$ partial $\eta^2=.151$
SCL Trauma Script	3.0 (3.3)	1.3 (2.3)	2.4 (2.0)	2.4 (3.0)	$F(1,34)=.067, p=.798,$ partial $\eta^2=.002$	$F(1,34)=.866, p=.359,$ partial $\eta^2=.025$	$F(1,33)=.864, p=.359,$ partial $\eta^2=.026$
SCL Trauma Imagine	2.2 (3.0)	.9 (1.8)	2.1 (2.0)	2.1 (2.7)	$F(1,33)=.303, p=.586,$ partial $\eta^2=.009$	$F(1,34)=.953, p=.336,$ partial $\eta^2=.027$	$F(1,33)=.810, p=.375,$ partial $\eta^2=.024$
SCL Neutral Script	-6.5 (4.5)	-4.5 (3.4)	-4.0 (3.9)	-3.0 (3.7)	$F(1,29)=2.167, p=.152,$ partial $\eta^2=.070$	$F(1,29)=1.169, p=.152,$ partial $\eta^2=.070$	$F(1,28)=.153, p=.699,$ partial $\eta^2=.005$
SCL Neutral Imagine	1.5 (3.0)	1.5 (3.0)	1.9 (2.0)	3.08 (4.08)	$F(1,34)=2.616, p=.115,$ partial $\eta^2=.071$	$F(1,34)=.015, p=.903,$ partial $\eta^2=.000$	$F(1,33)=1.900, p=.177,$ partial $\eta^2=.054$
F-EMG Trauma Script	.9 (10.7)	.4(8.2)	-2.8 (8.1)	.2 (7.3)	$F(1,27)=.409, p=.528,$ partial $\eta^2=.015$	$F(1,27)=.156, p=.696,$ partial $\eta^2=.006$	$F(1,26)=.310, p=.582,$ partial $\eta^2=.012$
F-EMG Trauma Imagine	3.7 (10.9)	1.5 (11.6)	-3.9 (11.7)	6.9 (8.4)	$F(1,27)=.070, p=.793,$ partial $\eta^2=.003$	$F(1,27)=1.132, p=.297,$ partial $\eta^2=.040$	$F(1,26)=2.698, p=.113,$ partial $\eta^2=.094$
F-EMG Neutral Script	.4 (4.8)	-3.0 (5.5)	-6.0 (6.5)	-1.9 (6.2)	$F(1,27)=1.467, p=.236,$ partial $\eta^2=.052$	$F(1,27)=.031, p=.861,$ partial $\eta^2=.001$	$F(1,26)=3.082, p=.091,$ partial $\eta^2=.106$
F-EMG Neutral Imagine	-5.1 (5.0)	-3.9 (4.3)	1.3 (4.4)	-.6 (6.5)	$F(1,26)=.679, p=.417,$ partial $\eta^2=.025$	$F(1,26)=.043, p=.838,$ partial $\eta^2=.002$	$F(1,26)=6.780, p<.05,$ partial $\eta^2=.207$
EMG Integral Trauma	1.3 (.4)	.9 (.3)	.7 (.5)	.9 (.4)	$F(1,36)=4.469, p<.05,$ partial $\eta^2=.110$	$F(1,36)=.747, p=.393,$ partial $\eta^2=.020$	$F(1,35)=3.249, p=.08,$ partial $\eta^2=.085$
EMG Amplitude Trauma	1.7 (.4)	1.5 (.4)	1.3 (.5)	1.3 (.4)	$F(1,36)=5.307, p<.05,$ partial $\eta^2=.128$	$F(1,36)=.206, p=.652,$ partial $\eta^2=.006$	$F(1,35)=.120, p=.731,$ partial $\eta^2=.003$
EMG Integral Neutral	1.1 (.4)	1.0 (.2)	.8 (.3)	1.0 (.4)	$F(1,35)=3.023, p=.091,$ partial $\eta^2=.080$	$F(1,35)=.021, p=.885,$ partial $\eta^2=.001$	$F(1,34)=1.661, p=.206,$ partial $\eta^2=.047$
EMG Amplitude Neutral	1.5 (.4)	1.5 (.3)	1.3 (.4)	1.3 (.4)	$F(1,35)=3.060, p=.089,$ partial $\eta^2=.080$	$F(1,35)=.072, p=.789,$ partial $\eta^2=.002$	$F(1,34)=.000, p=1.00,$ partial $\eta^2=.000$

None of the Post-SDI-A physiology measures significantly differed whether participants had three or four total days of experimentation, controlling for Pre-SDI-A score, Task, and Order of labeling. Those who completed only 3 days of the study had lower IES at Post-SDI-A, and therefore timing of study duration was entered as a covariate in this analysis (see Table 7).

Table 7. Differences in outcome based on 3 or 4 days of study duration.

Post-SDI-A Measure	3 vs. 4 days of Experiment
IES	$F(1,27)=4.348, p<.05, \text{partial } \eta^2=.139$
BDI	$F(1,29)=1.663, p=.207, \text{partial } \eta^2=.054$
HR Trauma Script	$F(1,23)=1.483, p=.236, \text{partial } \eta^2=.061$
HR Trauma Imagine	$F(1,23)=1.197, p=.285, \text{partial } \eta^2=.049$
HR Neutral Script	$F(1,23)=.776, p=.388, \text{partial } \eta^2=.033$
HR Neutral Imagine	$F(1,23)=.923, p=.347, \text{partial } \eta^2=.039$
SCL Trauma Script	$F(1,21)=.497, p=.488, \text{partial } \eta^2=.023$
SCL Trauma Imagine	$F(1,22)=.487, p=.493, \text{partial } \eta^2=.023$
SCL Neutral Script	$F(1,21)=.376, p=.546, \text{partial } \eta^2=.018$
SCL Neutral Imagine	$F(1,21)=1.004, p=.328, \text{partial } \eta^2=.046$
F-EMG Trauma Script	$F(1,14)=.064, p=.804, \text{partial } \eta^2=.005$
F-EMG Trauma Imagine	$F(1,14)=1.062, p=.320, \text{partial } \eta^2=.071$
F-EMG Neutral Script	$F(1,14)=3.331, p=.089, \text{partial } \eta^2=.192$
F-EMG Neutral Imagine	$F(1,14)=1.501, p=.241, \text{partial } \eta^2=.097$
EMG Integral Trauma	$F(1,27)=.064, p=.803, \text{partial } \eta^2=.002$
EMG Amplitude Trauma	$F(1,27)=.061, p=.807, \text{partial } \eta^2=.002$
EMG Integral Neutral	$F(1,26)=.001, p=.981, \text{partial } \eta^2=.000$
EMG Amplitude Neutral	$F(1,26)=.945, p=.340, \text{partial } \eta^2=.035$

Post-SDI-A Results Controlling for Pre-SDI-A Scores

Given the baseline differences in trauma severity, as measured by the IES, and in Age, these variables were used as covariates throughout, and duration of study (3 vs 4 Days) was used as a covariate for IES. The analyses in this section only include those for which there were baseline differences by either Order, Task, or Order X Task. These ANOVAs include the Post-SDI-A score as the dependent variable with the Pre-SDI-A score as a covariate (alongside IES, Age, and duration of study for IES only). When the Order X Task effect was not significant, it was removed from the model. There were no significant effects of Order, Task, or Order X Task on any of these variables (see Table 8). All CSQ scores were high (mean=26.5, SD= 4.0, range 25-28.5, with a maximum score of 32).

Table 8. Differences in Post-SDI-A responses by Order and Task

Outcome	Order (During v. After)	Task (Affect v. Shape)	Order X Task
IES	$F(1,25)=.036$, $p=.851$, partial $2=.001$	$F(1,25)=.000$, $p=.983$, partial $2=.000$	$F(1,24)=.416$, $p=.525$, partial $2=.017$
CSQ	$F(1,19)=.174$, $p=.681$, partial $2=.009$	$F(1,19)=.145$, $p=.708$, partial $2=.008$	$F(1,18)=.937$, $p=.346$, partial $2=.049$
HR Trauma Imagine (Appendix V)	$F(1,19)=.621$, $p=.440$, partial $2=.032$	$F(1,19)=.470$, $p=.501$, partial $2=.024$	$F(1,18)=3.764$, $p=.068$, partial $2=.173$
HR Neutral Script (Appendix VI)	$F(1,19)=.483$, $p=.495$, partial $2=.025$	$F(1,19)=.861$, $p=.365$, partial $2=.043$	$F(1,18)=.503$, $p=.485$, partial $2=.027$

HR Neutral Imagine (Appendix VII)	$F(1,19)=.093$, $p=.764$, partial $\eta^2=.005$	$F(1,19)=.668$, $p=.424$, partial $\eta^2=.034$	$F(1,18)=.672$, $p=.423$, partial $\eta^2=.036$
F-EMG Neutral Imagine (Appendix VIII)	$F(1,11)=.136$, $p=.720$, partial $\eta^2=.012$	$F(1,11)=.260$, $p=.620$, partial $\eta^2=.023$	$F(1,10)=.000$, $p=.996$, partial $\eta^2=.000$
EMG Integral Trauma (Appendix IX)	$F(1,23)=.388$, $p=.540$, partial $\eta^2=.017$	$F(1,23)=.036$, $p=.852$, partial $\eta^2=.002$	$F(1,22)=1.135$, $p=.298$, partial $\eta^2=.049$
EMG Amplitude Trauma (Appendix X)	$F(1,23)=.339$, $p=.566$, partial $\eta^2=.015$	$F(1,23)=.357$, $p=.556$, partial $\eta^2=.015$	$F(1,22)=.442$, $p=.513$, partial $\eta^2=.020$

Note: Analyses in Table 8 included Pre-SDI-A scores as a covariate, alongside baseline IES and Age. For the IES analysis, study duration (3 vs. 4 visits) was covaried.

Mixed Model Results

There was a significant effect of Time ($z = -3.73, p < .001$; Cohen's $d = .391$, Hedges' $g = .385$), a significant Order X Task effect on BDI ($z = -2.14, p < .05$), and a significant lower-order effect of Order ($z = 2.78, p < .001$) on BDI, with the After group significantly higher than the During group overall. Participants in the SL condition had significantly lower BDI overall in During compared to After ($p < .01$), whereas there were no differences in BDI in the AL group based on Order ($p = .99$; see Figures 6 and 7 below). Only the slope for SL-During was significantly different ($p < .01$), but none of the slopes significantly differed from each other (all $ps > .05$).

Figure 6. BDI from Pre- to Post-SDI-A.

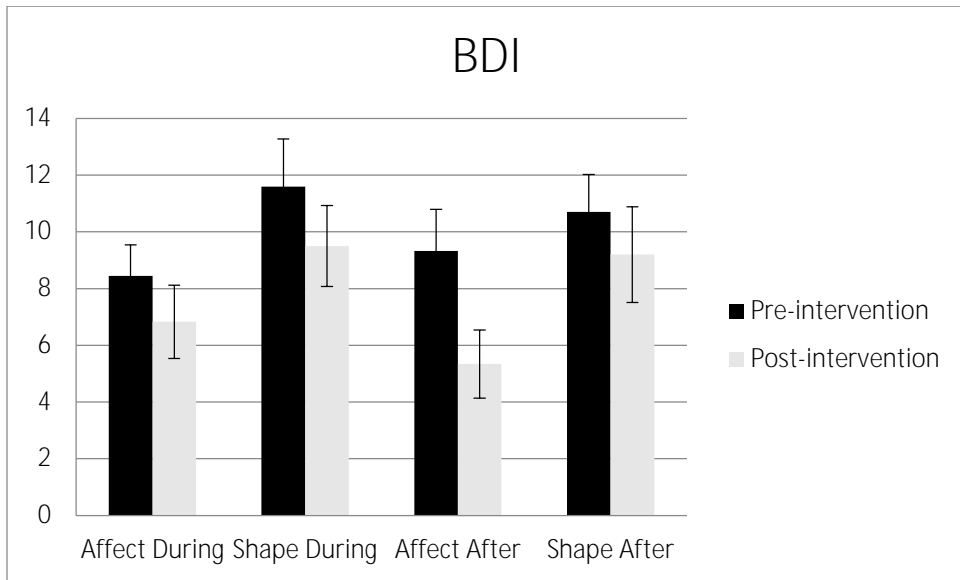
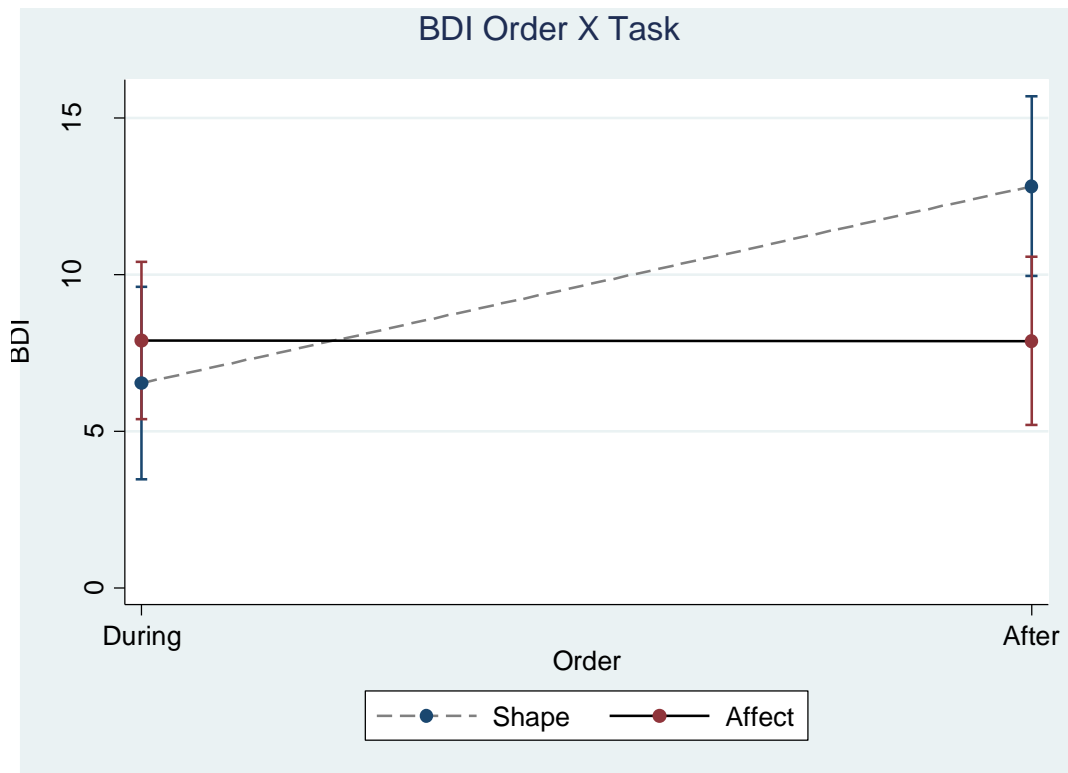
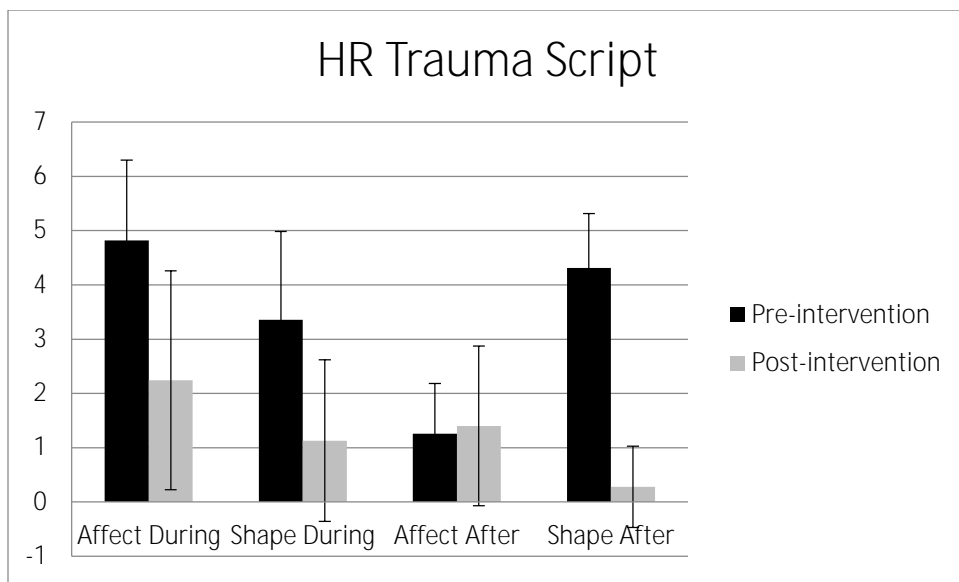


Figure 7. Order X Task Interaction on BDI.



There were no significant effects of the Order X Task X Time interactions on HR during the trauma script. However, there was a significant reduction in HR to the trauma script from Pre- to Post-SDI-A ($z: -2.32, p < .05$; Cohen's $d = .51$, Hedges' $g = .5012$; see Figure 8). Only the slope for SL-After was significant ($p < .05$), but there were no differences by condition in slope (all $ps > .05$).

Figure 8. HR from Pre- to Post-SDI-A.



There was a significant effect of Time X Task X Order on F-EMG during trauma imagine ($z = 1.97, p < .05$; see Figures 9 & 10). There were no significant lower-order effects. Only SL-After had a significant simple slope ($p < .05$), but there were no significant differences in simple slopes by Order. There were significant differences in slope between SL and AL in the After condition, with SL having a significantly negative slope over time ($p < .01$) that was not present in AL, but there were no differences by Condition for the slope in the During Labeling group ($p = .88$).

Figure 9. F-EMG Time X Order in Shape Labeling.

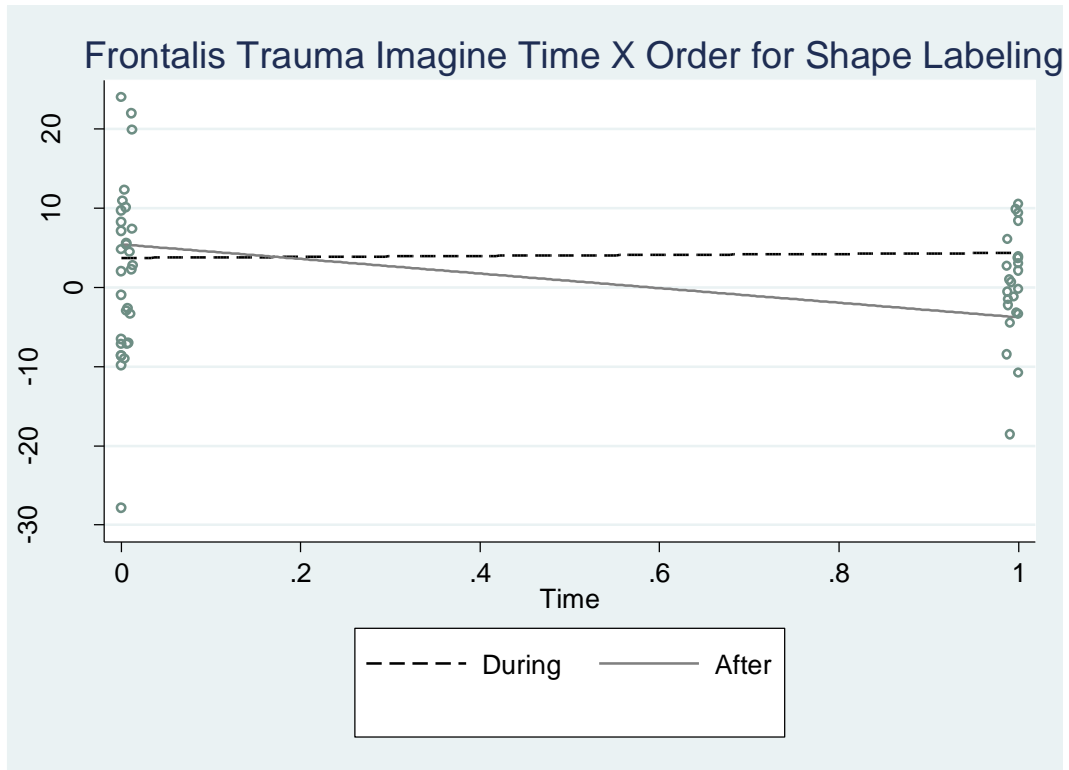
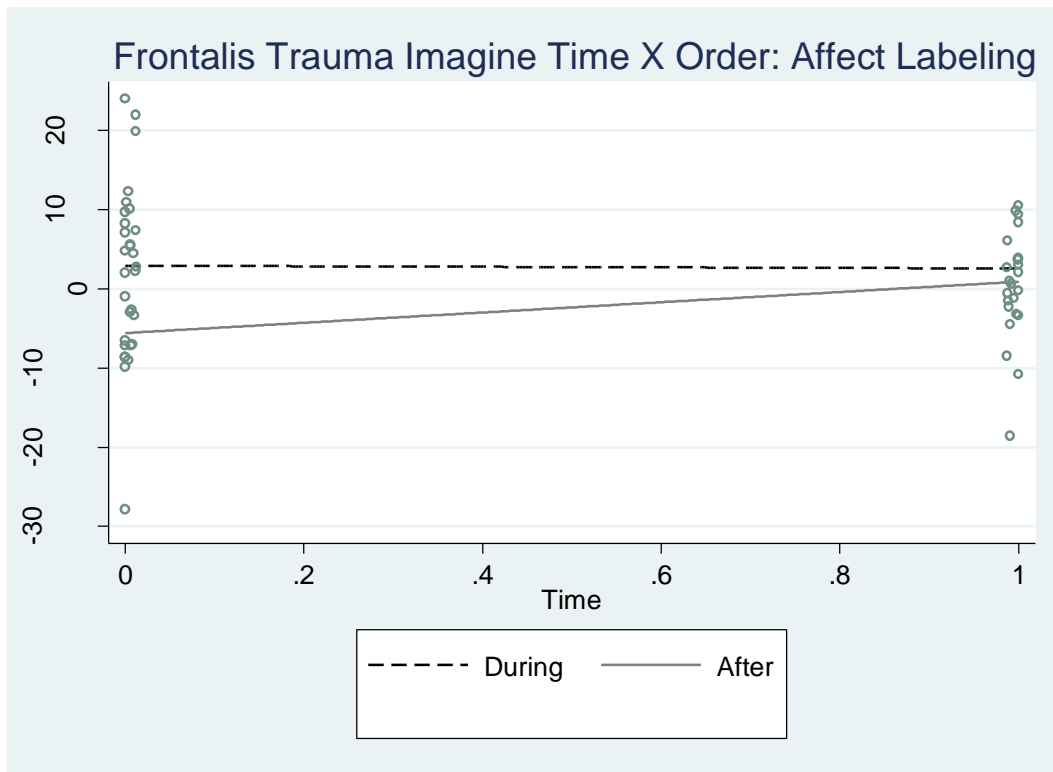
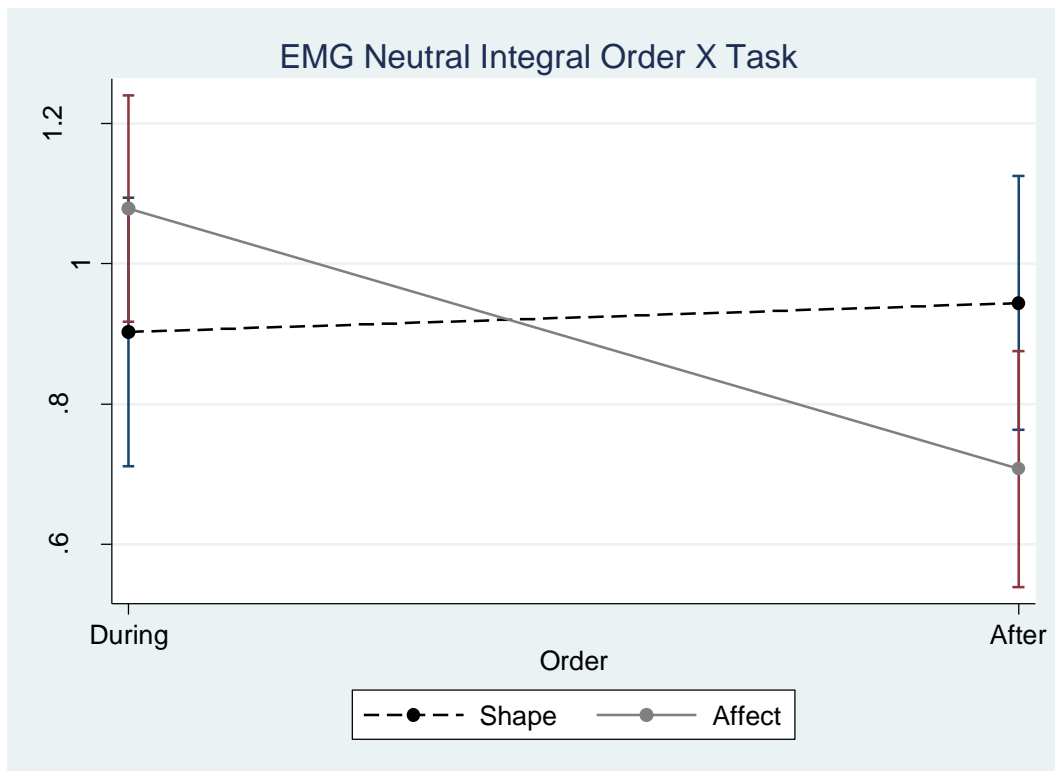


Figure 10. F-EMG Time X Order in Affect Labeling.



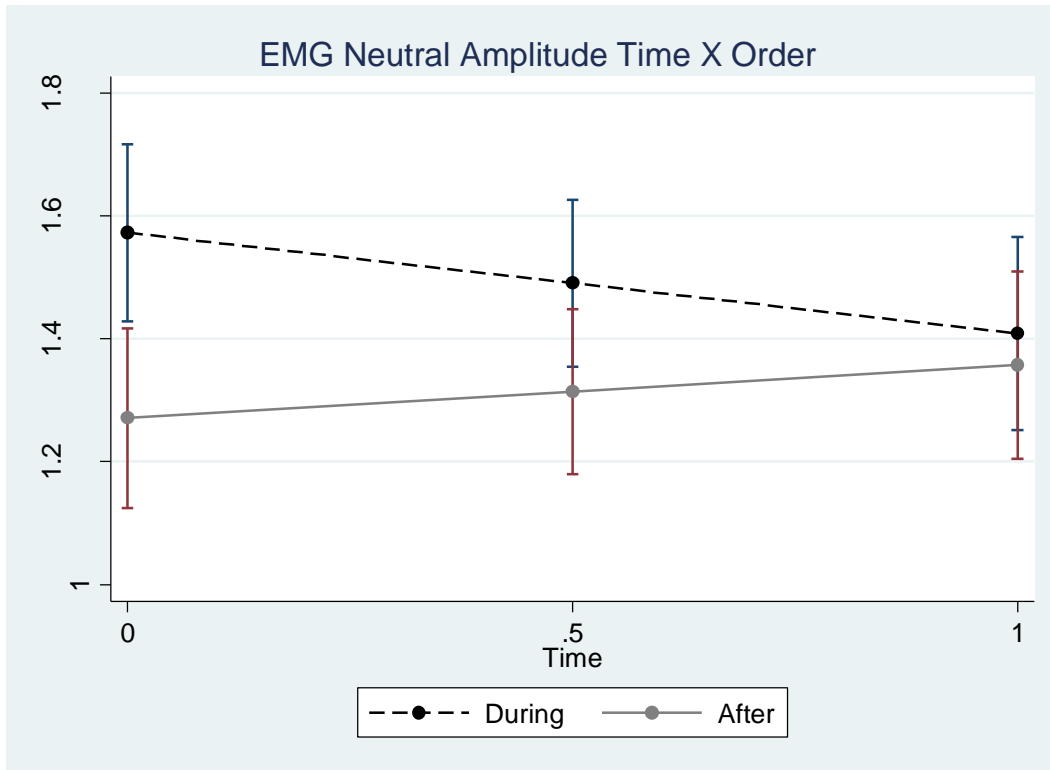
There was also a significant effect of Order X Task on EMG Neutral Integral ($z = -2.27$, $p < .05$; see Figure 11). In the AL condition, there were significant differences in the During vs. After comparison ($p < .0001$), with the After condition significantly lower overall, but there were not differences based on Order in the SL condition ($p = .69$).

Figure 11. EMG neutral integral Order X Task Interaction.



Finally, there was a significant effect of the Time X Order interaction ($z = 2.66$, $p < .01$; see Figure 12) and a significant main effect of Order ($z = -.286$, $p < .01$) on EMG Neutral Amplitude. Only the During group had a simple slope significantly different than zero ($p < .05$). The slopes were significantly different based on Order ($p < .01$), with the After slope positive and the During slope negative. However, this may have been driven by a trend toward non-significant baseline differences.

Figure 12: EMG neutral amplitude Time X Order interaction.



There were no significant effects of Time, Task, Order, or their interactions on SCL during the trauma script, trauma imagination, or neutral imagination, F-EMG during the trauma or neutral script, or EMG amplitude during the trauma script (see Table 10). Graphs demonstrating mean values are available in Appendix IV- Appendix XIX. SCL during the neutral script significantly increased over Time (Parameter estimate: 5.94 SE: 1.05, z : 5.64, $p < .000$, Cohen's $d = -1.54$, Hedges' $g = -1.52$, see Appendix XIII), though this may have been due to a trend toward baseline differences. Especially low baseline responding on this measure may have been caused by relief relative to the trauma script. The simple slopes for all groups were all significantly positive (all p s $< .05$) but there were no differences by either Order or Task in slope.

Table 10: Pre- to Post-SDI-A changes by Order and Task.

Outcome	Time	Order (During vs. After)	Task (Affect vs. Shape)	Order X Task	Time X Order	Time X Task	Time X Order X Task
BDI	Parameter estimate: -2.70, SE: .73, z: -3.73, $p < .001$; # d : .39, = $g = .39$	Parameter estimate: 6.28, SE: 2.25, z: 2.78, $p < .001$	Parameter estimate: 1.36, SE: 2.04, z: .67, $p = .505$	Parameter estimate: -6.29, SE: 2.94, z: -2.14, $p < .05$	Parameter estimate: .899, SE: 1.44, z: .62, $p = .53$	Parameter estimate: .53, SE: 1.46, z: .37, $p = .714$	Parameter estimate: -2.94, SE: 2.86, z: -1.03, $p = .30$
HR Trauma Script	Parameter estimate: -2.11, SE: .91, z: -2.32, $p < .05$; # d : .51, = $g = .5012$	Parameter estimate: -2.15, SE: 1.14, z: -1.89, $p = .059$	Parameter estimate: -.65, SE: 1.07, z: -.61, $p = .54$	Parameter estimate: -.89, SE: 2.22, z: -.40, $p = .687$	Parameter estimate: .87, SE: 1.82, z: .48, $p = .633$	Parameter estimate: 2.10, SE: 1.80, z: 1.16, $p = .244$	Parameter estimate: 5.14, SE: 3.38, z: 1.52, $p = .129$
SCL Trauma Script (Appendix XI)	Parameter estimate: -.99, SE: .78, z: -1.26, $p = .21$, Cohen's $d = .29$, Hedges' $g = .29$	Parameter estimate: -.48, SE: .87, z: -.55, $p = .58$	Parameter estimate: .67, SE: .82, z: .82, $p = .41$	Parameter estimate: -1.78, SE: 1.67, z: -1.08, $p = .29$	Parameter estimate: -1.08, SE: 1.58, z: -.69, $p = .49$	Parameter estimate: .48, SE: 1.57, z: .31, $p = .76$	Parameter estimate: -1.60, SE: 3.15, z: -.51, $p = .61$
SCL Trauma Imagine (Appendix XII)	Parameter estimate: -.756, SE: .696, z: -1.09, $p = .28$	Parameter estimate: -.19, SE: .81, z: -.24, $p = .81$	Parameter estimate: .54, SE: .77, z: .71, $p = .48$	Parameter estimate: -1.74, SE: 1.57, z: -1.11, $p = .27$	Parameter estimate: -.87, SE: 1.41, z: -.62, $p = .54$	Parameter estimate: .13, SE: 1.41, z: .09, $p = .93$	Parameter estimate: -1.46, SE: 2.81, z: -.52, $p = .60$

SCL Neutral Script (Appendix XIII)	Parameter estimate: 5.94 SE: 1.05, z: 5.64, $p < .000$, # $d = -1.54$, = $g = -1.52$	Parameter estimate: 1.40, SE: 1.21, z: 1.16, $p = .25$	Parameter estimate: -.28, SE: 1.09, z: -.25, $p = .80$	Parameter estimate: .82, SE: 2.28, z: .36, $p = .72$	Parameter estimate: -2.54, SE: 2.09, z: -1.21, $p = .23$	Parameter estimate: 1.46, SE: 2.11, z: .69, $p = .49$	Parameter estimate: -1.43, SE: 4.2, z: -.34, $p = .73$
SCL Neutral Imagine (Appendix XIV)	Parameter estimate: -.976, SE: .779, z: -1.25, $p = .21$, Cohen's $d = .24$, Hedges' $g = .24$	Parameter estimate: 1.09, SE: .90, z: 1.21, $p = .23$	Parameter estimate: .14, SE: .86, z: .16, $p = .88$	Parameter estimate: -1.36, SE: 1.77, z: -.77, $p = .44$	Parameter estimate: -.73, SE: 1.58, z: -.46, $p = .65$	Parameter estimate: .67, SE: 1.56, z: .43, $p = .67$	Parameter estimate: 2.43, SE: 3.10, z: .78, $p = .43$
F-EMG Trauma Script (Appendix XV)	Parameter estimate: -1.24 SE: 1.76, z: -.71, $p = .48$, Cohen's $d = .12$, Hedges' $g = .12$	Parameter estimate: -2.81, SE: 2.48, z: -1.14, $p = .26$	Parameter estimate: -.40, SE: 2.33, z: -.17, $p = .86$	Parameter estimate: -.11, SE: 4.84, z: -.02, $p = .98$	Parameter estimate: .99, SE: 3.54, z: .28, $p = .78$	Parameter estimate: 3.18, SE: 3.47, z: .92, $p = .34$	Parameter estimate: 2.74, SE: 7.03, z: .39, $p = .70$
F-EMG Trauma Imagine (Appendix XVI)	Parameter estimate: .625 SE: 5.03, z: .12, $p = .90$, Cohen's $d = .16$, Hedges' $g = .15$	Parameter estimate: 1.71, SE: 4.74, z: .36, $p = .72$	Parameter estimate: -.799, SE: 4.53, z: -.18, $p = .86$	Parameter estimate: -10.23, SE: 6.37, z: -1.60, $p = .11$	Parameter estimate: -9.85, SE: 6.27, z: -1.57, $p = .12$	Parameter estimate: -.97, SE: 6.43, z: -.15, $p = .88$	Parameter estimate: 16.68, SE: 8.45, z: 1.97, $p < .05$
F-EMG Neutral Script (Appendix XVII)	Parameter estimate: .428 SE: 1.42, z: .30, $p = .763$, Cohen's	Parameter estimate: -3.66, SE: 1.90, z: -1.92, $p = .05$	Parameter estimate: .663, SE: 1.79, z: .37, $p = .71$	Parameter estimate: -3.33, SE: 3.68, z: -.91, $p = .37$	Parameter estimate: 1.68, SE: 2.82, z: .60, $p = .55$	Parameter estimate: 4.27, SE: 2.71, z: 1.57, $p = .12$	Parameter estimate: 4.04, SE: 5.42, z: .75, $p = .46$

	$d=.05$, Hedges' $g=.05$						
EMG Integral Neutral (Appendix XVIII)	Parameter estimate: $-.06$ SE: $.06$, $z: -1.05$, $p=.29$, Cohen's $d=.12$, Hedges' $g=.12$	Parameter estimate: $.04$, SE: $.140$, $z: .29$, $p=.77$	Parameter estimate: $.18$, SE: $.13$, $z: 1.38$, $p=.17$	Parameter estimate: $-.41$, SE: $.18$, $z: -2.27$, $p<.05$	Parameter estimate: $.21$, SE: $.12$, $z: 1.80$, $p=.07$	Parameter estimate: $-.11$, SE: $.12$, $z: -.96$, $p=.34$	Parameter estimate: $.18$, SE: $.31$, $z: .54$, $p=.59$
EMG Amplitude Neutral (Appendix XIX)	Parameter estimate: $-.16$ SE: $.07$, $z: -2.45$, $p<.05$, # $d=.05$, = $g=.05$	Parameter estimate: $-.31$, SE: $.11$, $z: -.29$, $p<.01$	Parameter estimate: $-.10$, SE: $.10$, $z: -1.06$, $p=.29$	Parameter estimate: $-.21$, SE: $.20$, $z: -1.06$, $p=.29$	Parameter estimate: $.25$, SE: $.09$, $z: 2.66$, $p<.01$	Parameter estimate: $-.10$, SE: $.10$, $z: -.97$, $p=.33$	Parameter estimate: $-.195$, SE: $.187$, $z: -1.05$, $p=.30$

Word Choice

Word choice was analyzed for participants in the AL-During and AL-After conditions. The average number of valid word choice responses on the trials including a neutral and negative option during Days 1 and 8 of the Training was 10.12 (SD=2.99) and 10.44, (SD=3.05), respectively out of 12 trials. Accounting for the overall number of valid choices during SDI-T, participants chose negative emotion words over neutral emotion words on 51.0% (SD=24.2%) of trials on Training 1 and on 52.4% (SD=33.8%) of trials on Training 2.

Average percent of emotion word choices across both days of the Training were predictive of reduction in EMG Amplitude during trauma imagination ($=-.546, t=-2.350, p<.05$, Adjusted $R^2=.295$) and a reduction in EMG Integral during trauma imagination ($=-.566, t=-2.393, p<.05$, Adjusted $R^2=.316$) such that those who chose more emotion words demonstrated a greater reduction in EMG. Percentage of emotion word choices was not a significant predictor of reductions in any other outcome variable (see Table 11 below).

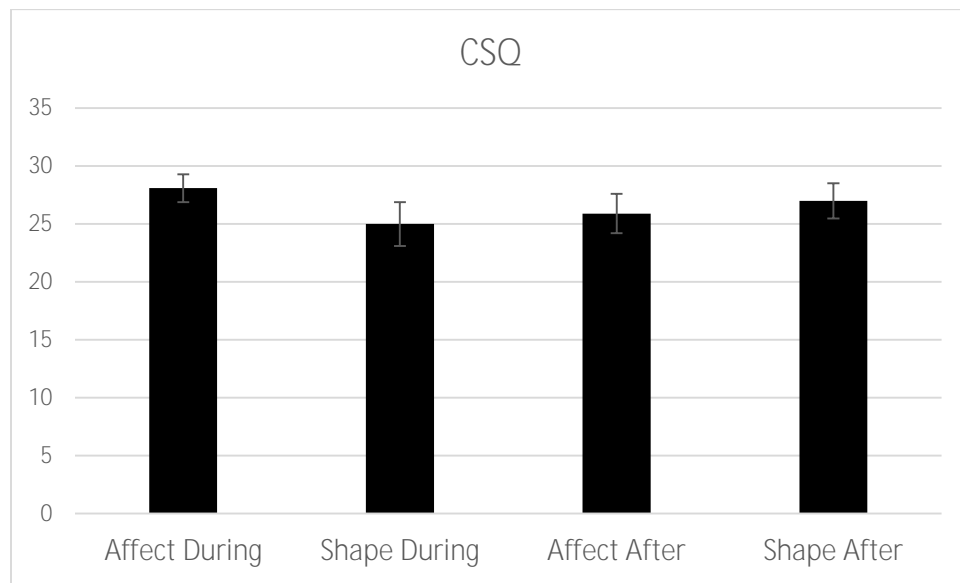
Table 11: Word Choice as a predictor of training response.

Outcome (Change Score)	% Neg Emotion Words	Order (During vs. After)	Emotion Words X Order
IES	$=-.315, t=-1.770, p=.087$, Adjusted $R^2=.100$	$=-.009, t=-.050, p=.962$, Adjusted $R^2=.000$	$=-.170, t=-.310, p=.757$, Adjusted $R^2=.003$
BDI	$=-.166, t=-.900, p=.377$, Adjusted $R^2=.027$	$=.000, t=.000, p=.998$, Adjusted $R^2=.000$	$=.019, t=.030, p=.974$, Adjusted $R^2=.019$
HR Trauma Script	$=-.385, t=-1.342, p=.213$, Adjusted $R^2=.137$	$=.525, t=1.829, p=.101$, Adjusted $R^2=.177$	$=.466, t=.431, p=.678$, Adjusted $R^2=.016$
HR Trauma Imagine	$=-.255, t=-1.254, p=.241$, Adjusted $R^2=.060$	$=.840, t=4.125, p<.01$, Adjusted $R^2=.594$	$=.726, t=.992, p=.350$, Adjusted $R^2=.038$
SCL Trauma Script	$=.200, t=.675, p=.513$, Adjusted $R^2=.039$	$=-.174, t=-.590, p=.567$, Adjusted $R^2=.021$	$=-.022, t=-.025, p=.981$, Adjusted $R^2=.000$
SCL Trauma Imagine	$=.178, t=.601, p=.560$, Adjusted $R^2=.031$	$=-.186, t=-.629, p=.542$, Adjusted $R^2=.026$	$=.064, t=.315, p=.760$, Adjusted $R^2=.000$

EMG Trauma Amplitude	$-.546, t=-2.350, p<.05,$ Adjusted $R^2=.295$	$=.315, t=1.358, p=.200,$ Adjusted $R^2=.065$	$=.005, t=.007, p=.995,$ Adjusted $R^2=.000$
EMG Trauma Integral	$-.566, t=-2.393, p<.05,$ Adjusted $R^2=.316$	$=.210, t=.890, p=.391,$ Adjusted $R^2=.022$	$=.370, t=.512, p=.619,$ Adjusted $R^2=.015$
F-EMG Trauma Script	$=.591, t=1.673, p=.145,$ Adjusted $R^2=.305$	$=-.009, t=-.025, p=.981,$ Adjusted $R^2=.041$	$=.624, t=.328, p=.756,$ Adjusted $R^2=.014$
F-EMG Trauma Imagine	$=.710, t=2.430, p=.051,$ Adjusted $R^2=.440$	$=.083, t=.283, p=.787,$ Adjusted $R^2=.113$	$=.899, t=.584, p=.584,$ Adjusted $R^2=.029$

Participant Satisfaction

There were no differences in satisfaction ratings based on Order ($F(1,21)=.21, p=.65$), Task ($F(1,21)=.76, p=.395$), or their Order X Task interaction ($F(1,20)=1.47, p=.2402, R^2=.068$). However, all satisfaction ratings were high (range 25-28.1 out of a possible 32).



Pilot Study Discussion

A variety of baseline differences in self-reported and physiological trauma severity preclude firm conclusions above the benefits of labeling Task, Order, or the interaction between Order and Task. However, consistent with our hypotheses, there were significant reductions over the course of the training on self-reported and physiological measures of distress.

Specifically, there were significant reductions in depression severity, HR during the trauma script, and EMG Neutral Amplitude over time. Surprisingly, there was a significant increase in SCL during the neutral script over time, though this might have been driven by a trend toward baseline differences.

Contrary to our hypotheses, there was only one significant difference over Time in trauma reactivity by Task (i.e., Shape vs. Affect labeling) in terms of F-EMG during trauma imagination, but this effect favored Shape Labeling. Post-hoc tests of significant Time effects (in the absence of Task effects) revealed benefits in Shape Labeling, but not Affect Labeling, for both BDI and HR during the trauma script. This was unexpected given the prior literature supporting the utility of affect labeling in other anxiety populations. However, the opportunity to detect strong differences by labeling type was confounded by baseline differences across a variety of measures and by a small sample size. In terms of the Order of labeling effects over time, the only significant differences that emerged before post-hoc testing were for EMG Amplitude during the neutral script with benefits in the During condition. While the F-EMG during trauma imagination had a significant three-way interaction between Time, Order, and Task, there were not significant differences in slope between During and After, though only the Shape Labeling After condition had a significant slope. Post-hoc tests demonstrated a benefit in the Shape Labeling group for the During condition on BDI and the After condition on HR during the trauma script. These post-hoc analyses should be interpreted with caution given that there were not omnibus differences by Order in slope for those tests. Therefore, the only significant omnibus result for Order demonstrates that the During condition performed better than the After condition. The direction of these effects were contrary to the hypothesis that the After condition

would result in improved performance due to reduced distraction. Instead, labeling in the middle of the emotionally evocative stimulus was more beneficial.

There were significant interactions between type of labeling (i.e., Task) and timing of labeling on depression severity. Specifically, those in the Shape Labeling condition reported lower overall depression if their labeling occurred During, as opposed to After, imagination. In contrast, the timing of labeling did not dictate depression severity for those in Affect Labeling. For another outcome variable, EMG Integral during neutral imagination, those in Affect Labeling had lower reactivity overall in the After compared to During group, but the timing of labeling did not determine responding for those in Shape Labeling.

Greater percentage of negative emotion words chosen, relative to neutral emotion words, was predictive of steeper reductions in EMG Amplitude and Integral during the trauma imagination. This suggests that while there were not benefits overall of Affect Labeling compared to Shape Labeling, those who chose more negative emotional labels to describe their current emotions experienced a greater benefit compared to those who chose more neutral words.

Finally, satisfaction ratings were high across all conditions, but there were no differences between conditions. This was a promising finding that suggests that the training was feasible and acceptable to participants. Further, this finding provided support for continued investigation into the benefits of a similar training in an expanded sample.

This study provides preliminary support for significant reductions in both self-reported depression and physiological reductions during the trauma script from pre-to post-training. Unlike traditional imaginal exposure sessions that occur over 12 sessions for up to 90 minutes (Foa et al, 1992), these findings suggest that even minimal, one minute trials of imaginal

exposure confer benefit on trauma reactivity. However, SCL during the neutral script significant increased from Pre- to Post-training, perhaps because pre-training levels were very low, reflective of anticipatory anxiety at baseline on Day 1.

Given the baseline differences encountered in this study, the Main Study used randomization stratified by trauma severity. Both significant effects overall and improvements over Time demonstrated benefit of labeling During imagination compared to After. Therefore, this provided some justification for including labeling During, rather than After, imagination in the main study. Further, Affect Labeling is purported to confer benefit via attenuations of the amygdala as modulated by the right ventrolateral prefrontal cortex (Lieberman et al., 2007). Therefore, in order to maximize the potential for training in attenuation of physiology, labeling was provided in the middle of a phase where the amygdala should be hyperactive (Phelps et al., 2004).

Main Study Methods

Study Design

The main study used a 3 (Affect Labeling “AL” vs. Distraction Labeling “DL” vs. Exposure Only “EO”) X 2 (Pre- vs. Post-Training) design.

Participants

Participants ($n=66$, 61 completers) included undergraduate students (76%) from psychology courses and the rest of the UCLA campus, and community members (24%). The average age was 24.6 years old ($SD=10.6$, range 18-63) on average, 72% were female, and the sample was ethnically diverse. See Table 12 below for full demographic details.

Table 12. Demographic Details for the Main Study

Demographic Variable	Total Sample	Exposure Only (EO)	Affect Labeling (AL)	Distraction Labeling (DL)	Test Statistic by Condition
Age	24.6 (10.6)	25.1 ($SD=9.3$)	27.14 ($SD=14.47$)	21.11 ($SD=5.23$)	$F(2,58)=1.69, p=.1938$
Gender (% Female)	72.13%	76.20%	71.40%	68.40%	$2=.3074, p=.858$
Ethnicity					Fisher's exact $p=.530$
Hispanic/Latino	27.90%	28.60%	28.60%	26.20%	
Asian/Asian American	23%	28.60%	23.80%	15.80%	
White/Caucasian	20%	19.05%	19.05%	21.05%	
African American	11.50%	19.05%	9.52%	5.26%	
Other	17.60%	4.70%	19.03%	36.70%	
English First Language (%)	73.77%	76.20%	76.20%	68.40%	$2=.4081, p=.815$

Eligibility criteria for the main study were similar to the Pilot Study with the following exceptions made to increase generalizability of the findings: 1) dissociation was not used as a rule-out criterion based on a recent review suggesting that dissociation does not impede fear extinction (van Minnen, Harned, Zoellner, & Mills, 2012), and 2) those with mild or well-

controlled asthma were permitted to participate. See Appendix XX for eligibility and randomization details.

Measures

Clinician Administered and Self-report Measures

All of the clinician-administered and self-report measures described in the pilot study were used in the main study, including the PCL, DES, ISAS, BDI, PDS, and CSQ. Unlike the pilot study, the main study included the provision of the PDS at both Pre- and Post-SDI-A. A functional impairment scale on the PDS (PDS-Func) was analyzed separately at Pre- and Post-SDI-A.

SDI-A. The SDI-A was identical to the assessment conducted in the Pilot Study (see Appendix XXI). All of the psychophysiological measures described in the Pilot Study were used in this study, including SCL, HR, F-EMG, and startle blink EMG. This assessment occurred at both Pre- and Post-SDI-A, and the main includes analyses of the process of change in physiology measures throughout SDI-T.

Linguistic Inquiry and Word Count (LIWC; Pennebaker, Booth, & Francis, 2007).

LIWC is a software program that allows for entry of text word choices, and categorizes input into several types of word choices. It is a widely used software that has been applied to a variety of linguistic analyses (Kahn, Tobin, Massey, & Anderson, 2007). Word choice during the affect labeling condition were entered into LIWC software. While over 70 language dimensions are provided in the output from LIWC, the following categories were used for the current study: anxiety, sadness, and anger.

Training (SDI-T) Trials

Participants were provided with Training-specific instructions prior to the first trial. Regardless of experimental condition, each trial included one script presentation for 30 s, followed by the audio prompt to “Please imagine as vividly as possible the details of the event that you just heard about.” Then, participants were given 15 s to imagine the event. At this point, participants engaged in a 30s task (detailed below) that was specific to their experimental condition. Then participants were prompted to “Please return to imagination” for 15 s. Finally, participants were instructed to “Please relax” for 60 s. Then the next trial began.

Exposure Only experimental condition (EO; $n=23$, 21 completers). Prior to beginning the training trials, participants were read the following instructions:

“How are you feeling? Now we’ll continue to the next computer task, if that’s okay with you. During this part of the task, you will only hear the trauma script. Please listen to the script closely and try to visualize the contents of the script. As with the prior task, you will imagine the contents of the script as vividly as possible after you have heard it, but this time you will only imagine for 15 seconds. Then you will hear a voice asking that you stop imaging and sit quietly for 30 seconds. You will hear another voice that says “Please return to imagination” at which point you will return to imagining the contents of the traumatic event as vividly as possible again for 15 seconds. Then, you will hear a voice indicating that you can relax for 60 seconds. Then, the script will be presented again. You will hear the script a total of 12 times during this task. Do you have any questions?”

After 15 s of imagination, participants in the exposure only condition were instructed to “Please sit quietly for the next 30 seconds.” This instruction was presented both in audio and visual prompts, and the visual prompt remained on the screen until participants were instructed to return to imagination.

Affect Labeling experimental condition (AL; $n=21$ completers). The instructions provided to the AL condition were as follows:

“How are you feeling? Now we’ll continue to the next computer task, if that’s okay with you. During this part of the task, you will only hear the trauma script. Please listen to the script closely and try to visualize the contents of the script. As with the prior task, you will imagine the contents of the script as vividly as possible after you have heard it, but this time you will only imagine for 15 seconds. Then you will hear a voice asking that you stop imaging and to “Please create a sentence using one negative word to describe your current emotion and one negative word to describe the event.” We will ask you to say this sentence aloud, and we will audio record your sentence. The sentence includes two parts, a stimulus (or the event) and a response (or your emotional reaction right now). An example is “Thinking about the violent attack makes me very angry,” where “violent” describes the attack and “angry” describes what you’re feeling in this moment as you think about it. Another example is “Hearing about the disgusting [insert event word here] makes me feel very sad,” where “disgusting” describes the traumatic event, and “very sad” describes what you’re feeling in this moment as you think about it. Does this make sense so far?

You will have 30 seconds to say your sentence aloud, and you then will hear another voice that says “Please return to imagination” at which point you will return to imagining the contents of the traumatic event as vividly as possible again for 15 seconds. Then you will hear a voice indicating that you can relax for 60 seconds. Then, the script will be presented again. You will be asked to create a new sentence for every OTHER script. Sometimes the task will ask you to simply repeat the most recent sentence that you spoke aloud. You will hear the script a total of 12 times during this task. Do you have any questions? Just to make sure that you understand, can you repeat back to me what I’m asking you to do?”

Following imagination, participants in the AL condition were instructed to “Please create a sentence using one negative word to describe your current emotion and one negative word to describe the event.” They had 30 s to verbalize their sentence, which was written down by the experimenter.

Distraction Labeling experimental condition (DL; n=22, 19 completers). Prior to training trials, participants in the DL condition were provided with the following instructions:

“How are you feeling? Now we’ll continue to the next computer task, if that’s okay with you. During this part of the task, you will only hear the trauma script. Please listen to the script closely and try to visualize the contents of the script. As with the prior task, you will imagine the contents of the script as vividly as possible after you have heard it, but this time you will only imagine for 15 seconds. Then you will hear a voice asking that you stop imaging and to ‘Please create a sentence using one word to describe an object or piece of furniture in your home and one word to describe the room in which the

furnishing is kept.’ We will ask you to say this sentence aloud, and we will audio record your sentence. The sentence includes two parts, the piece of furniture, and the room. An example is ‘I have a coffee table in my living room,’ in which ‘table’ is the word used to describe the object or piece of furniture found in your home, and ‘living room’ is the word or two used to describe a room or location in which the furnishing is found.

Another example is ‘There is a dresser in my bedroom,’ where ‘dresser’ is the word used to describe the object or piece of furniture and ‘bedroom’ is used to describe the room in which it’s kept. Does this make sense so far?

You will have 30 seconds to say your sentence aloud, and you will then hear another voice that says ‘Please return to imagination’ at which point you will return to imagining the contents of the traumatic event as vividly as possible again for 15 seconds. Then you will hear a voice indicating that you can relax for 60 seconds. Then, the script will be presented again. You will be asked to create a new sentence for every OTHER script. Sometimes the task will ask you to simply repeat the most recent sentence that you spoke aloud. You will hear the script a total of 12 times during this task. Do you have any questions? Just to make sure that you understand, can you repeat back to me what I’m asking you to do?”

Procedure

Interested participants completed the MINI and self-report questionnaires either on a phone screen (for community members) or in person (for UCLA students) to determine eligibility. UCLA students completed the PCL during the psychology department’s mass testing, whereas community members completed the PCL on the phone screen. Participants were

required to score a 34 or greater on the PCL in order to be invited to participate. Once eligible, participants completed the script generation procedures detailed in the methods of the pilot study. These scripts were rated by two independent raters for speed and neutrality of the recording on a 7 point Likert scale (ranging from - 3 to + 3) rating the speed and neutrality of script recordings. Consensus within 1 point occurred on 87.2% of speed ratings and 98% of neutrality ratings. For ratings that diverged by two points or more, the raters discussed their ratings and came to a final consensus for that script. Mean ratings for neutrality and speed across both trauma and neutral scripts were consistently between -1 and 1 (range -.53 to .50) indicating that the recordings were on average appropriately neutral and an appropriate speed. The raters indicated that there was no difference between groups on speed of the trauma recordings (rater A: $F(2,63)=.11, p=.89$; rater B: $F(2,63)=.09, p=.92$) or speed of the neutral recordings (rater A: $F(2,63)=.34, p=.71$; rater B: $F(2,63)=.28, p=.76$). Similarly, the raters did not differ in their ratings of neutrality of script delivery for the trauma script (rater A: $F(2,63)=.22, p=.81$; rater B: $F(2,63)=.20, p=.82$) or the neutral script (rater A: $F(2,63)=.44, p=.66$; rater B: $F(2,63)=.46, p=.63$).

Randomization to one of three groups (EO, AL, or DL), occurred after script generation and was stratified by trauma severity (as measured by the PCL) and community status (UCLA student vs. community member). A stratified randomization procedure was created using Stata 13 (StataCorp, 2013). Then, participants had the psychophysiological electrodes affixed and completed the Pre-SDI-A followed by 12 SDI-T trials, based on experimental condition. Day 1 was completed in 2-2.5 hours.

Participants returned for to the laboratory for Day 8 of the study one week later. They were attached to the psychophysiological electrodes and completed 12 more SDI-T trials. Day 8

of the study lasted approximately 1 hour. Finally, participants returned one week later for Day 15 of the study, during which they completed the Post-SDI-A and the final questionnaires.

UCLA students received 5 course credits for participation, and community members received either \$40 or \$80 based on the date of study enrollment.

Data Analytic Strategy

As with the pilot study, first, all data was examined for outliers, operationalized as three standard deviations above the mean (Guttman, 1973), and were Winsorized to the closest non-outlier value. Less than 2% of the data for Pre- and Post-analyses and process analyses were Winsorized. All data were then inspected for normality and homoscedasticity, and an ICC was calculated for each outcome variable. Next, a series of univariate ANOVAs was run on all outcome variables to confirm that there were no differences in baseline self-report or physiology by attrition status. A similar analysis was run to confirm the lack of baseline differences by experimental condition.

When examining Pre- to Post-Training changes, two-level growth curve models were calculated using the mixed command in Stata 13 using maximum likelihood to account for the autocorrelation among observations (see Appendix XXIII for ICC calculation). Level 1 included Time (Pre- and Post-Training) which was modeled as a continuous linear predictor. Level 2 included Condition as a categorical predictor. Random effects of intercepts were included in all models, and random effects of Time were included as indicated. When random effects of time were included, unstructured variance/covariance structures were estimated, and otherwise homogeneous variance/covariance structures were estimated.

The model for Specific Aims 1-3 was as follows:

$$\begin{aligned} \check{z}_{it} &= \beta_0 + \beta_1 t + \beta_2 \text{Condition} + \beta_3 \text{Time} \times \text{Condition} + \beta_4 \text{Time}^2 + \beta_5 \text{Time} \times \text{Condition}^2 \\ &+ \beta_6 \text{Time} \times \text{Condition} \times \text{Condition}^2 + \beta_7 \text{Time} \times \text{Condition} \times \text{Condition}^3 + \beta_8 \text{Time} \times \text{Condition} \times \text{Condition}^4 \\ &+ \beta_9 \text{Time} \times \text{Condition} \times \text{Condition}^5 + \beta_{10} \text{Time} \times \text{Condition} \times \text{Condition}^6 + \beta_{11} \text{Time} \times \text{Condition} \times \text{Condition}^7 \\ &+ \beta_{12} \text{Time} \times \text{Condition} \times \text{Condition}^8 + \beta_{13} \text{Time} \times \text{Condition} \times \text{Condition}^9 + \beta_{14} \text{Time} \times \text{Condition} \times \text{Condition}^{10} \\ &+ \beta_{15} \text{Time} \times \text{Condition} \times \text{Condition}^{11} + \beta_{16} \text{Time} \times \text{Condition} \times \text{Condition}^{12} + \beta_{17} \text{Time} \times \text{Condition} \times \text{Condition}^{13} \\ &+ \beta_{18} \text{Time} \times \text{Condition} \times \text{Condition}^{14} + \beta_{19} \text{Time} \times \text{Condition} \times \text{Condition}^{15} + \beta_{20} \text{Time} \times \text{Condition} \times \text{Condition}^{16} \\ &+ \beta_{21} \text{Time} \times \text{Condition} \times \text{Condition}^{17} + \beta_{22} \text{Time} \times \text{Condition} \times \text{Condition}^{18} + \beta_{23} \text{Time} \times \text{Condition} \times \text{Condition}^{19} \\ &+ \beta_{24} \text{Time} \times \text{Condition} \times \text{Condition}^{20} + \beta_{25} \text{Time} \times \text{Condition} \times \text{Condition}^{21} + \beta_{26} \text{Time} \times \text{Condition} \times \text{Condition}^{22} \\ &+ \beta_{27} \text{Time} \times \text{Condition} \times \text{Condition}^{23} + \beta_{28} \text{Time} \times \text{Condition} \times \text{Condition}^{24} + \beta_{29} \text{Time} \times \text{Condition} \times \text{Condition}^{25} \\ &+ \beta_{30} \text{Time} \times \text{Condition} \times \text{Condition}^{26} + \beta_{31} \text{Time} \times \text{Condition} \times \text{Condition}^{27} + \beta_{32} \text{Time} \times \text{Condition} \times \text{Condition}^{28} \\ &+ \beta_{33} \text{Time} \times \text{Condition} \times \text{Condition}^{29} + \beta_{34} \text{Time} \times \text{Condition} \times \text{Condition}^{30} + \beta_{35} \text{Time} \times \text{Condition} \times \text{Condition}^{31} \\ &+ \beta_{36} \text{Time} \times \text{Condition} \times \text{Condition}^{32} + \beta_{37} \text{Time} \times \text{Condition} \times \text{Condition}^{33} + \beta_{38} \text{Time} \times \text{Condition} \times \text{Condition}^{34} \\ &+ \beta_{39} \text{Time} \times \text{Condition} \times \text{Condition}^{35} + \beta_{40} \text{Time} \times \text{Condition} \times \text{Condition}^{36} + \beta_{41} \text{Time} \times \text{Condition} \times \text{Condition}^{37} \\ &+ \beta_{42} \text{Time} \times \text{Condition} \times \text{Condition}^{38} + \beta_{43} \text{Time} \times \text{Condition} \times \text{Condition}^{39} + \beta_{44} \text{Time} \times \text{Condition} \times \text{Condition}^{40} \\ &+ \beta_{45} \text{Time} \times \text{Condition} \times \text{Condition}^{41} + \beta_{46} \text{Time} \times \text{Condition} \times \text{Condition}^{42} + \beta_{47} \text{Time} \times \text{Condition} \times \text{Condition}^{43} \\ &+ \beta_{48} \text{Time} \times \text{Condition} \times \text{Condition}^{44} + \beta_{49} \text{Time} \times \text{Condition} \times \text{Condition}^{45} + \beta_{50} \text{Time} \times \text{Condition} \times \text{Condition}^{46} \\ &+ \beta_{51} \text{Time} \times \text{Condition} \times \text{Condition}^{47} + \beta_{52} \text{Time} \times \text{Condition} \times \text{Condition}^{48} + \beta_{53} \text{Time} \times \text{Condition} \times \text{Condition}^{49} \\ &+ \beta_{54} \text{Time} \times \text{Condition} \times \text{Condition}^{50} + \beta_{55} \text{Time} \times \text{Condition} \times \text{Condition}^{51} + \beta_{56} \text{Time} \times \text{Condition} \times \text{Condition}^{52} \\ &+ \beta_{57} \text{Time} \times \text{Condition} \times \text{Condition}^{53} + \beta_{58} \text{Time} \times \text{Condition} \times \text{Condition}^{54} + \beta_{59} \text{Time} \times \text{Condition} \times \text{Condition}^{55} \\ &+ \beta_{60} \text{Time} \times \text{Condition} \times \text{Condition}^{56} + \beta_{61} \text{Time} \times \text{Condition} \times \text{Condition}^{57} + \beta_{62} \text{Time} \times \text{Condition} \times \text{Condition}^{58} \\ &+ \beta_{63} \text{Time} \times \text{Condition} \times \text{Condition}^{59} + \beta_{64} \text{Time} \times \text{Condition} \times \text{Condition}^{60} + \beta_{65} \text{Time} \times \text{Condition} \times \text{Condition}^{61} \\ &+ \beta_{66} \text{Time} \times \text{Condition} \times \text{Condition}^{62} + \beta_{67} \text{Time} \times \text{Condition} \times \text{Condition}^{63} + \beta_{68} \text{Time} \times \text{Condition} \times \text{Condition}^{64} \\ &+ \beta_{69} \text{Time} \times \text{Condition} \times \text{Condition}^{65} + \beta_{70} \text{Time} \times \text{Condition} \times \text{Condition}^{66} + \beta_{71} \text{Time} \times \text{Condition} \times \text{Condition}^{67} \\ &+ \beta_{72} \text{Time} \times \text{Condition} \times \text{Condition}^{68} + \beta_{73} \text{Time} \times \text{Condition} \times \text{Condition}^{69} + \beta_{74} \text{Time} \times \text{Condition} \times \text{Condition}^{70} \\ &+ \beta_{75} \text{Time} \times \text{Condition} \times \text{Condition}^{71} + \beta_{76} \text{Time} \times \text{Condition} \times \text{Condition}^{72} + \beta_{77} \text{Time} \times \text{Condition} \times \text{Condition}^{73} \\ &+ \beta_{78} \text{Time} \times \text{Condition} \times \text{Condition}^{74} + \beta_{79} \text{Time} \times \text{Condition} \times \text{Condition}^{75} + \beta_{80} \text{Time} \times \text{Condition} \times \text{Condition}^{76} \\ &+ \beta_{81} \text{Time} \times \text{Condition} \times \text{Condition}^{77} + \beta_{82} \text{Time} \times \text{Condition} \times \text{Condition}^{78} + \beta_{83} \text{Time} \times \text{Condition} \times \text{Condition}^{79} \\ &+ \beta_{84} \text{Time} \times \text{Condition} \times \text{Condition}^{80} + \beta_{85} \text{Time} \times \text{Condition} \times \text{Condition}^{81} + \beta_{86} \text{Time} \times \text{Condition} \times \text{Condition}^{82} \\ &+ \beta_{87} \text{Time} \times \text{Condition} \times \text{Condition}^{83} + \beta_{88} \text{Time} \times \text{Condition} \times \text{Condition}^{84} + \beta_{89} \text{Time} \times \text{Condition} \times \text{Condition}^{85} \\ &+ \beta_{90} \text{Time} \times \text{Condition} \times \text{Condition}^{86} + \beta_{91} \text{Time} \times \text{Condition} \times \text{Condition}^{87} + \beta_{92} \text{Time} \times \text{Condition} \times \text{Condition}^{88} \\ &+ \beta_{93} \text{Time} \times \text{Condition} \times \text{Condition}^{89} + \beta_{94} \text{Time} \times \text{Condition} \times \text{Condition}^{90} + \beta_{95} \text{Time} \times \text{Condition} \times \text{Condition}^{91} \\ &+ \beta_{96} \text{Time} \times \text{Condition} \times \text{Condition}^{92} + \beta_{97} \text{Time} \times \text{Condition} \times \text{Condition}^{93} + \beta_{98} \text{Time} \times \text{Condition} \times \text{Condition}^{94} \\ &+ \beta_{99} \text{Time} \times \text{Condition} \times \text{Condition}^{95} + \beta_{100} \text{Time} \times \text{Condition} \times \text{Condition}^{96} + \beta_{101} \text{Time} \times \text{Condition} \times \text{Condition}^{97} \\ &+ \beta_{102} \text{Time} \times \text{Condition} \times \text{Condition}^{98} + \beta_{103} \text{Time} \times \text{Condition} \times \text{Condition}^{99} + \beta_{104} \text{Time} \times \text{Condition} \times \text{Condition}^{100} \end{aligned}$$

Where y_{it} represents the dependent variable for time t nested within individual i , t represents time for an individual i , β_0 is the intercept of the dependent variable for individual i , and β_1 is the growth parameters for an individual i , β_2 is the Condition comparison between Exposure and Affect Labeling (Affect Labeling =0 vs. Exposure =1), β_3 is the Condition comparison between Affect Labeling and Distract Labeling (Affect =0 vs. Distract =1), β_4 is an overall intercept (i.e., overall score on the dependent variable at Pre-SDI-A in Affect Labeling), β_5 is the mean difference in the dependent variable between Exposure and Affect Labeling Pre-SDI-A, β_6 is the mean difference in the dependent variable between Affect Labeling and Distract Labeling at Pre-SDI-A for SL, β_7 is the mean effect of time for Affect Labeling, β_8 is the mean difference in the time effect for Exposure vs. Affect Labeling, β_9 is the mean difference in the time effect for Affect Labeling vs. Distract Labeling, σ^2 is the within-person error variance for participant i at time t , ϵ_{it} is each participant's deviation from the overall intercept controlling for Condition, and δ_{it} is each participant's deviation from the overall slope controlling for Condition.

When there was no significant Time x Condition interaction, this parameter was removed from the model. PDS-Func is a count variable, and thus a multilevel mixed effect Poisson regression was used for this model. ICC is not calculated for this model as the outcome is not continuous (Lindsey, 2012). All participants were included in analyses regardless of attrition

status. Cohen's *d* and Hedges' *g* effect size estimates were calculated as differences from Pre- to Post-SDI-A.

Baseline PDS and PDS-Func were explored as potential moderators of physiological outcomes in Pre- and Post-analyses, consistent with Specific Aim 4. Multilevel models were run including a three-way interaction between Time, Condition, and the centered moderator of interest alongside each lower-order interaction and main effect. In the event of a non-significant three-way moderation, this term was removed from the model but the lower-order interaction terms were retained alongside main effects. Finally, lower-order interactions were removed from the model and the moderator was examined as a predictor of outcome controlling for Time and Condition. The model for these analyses was as follows:

$$\begin{aligned} \hat{Y}_{ij} &= \beta_0 + \beta_1 \text{Time} + \beta_2 \text{Condition} + \beta_3 \text{Moderator} + \beta_4 \text{Time} \times \text{Condition} \\ &+ \beta_5 \text{Time} \times \text{Moderator} + \beta_6 \text{Condition} \times \text{Moderator} + \beta_7 \text{Time} \times \text{Condition} \times \text{Moderator} + \epsilon_{ij} \end{aligned}$$

Where Moderator indicates the inclusion of either PDS or PDS-Func.

Next, word choice throughout the training trials was used as a moderator of the relationship between Time, Condition, and Outcome, consistent with Specific Aim 5. Sentences were entered into LIWC software and several categories of interest were explored including anxious, angry, and sad word choices. The model for these analyses was as follows:

$$\begin{aligned} \hat{Y}_{ij} &= \beta_0 + \beta_1 \text{Time} + \beta_2 \text{Condition} + \beta_3 \text{Moderator} + \beta_4 \text{Time} \times \text{Condition} \\ &+ \beta_5 \text{Time} \times \text{Moderator} + \beta_6 \text{Condition} \times \text{Moderator} + \beta_7 \text{Time} \times \text{Condition} \times \text{Moderator} + \epsilon_{ij} \end{aligned}$$

In this model, emotion indicates the inclusion of an emotion word.

Next, a comparison of effect size estimates were calculated in relation to a recent meta-analysis of CBT for PTSD (Hofmann & Smits, 2008). Whereas this meta-analysis calculated effect sizes in relation to a placebo control condition, the current study did not include a placebo control. Therefore, effect size estimates were re-calculated using the Pre- and Post- means provided in each study. Self-report measures were chosen as the variable under consideration wherever possible for closer comparison to the main study.

Following the Pre-to Post SDI-A analyses, a series of process analyses included each trial in the SDI-A and SDI-T across each psychophysiology measure except startle, which was not collected during the training trials. This series of analysis allows for a more nuanced understanding of changes in physiology throughout the training, consistent with the goals of Specific Aim 6. Trials of gold-standard treatments for PTSD have revealed non-linear improvements in symptoms, with some studies showing initial worsening followed by eventual alleviation of distress (Nishith, Resick, & Griffin, 2002). Further, naturalistic observation of symptom trajectories over time without training reveal non-linear trends for certain symptom clusters of PTSD (specifically arousal; O'Donnell, Elliot, Lau, & Creamer, 2007), and there is a strong literature suggesting non-linearity in rates of inhibitory learning throughout extinction training (Plendl, 2010). Therefore, these analyses were included to allow for an exploration of non-linear changes in outcomes. Process analyses were computed using multilevel modeling following the same parameters as described above, including 28 trials nested within participants

(24 Training trials plus 2 Pre- and 2 Post-trials) and estimation of up to a quintic effect of Time (see Appendix XXIV for ICC for the process analyses). The imagination portion of each trial was calculated as an average of imagination before and after Affect Labeling to reduce the number of analyses. Finally, a univariate ANOVA was calculated using client satisfaction, as measured by the CSQ, by Condition.

In terms of missing data at Pre-SDI-A, 2 people had unusable EMG data for the trauma script, 1 person had unusable EMG data for the neutral script, and 4 participants had unusable F-EMG. At Post-SDI-A (excluding those who discontinued), 4 participants had unusable F-EMG during the trauma script, and 5 had unusable F-EMG during the neutral script.

Results

SDI-T Discontinuation

One participant discontinued participation in between SDI-A and the first SDI-T trial. Four other participants discontinued participation after 12 SDI-T trials citing the following reasons by Condition: 1) EO: scheduling conflict and reason not stated; 2) DL: personal events and nightmare increase. Mean scores on all outcome and moderator variables are presented in Table 10 below. Low drop-out prevents statistical comparison between the groups.

Table 13. Baseline means by completer status in the Main Study.

Baseline Means by Completer Status		
Self-Report	Completer Mean (SD)	Drop-Out Mean (SD)
PDS	21.5 (12.1)	21.2 (12.7)
IES	21.6 (12.4)	15.8 (14.8)
BDI	13.5 (7.3)	17.6 (7.6)
PDS-Func	4.3 (2.7)	4.4 (4.0)
DES	16.5 (9.9)	15.2 (9.6)

Physiology		
HR		
Trauma Script	5.3 (4.7)	4.2 (7.9)
Trauma Imagine	2.3 (4.0)	2.0 (3.1)
Neutral Script	2.5 (3.4)	4.4 (5.7)
Neutral Imagine	.5 (3.6)	1.3 (3.1)
SCL		
Trauma Script	1.3 (1.9)	.8 (3.2)
Trauma Imagine	1.1 (2.1)	.5 (3.1)
Neutral Script	.9 (2.1)	.01 (2.6)
Neutral Imagine	.6 (2.1)	-.6 (2.1)
EMG-Amp		
Trauma Imagine	3.2 (.7)	3.1 (.6)
Neutral Imagine	3.3 (.6)	3.4 (.9)
EMG-Int		
Trauma Imagine	1.6 (.6)	1.4 (.3)
Neutral Imagine	1.6 (.6)	1.9 (.6)
F-EMG		
Trauma Script	1.5 (7.0)	1.4 (2.2)
Trauma Imagine	3.6 (11.8)	2.3 (4.1)
Neutral Script	-.4 (5.9)	.8 (1.9)
Neutral Imagine	-1.4 (6.4)	-1.2 (3.1)

Baseline Differences by Experimental Condition

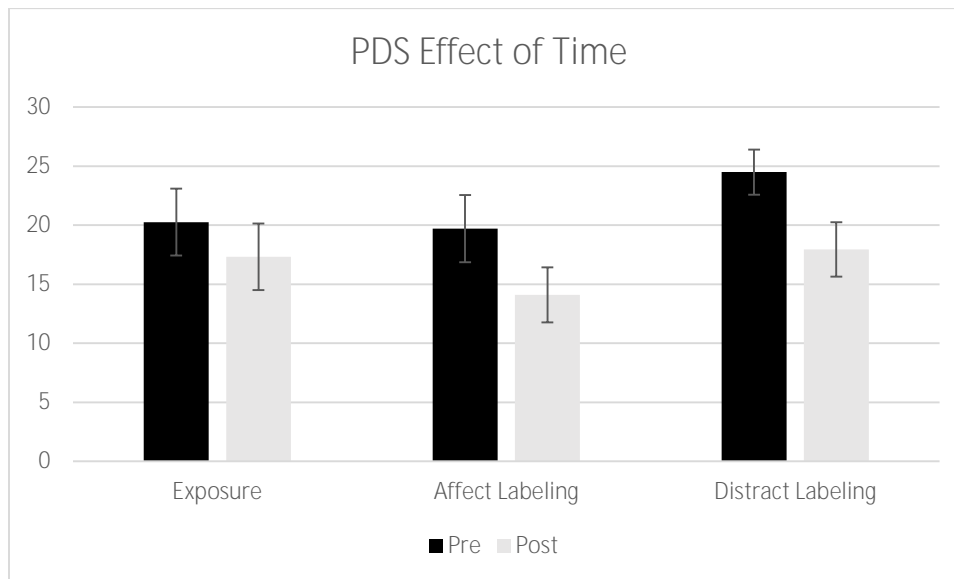
There were no baseline differences in PDS ($F(2,63)=1.04, p=.360, R^2=.032$), IES ($F(2,63)=.45, p=.642, R^2=.014$), BDI ($F(2,63)=2.96, p=.06, R^2=.086$), PDS-Func (Fisher's Exact $p=.259$), DES ($F(2,63)=.93, p=.4013, R^2=.0286$), or by Condition. Similarly, there were no baseline differences in HR to the trauma ($F(2,63)=2.58, p=.084, R^2=.0756$) or neutral script ($F(2,63)=1.89, p=.1596, R^2=.0566$), HR to trauma ($F(2,63)=1.13, p=.3296, R^2=.0346$) or neutral imagination ($F(2,63)=.36, p=.6976, R^2=.0114$), SCL to the trauma ($F(2,63)=.24, p=.788, R^2=.0075$) or neutral script ($F(2,63)=.45, p=.640, R^2=.0141$), SCL to the trauma ($F(2,63)=.05, p=.9466, R^2=.0017$) or neutral imagination ($F(2,63)=.49, p=.6171, R^2=.0152$). There were also

no differences in baseline F-EMG to the trauma ($F(2,59)=.60, p=.5505, R^2=.02$) or neutral script ($F(2,59)=.11, p=.89, R^2=.004$), F-EMG to the trauma ($F(2,59)=2.05, p=.14, R^2=.07$) or neutral imagination ($F(2,59)=.82, p=.45, R^2=.03$), EMG Amplitude trauma ($F(2,61)=1.90, p=.16, R^2=.06$) or neutral ($F(2,62)=.41, p=.66, R^2=.01$), or EMG Integral trauma ($F(2,61)=1.02, p=.37, R^2=.03$) or neutral ($F(2,62)=.27, p=.76, R^2=.01$). Furthermore, there were no differences in rates of a PTSD diagnosis ($\chi^2=3.04, p=.218$), major depression ($\chi^2=4.53, p=.104$), or type of trauma (Fisher's exact $p=.747$; see Appendix XXII) by Condition.

Self-Report Pre-to Post-SDI-A Changes

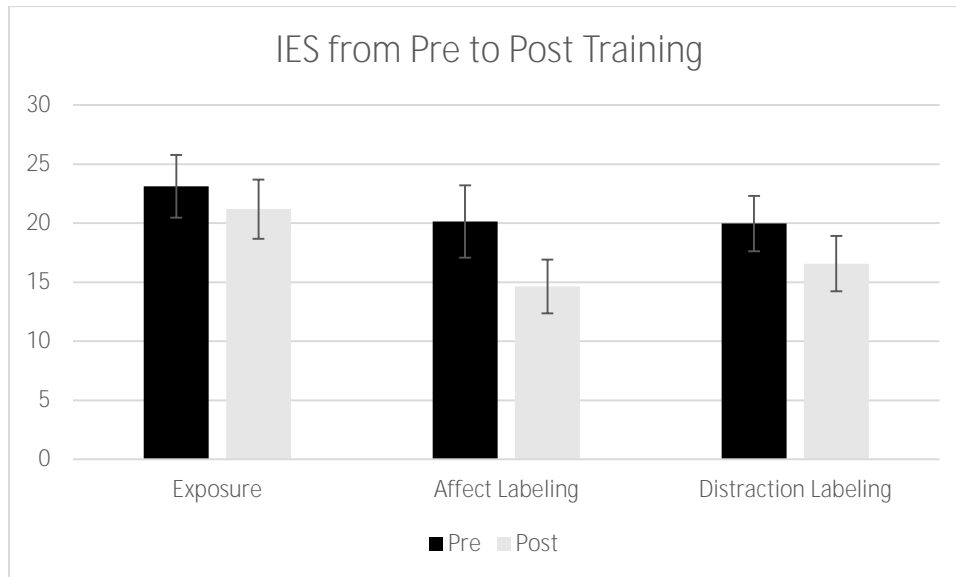
There was a significant effect of Time ($z= -4.44, p<.001, \text{Cohen's } d=.453, \text{Hedges' } g=.450$; see Figure 13) but no effect of Condition (omnibus $\chi^2=2.40, p=.3015$) or Time x Condition interaction (omnibus $\chi^2=.76, p=.6822$) on PDS. Reductions of at least 50% of symptom severity reported on the PDS were observed in 75.5% of participants (Griffin, Uhlmansiek, Resick, & Mechanic, 2004). Of those who scored above the clinical cutoff on the PDS at baseline, 72% experienced a 50% reduction in symptoms from Pre- to Post-SDI-A and 44.4% were below the clinical cutoff at Post-SDI-A. Tests of the simple slopes revealed that whereas both AL and DL slopes were significantly less than 0 ($p<.01$), the EO slope was not ($p=.055$). However, tests comparing the simple slopes between Condition were not significantly different (all $ps<.05$). If the labeling groups were collapsed together in order to compare Labeling vs. No-Labeling, there was not a significant Time X Labeling interaction ($z= -.88, p=.377$).

Figure 13. PDS from Pre- to Post-SDI-A.



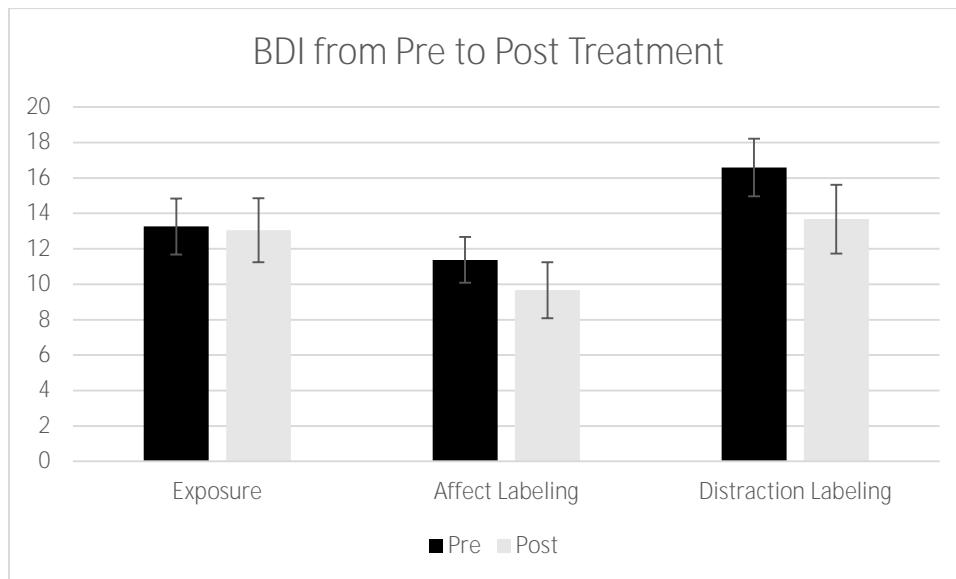
Similarly, there was a significant effect of Time ($z = -3.08, p < .01$, Cohen's $d = .3085$, Hedges' $g = .3058$; see Figure 13), but no effect of Condition (omnibus $\chi^2 = 1.75, p = .417$) or Time X Condition interaction (omnibus $\chi^2 = .89, p = .6409$) for IESⁱ. Tests of the simple slope revealed that only the AL condition had a significantly negative slope ($p < .05$), whereas both DL ($p = .219$) and EO did not ($p = .117$). However, comparison of simple slopes did not reveal any significant differences between them (all $ps > .05$). Further, if the labeling group was collapsed together to compare Labeling vs. No-Labeling, there was not a Time X Labeling/No-Labeling effect ($z = -.32, p = .746$).

Figure 14. IES from Pre- to Post-Training.



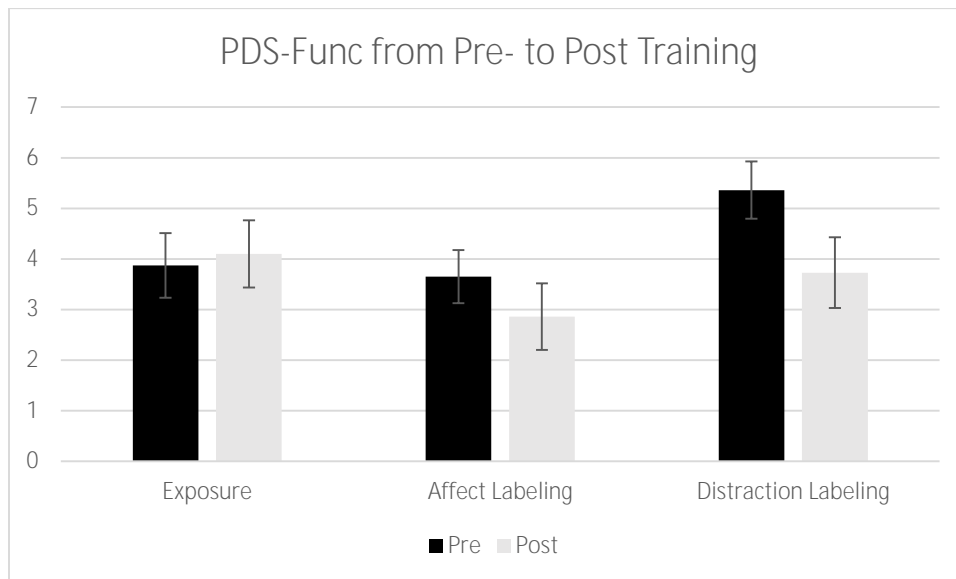
There was not a significant effect of Time ($z = -1.84, p = .066$, Cohen's $d = .219$, Hedges' $g = .219$), Condition (omnibus $\chi^2 = 5.81, p = .0548$), or a Time X Condition interaction (omnibus $\chi^2 = 1.07, p = .5871$; see Figure 15) on BDI. However, as demonstrated below, whereas the EO group reported nearly identical depression levels at Pre- and Post-Training, both labeling groups reported slight improvements in depression. None of the individual simple slopes were significant (all $ps > .05$) and none of the slopes differed by Condition (all $ps > .05$).

Figure 15: BDI from Pre to Post-Training.



There was a significant effect of Time ($z = -1.99, p < .05$, Cohen's $d = .253$, Hedges' $g = .252$; see Figure 16), but no effect of Condition (omnibus $\chi^2 = 3.53, p = .1708$) or Time x Condition interaction ($\chi^2 = 2.53, p = .2819$) on PDS-Func. However, it is important to note that while both AL and DL reported slightly improved functioning from Pre- to Post-Training, the EO group reported slightly worsened functioning, though none of the slopes were significantly different than zero and there were not significant differences in slopes by Condition (all $ps > 0.5$).

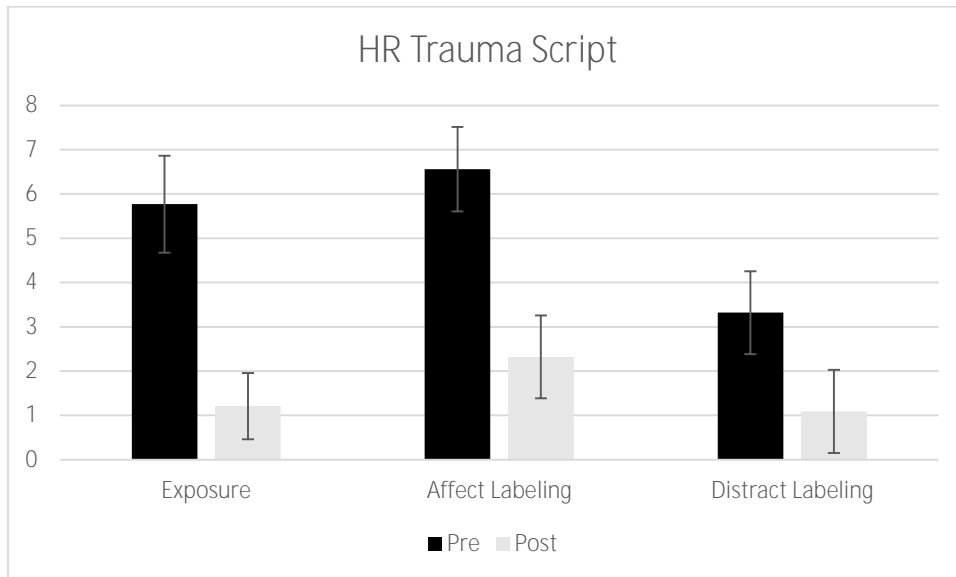
Figure 16. PDS-Func from Pre- to Post-Training.



Physiological Pre-to Post-Training Changes

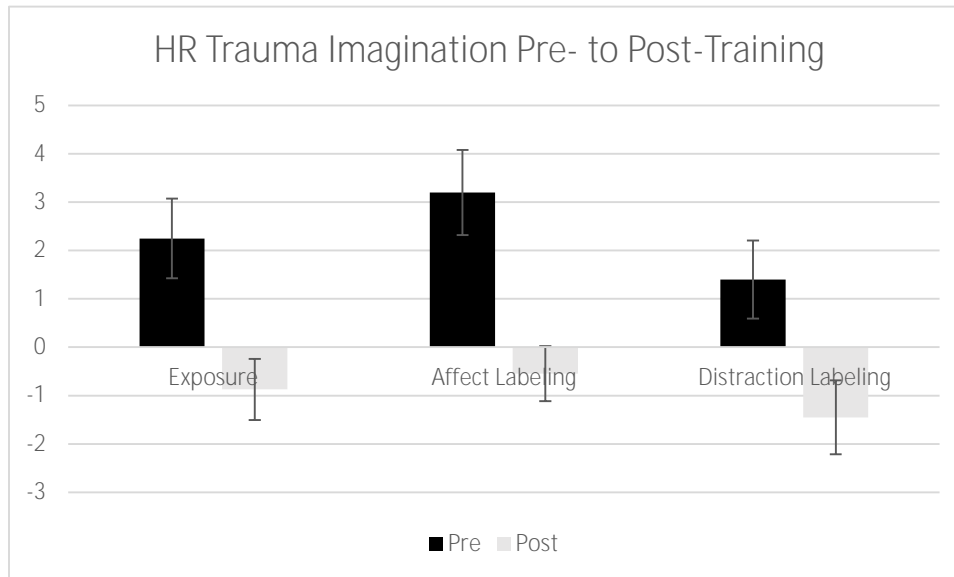
There was a significant effect of Time ($z = -6.50, p < .001$, Cohen's $d = .837$, Hedges' $g = .827$, see Figure 17), but not Condition ($\chi^2 = 4.91, p = .086$), or a Time x Condition interaction (omnibus $\chi^2 = 2.11, p = .3491$) for HR during the trauma script. While it appears that there are baseline differences by Condition, these differences are not significant. All simple slopes were significant and negative (all $ps < .05$) and did not differ by Condition (all $ps > .05$).

Figure 17. HR trauma script pre to post-training.



There was a significant effect of Time ($z = -6.15, p < .01$, Cohen's $d = .924$, Hedges' $g = .914$, see Figure 18), but not Condition ($\chi^2 = 2.78, p = .2496$) or a Time x Condition interaction (omnibus $\chi^2 = .49, p = .7846$) for HR during trauma imagination. All simple slopes were significant and negative (all $ps < .05$) and did not differ by Condition (all $ps > .05$).

Figure 18. HR imagine Pre- to Post-Training.

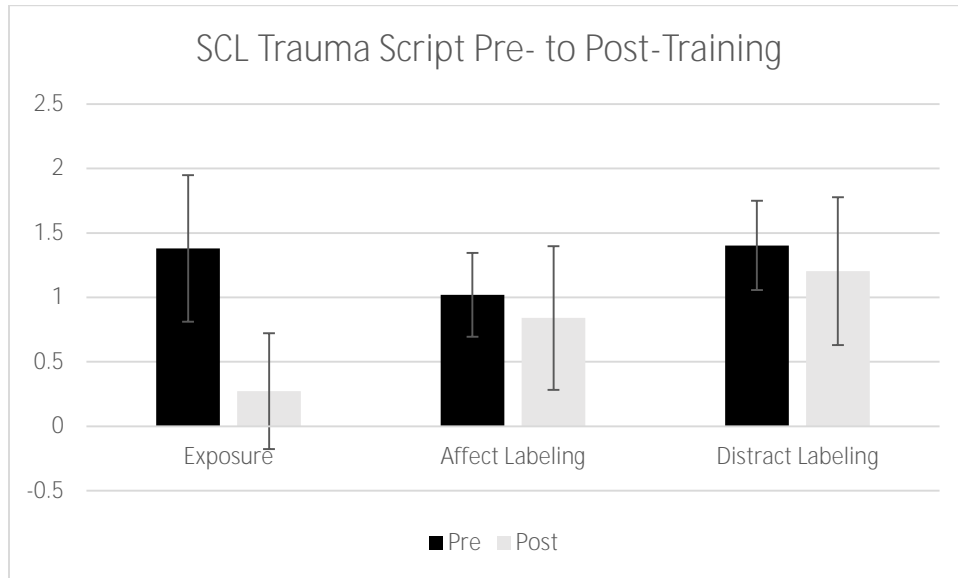


In contrast, there was not a significant effect of Time ($z = -1.80, p = .972$, Cohen's $d = .3153$, Hedges' $g = .3150$), Condition (omnibus $\chi^2 = .87, p = .6472$) or a Time x Condition interaction (omnibus $\chi^2 = 4.57, p = .1016$) for HR during the neutral script. Tests of simple slopes demonstrated a significant reduction for EO ($p < .05$) but not for either DL or AL condition, although there were not statistical differences in simple slopes by Condition (all $ps > .05$). There was a significant effect of Time ($z = -2.63, p < .01$, Cohen's $d = .4040$, Hedges' $g = .4007$) but not an effect of Condition (omnibus $\chi^2 = .33, p = .8483$) or a Time X Condition interaction (omnibus $\chi^2 = 2.52, p = .284$) for HR to neutral imagination. Tests of simple slope demonstrated a significant reduction for AL ($p < .01$) but not for either DL or EO, although tests of simple slope did not reveal any differences by Condition (all $ps > .05$).

There was not a significant effect of Time ($z = -1.53, p = .126$, Cohen's $d = .232$, Hedges' $g = .233$, see Figure 19), Condition (omnibus $\chi^2 = .66, p = .7204$), or a significant Time x Condition interaction (omnibus $\chi^2 = 1.46, p = .483$) for SCL during the trauma script. None of the individual

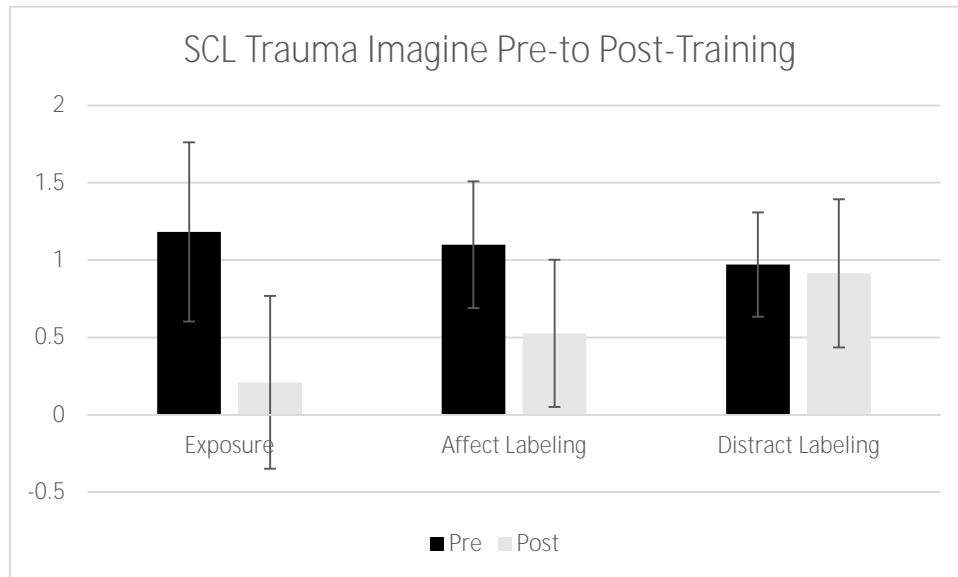
simple slopes were significantly different than 0 or significantly different by Condition (all $ps > .05$).

Figure 19. SCL trauma script Pre- to Post-training.



Similarly, there was not a significant effect of Time ($z = -1.58, p = .114$, Cohen's $d = .249$, Hedges' $g = .248$, see Figure 20), Condition (omnibus $\eta^2 = .12, p = .9433$), or a significant Time x Condition interaction (omnibus $\eta^2 = .93, p = .6270$) for SCL during trauma imagination. While there was not a significant difference based on Condition, the DL condition experienced less reduction compared to the EO and AL. None of the individual simple slopes were significantly different than zero or different based on condition (all $ps > .05$).

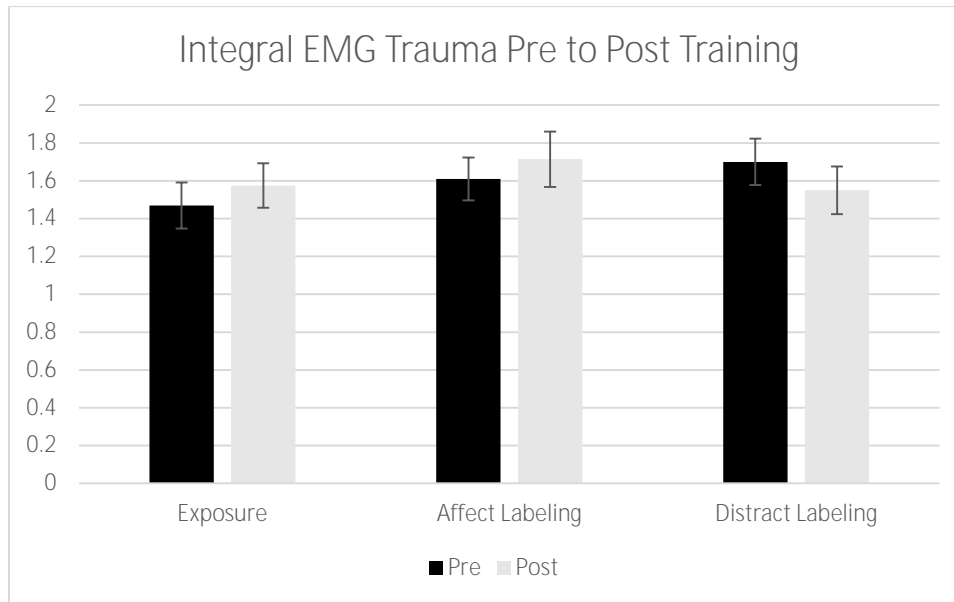
Figure 20: SCL trauma imagine pre- to post-training.



There were no significant effects for SCL during the neutral script (Time: $z = -.31$, $p = .756$, Cohen's $d = .040$; Hedges' $g = .040$; Condition: $\eta^2 = .84$, $p = .6571$; Time X Condition: $\eta^2 = .35$, $p = .8412$) or neutral imagination (Time: $z = -.02$, $p = .986$, Cohen's $d = -.006$, Hedges' $g = -.006$; Condition: $\eta^2 = .54$, $p = .7642$; Time X Condition: $\eta^2 = .65$, $p = .7238$). None of the individual simple slopes were significantly different than zero or different based on condition for SCL during neutral script or imagination (all $ps > .05$).

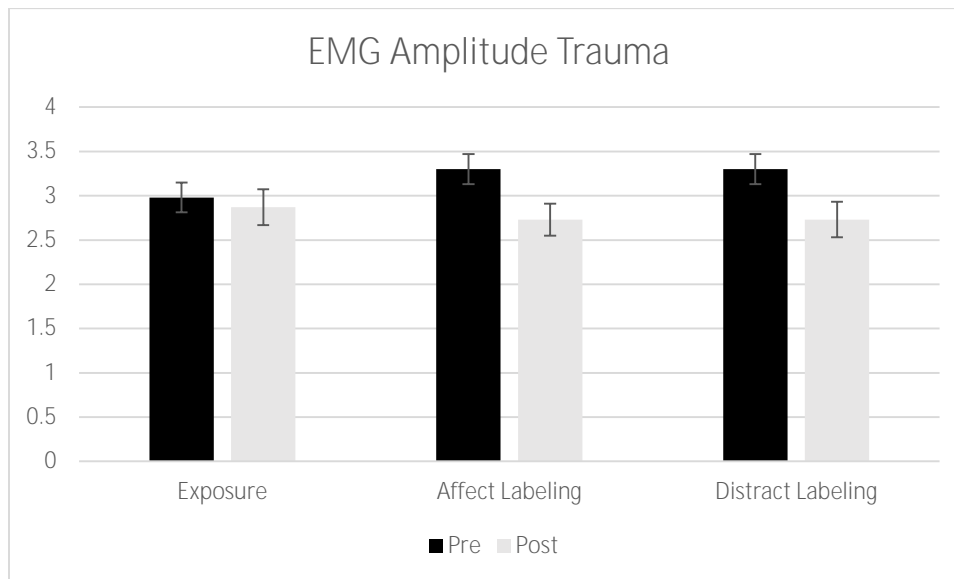
There was not a significant effect of Time ($z = .33$, $p = .741$, Cohen's $d = -.05$, Hedges' $g = -.05$; see Figure 21), Condition ($\eta^2 = 1.07$, $p = .5861$) or a Time x Condition interaction ($\eta^2 = 2.44$, $p = .2956$) for the integral of EMG during trauma imagination. None of the individual simple slopes were significantly different than zero or different based on condition (all $ps > .05$).

Figure 21. EMG trauma integral Pre- to Post-training.



There was a significant effect of Time ($z=-3.84$, $p<.001$, Cohen's $d=.532$, Hedges' $g=.530$, see Figure 22), but there was not an effect of Condition ($\eta^2=1.40$, $p=.4957$) or a Time x Condition interaction ($\eta^2=4.86$, $p=.0882$) on amplitude of EMG during trauma imagination. Whereas there was not a significant Time X Condition interaction, both AL ($p<.001$) and DL ($p<.01$) experienced significantly negative simple slopes, whereas the EO ($p=.492$) did not. There were significant differences between slopes when comparing DL and EO ($p<.05$), but not in any other comparison (all $ps>.05$). If the AL and DL were combined for a follow-up analysis comparing Label to No-Label, there was a Time X Condition interaction ($p<.05$). There was a significant slope for Label ($p<.01$) that was not present in No-Label ($p=.479$) and these slopes were significantly different ($p<.05$).

Figure 22. EMG amplitude Pre to Post-Training.

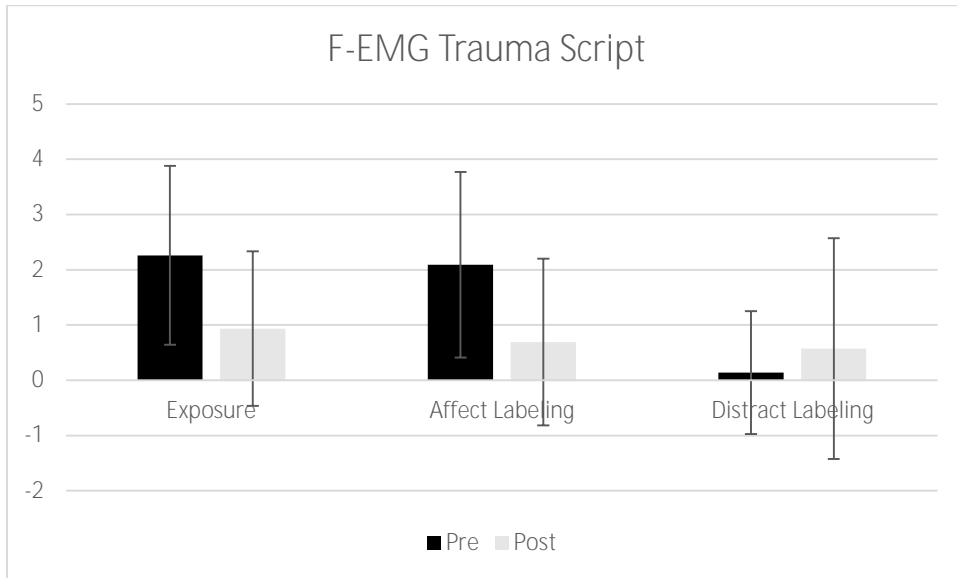


There was not a significant effect of Time ($z = -.29, p = .769$, Cohen's $d = .055$, Hedges' $g = .054$), Condition ($\eta^2 = .22, p = .896$), or a Time x Condition interaction ($\eta^2 = .28, p = .8677$) on EMG integral during neutral imagination. None of the individual simple slopes were significantly different from zero, or significantly different from each other (all $ps > .05$). There was an effect of Time ($z = -3.06, p < .01$, Cohen's $d = .436$, Hedges' $g = .435$), but no effect of Condition ($\eta^2 = .47, p = .791$) and no Time x Condition interaction ($\eta^2 = 1.11, p = .575$) on EMG amplitude during neutral imagination. The simple slope was different than zero for DL ($p < .05$) but not AL ($p = .166$) or EO ($p = .158$). None of the simple slopes EMG amplitude during neutral imagination were significantly different by Condition (all $ps > .05$).

There was not a significant effect of Time ($z = -.83, p = .405$, Cohen's $d = .113$; Hedges' $g = .112$, see Figure 23), Condition ($\eta^2 = 1.08, p = .583$), or a Time x Condition interaction ($\eta^2 = .58, p = .747$) on F-EMG during the trauma script. None of the simple slopes were significantly

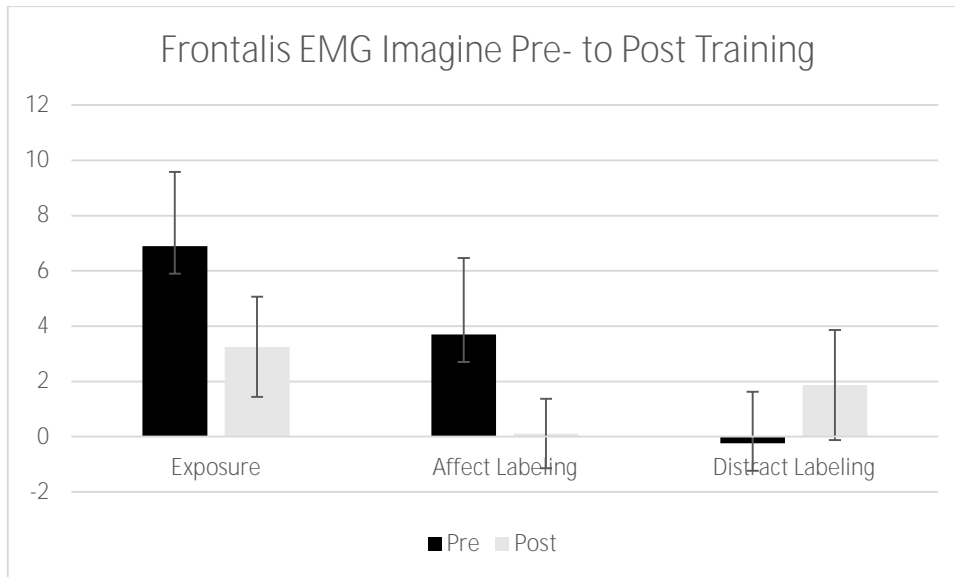
different than zero or significant different by Condition (all $ps > .05$). Whereas the graph below suggests the possibility of baseline differences, these were not statistically significant ($p = .55$).

Figure 23. F-EMG trauma script Pre- to Post-Training.



There was not a significant effect of Time ($z = -1.28, p = .201, \text{Cohen's } d = .184, \text{Hedges' } g = .1812$, see Figure 24), Condition ($\eta^2 = 4.11, p = .1284$), or a Time x Condition interaction ($\eta^2 = 2.89, p = .2358$) on F-EMG during trauma imagination. None of the simple slopes were significantly different than zero or significant different by Condition (all $ps > .05$). Whereas the graph below suggests the possibility of baseline differences, these were not statistically significant ($p = .14$).

Figure 24. F-EMG trauma imagine Pre- to Post-Training.



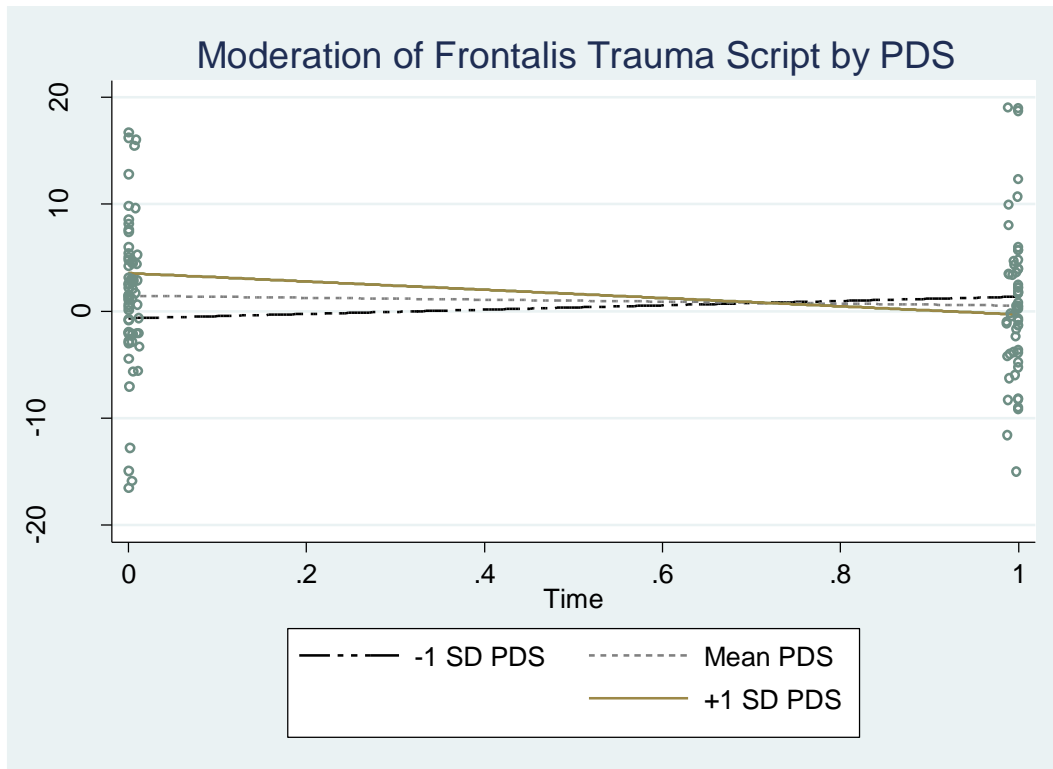
There was not a significant effect of Time ($z = .10, p = .92$, Cohen's $d = -.02$, Hedges' $g = -.02$), Condition ($\eta^2 = .09, p = .96$), or a Time x Condition interaction ($\eta^2 = .39, p = .82$) on F-EMG during the neutral script. There was not a significant effect of Time ($z = .90, p = .37$, Cohen's $d = -.17$, Hedges' $g = -.17$), Condition ($\eta^2 = .324, p = .20$), or a Time x Condition interaction ($\eta^2 = .302, p = .22$) on F-EMG during neutral imagination. None of the simple slopes were significantly different than zero or significant different by Condition for either F-EMG during the neutral script or imagination (all $ps > .05$).

Moderators of Pre-Post Physiological Analyses by Trauma Severity

PDS did not moderate the relationship between Condition, Time, and F-EMG ($\eta^2 = 1.14, p = .565$) or Condition and F-EMG ($\eta^2 = 2.86, p = .2397$) during the trauma script. However, PDS moderated the relation between Time and F-EMG during the trauma script ($z = -2.91, p < .01$; see Figure 24). In addition, there was a main effect of PDS controlling for Time, Condition, and the Time X PDS interaction ($z = 2.58, p < .05$). The simple slope of PDS at 1 SD above the mean was

negative and significant ($p < .05$), whereas the slopes at the mean or lower on PSD were not ($p > .05$). As demonstrated in the graph below, participants with higher baseline PDS had steeper reductions in F-EMG during the trauma script compared to those at 1 SD below the mean on PDS ($p < .01$).

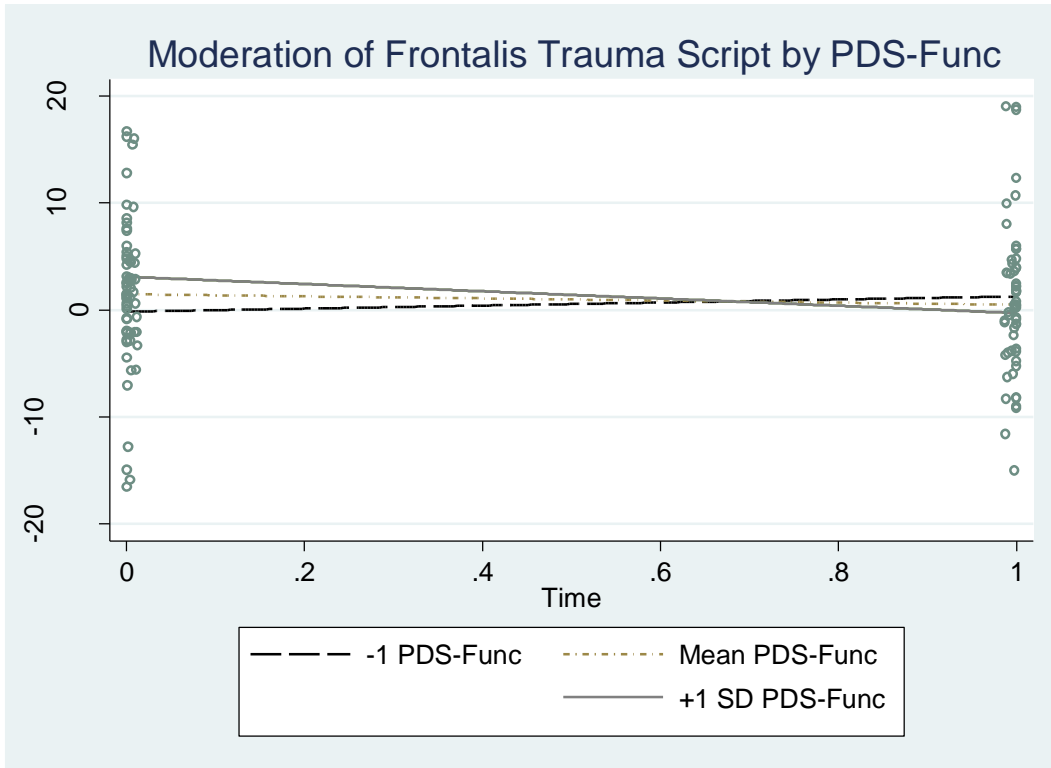
Figure 24. Moderation of F-EMG trauma script by PDS.



Finally, PDS-Func did not moderate the relation between Time, Condition, and F-EMG during trauma script ($\eta^2 = 5.59, p = .0611$) or between Condition and F-EMG during the trauma script ($\eta^2 = .60, p = .7408$). There was a Time X PDS-Func interaction on F-EMG during the trauma script ($z = -2.18, p < .05$; see Figure 25), but not a main effect of PDS-Func controlling for Time, Condition, and the Time X PDS-Func interaction ($z = 1.92, p = .055$). The simple slope for PDS-Func at 1 SD above the mean was negative and significant ($p < .05$), whereas the slopes at

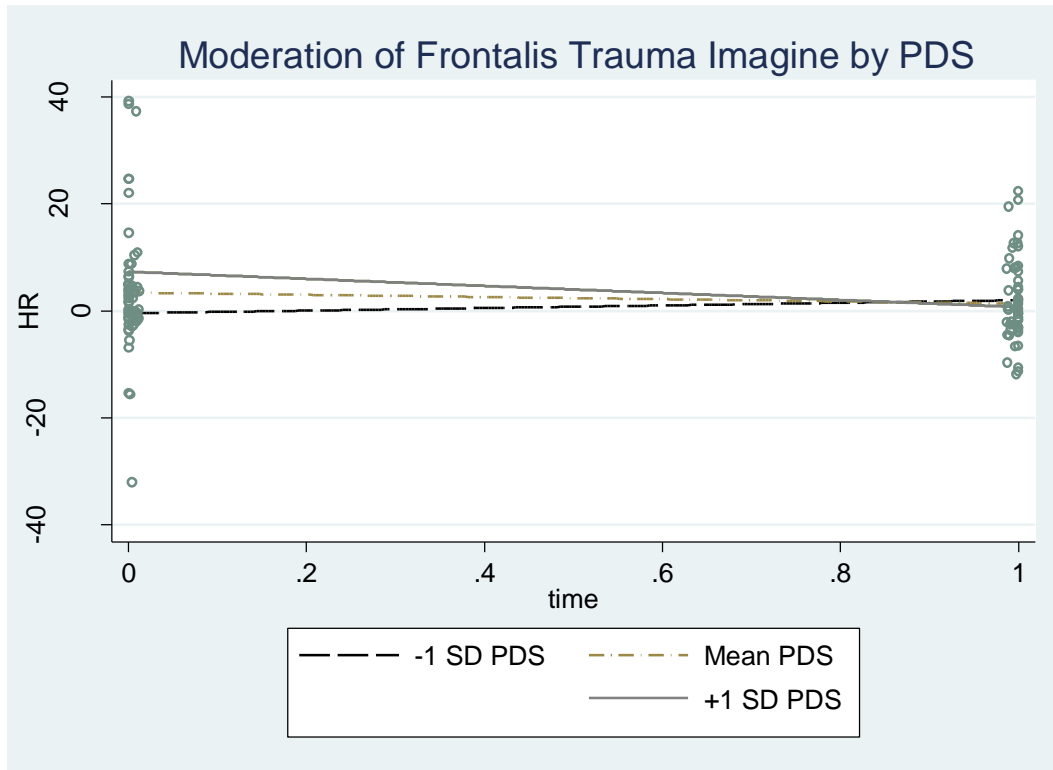
the mean or lower on PDS were not (all $ps > .05$). The simple slopes for PDS-Func at 1 SD below and above the mean were significantly different ($p < .05$).

Figure 25. Moderation of F-EMG trauma script by PDS-Func.



PDS did not moderate the relationship between Condition, Time, and F-EMG ($\eta^2 = 3.02$, $p = .2213$) or Condition and F-EMG ($\eta^2 = 3.67$, $p = .1595$) during the trauma imagination. However, PDS moderated the relation between Time and F-EMG during the trauma imagination ($z = -3.12$, $p < .01$, see Figure 26). The simple slope for PDS at 1 SD above the mean was negative and significantly different than zero ($p < .05$), whereas the slope for PDS at the mean or 1 SD below the mean was not ($p > .05$). The simple slope for PDS at 1 SD above the mean was significantly different than those with PDS 1 SD below the mean ($p < .05$).

Figure 26. Moderation of F-EMG trauma imagine by PDS.

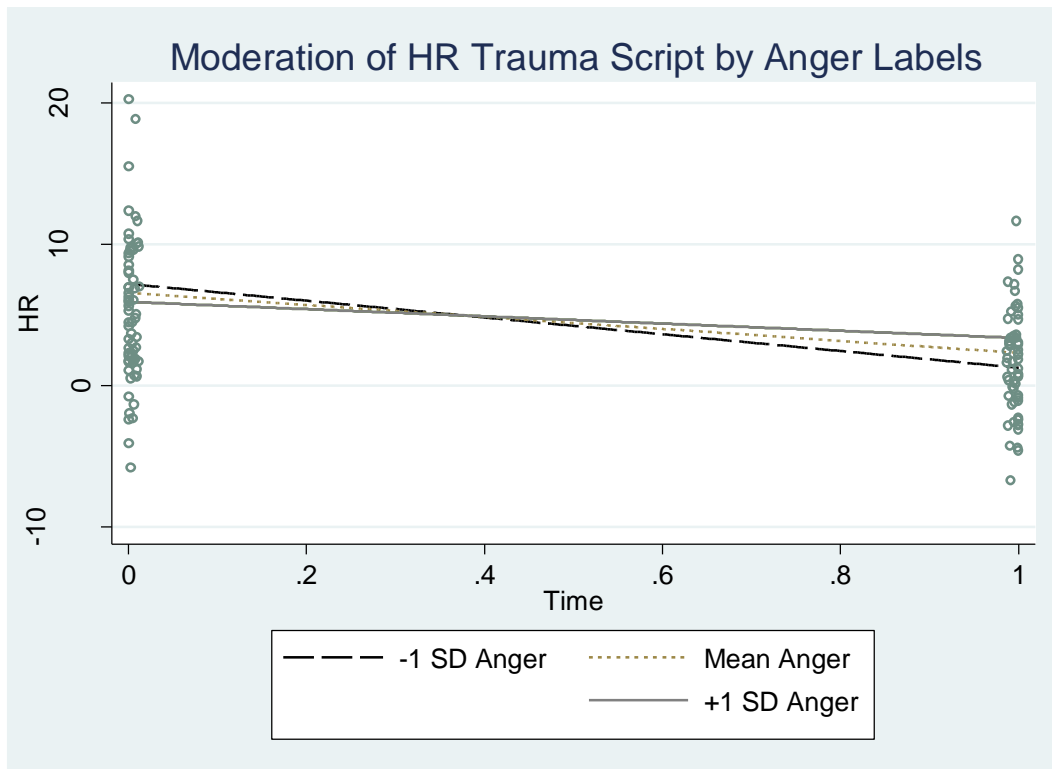


Impact of Labeling on Outcome

Within the Affect Labeling group, there was a main effect of using Anxiety Labels on HR during the trauma script ($z=-2.84, p<.01$) such that those who used more Anxiety Labels during SDI-T had significantly lower HR overall (see Table 14 below). However, there was not a Time x Anxiety Label interaction on HR during the trauma script ($z=.58, p=.565$). Similarly, there was a main effect of Anxiety Labels on F-EMG during both the trauma script ($z=-2.42, p<.05$) and trauma imagination ($z=-2.23, p<.05$), but no Time X Anxiety Label interaction on F-EMG. There was a main effect of Sad Labels on F-EMG during trauma imagination ($z=2.57, p<.01$) such that greater use of Sad Labels predicted higher overall levels of F-EMG reactivity.

Anger Labels significantly moderated the interaction between Time and HR during the trauma script ($z=2.56, p<.05$, see Figure 27). The simple slopes were significantly different than zero at 1 SD below, above, and at the mean ($p<.01$). The simple slopes were significantly different between those at 1 SD above and below the mean on Anger Labels ($p<.05$), with a steeper slope for 1 SD below the mean. However, there was not a main effect of Anger Labels on HR during the trauma script ($z=-.78, p=.436$), and there were not effects of Sad Labels on HR during the trauma script (see Table below 14).

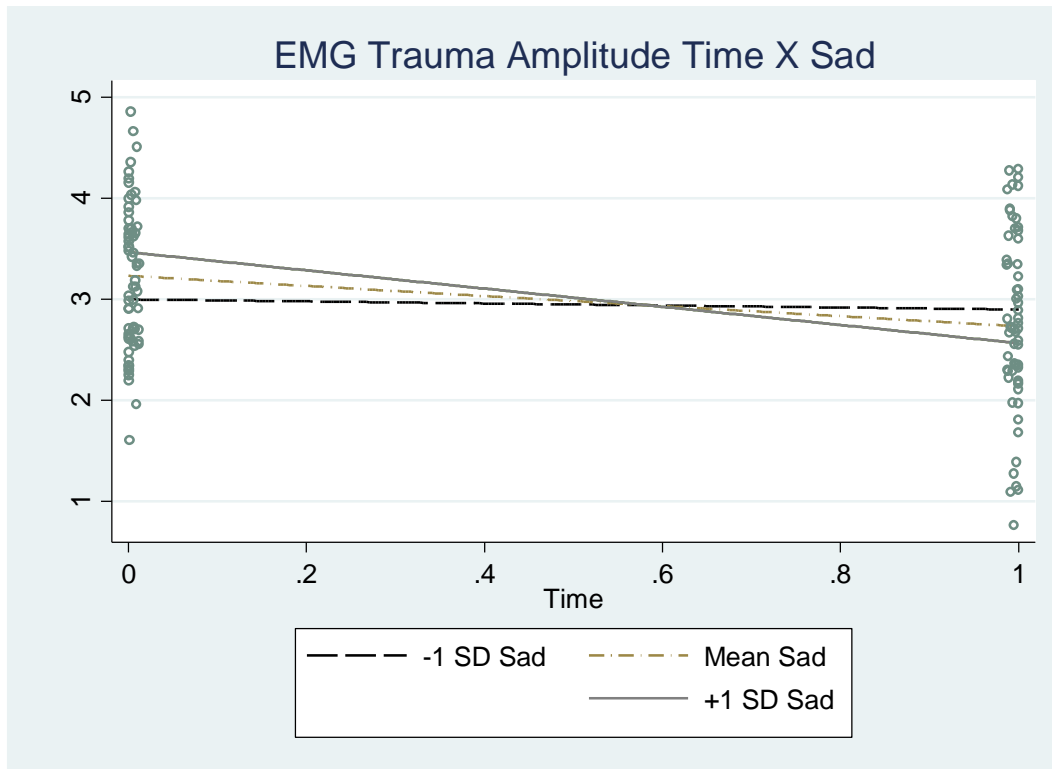
Figure 27. Moderation of HR trauma script by Anger.



Sad Labels were a significant moderator of the relationship between Time and EMG Amplitude during trauma imagination ($z=-2.47, p<.05$, see Figure 28). The simple slopes were significantly different than zero at the mean and 1 SD above the mean on Sad Labels ($ps<.01$), but not at 1 SD below the mean ($p=.657$). The simple slopes between those at 1 SD below and 1

SD above the mean on Sad Labels were significantly different from each other ($p < .05$), with the slope for 1 SD above the mean on Sad Labels significantly steeper than 1 SD below the mean on Sad Labels. However, there was not a significant main effect of Sad Labels ($z = 1.65$, $p = .098$). EMG Integral during trauma imagination was not moderated by any Affect Labeling categories.

Figure 28. Moderation of EMG Amplitude by Time X Sad Labels.



Finally, Anger Labels were a significant predictor of overall PDS ($z = 2.00$, $p < .05$), with higher reporting of Anger Labels associated with higher PDS. However, there was not a Time X Anger Label effect on PDS ($z = -.19$, $p = .851$).

Table 14. Moderation of Physiology by Affect Labels.

Outcome	Anxiety	Anger	Sadness
HR Trauma Script			
<i>Time</i>	$z=-5.69, p<.001$	$z=-6.52, p<.001$	$z=-5.69, p<.001$
<i>Emotion</i>	$z=-2.84, p<.01$	$z=-.78, p=.436$	$z=.57, p=.571$
<i>Time X Emotion</i>	$z=.58, p=.565$	$z=2.46, p<.05$	$z=-.63, p=.529$
HR Trauma Imagine			
<i>Time</i>	$z=-5.47, p<.001$	$z=-5.47, p<.001$	$z=-5.47, p<.001$
<i>Emotion</i>	$z=-1.15, p=.250$	$z=.72, p=.471$	$z=.46, p=.649$
<i>Time X Emotion</i>	$z=.47, p=.637$	$z=1.86, p=.063$	$z=-1.44, p=.149$
EDA Trauma Script			
<i>Time</i>	$z=-.38, p=.704$	$z=-.38, p=.704$	$z=-.38, p=.704$
<i>Emotion</i>	$z=.36, p=.718$	$z=-.34, p=.733$	$z=.26, p=.792$
<i>Time X Emotion</i>	$z=-.61, p=.543$	$z=.71, p=.478$	$z=-.39, p=.694$
EDA Trauma Imagine			
<i>Time</i>	$z=-1.17, p=.243$	$z=-1.17, p=.243$	$z=-1.17, p=.243$
<i>Emotion</i>	$z=.05, p=.962$	$z=-.43, p=.664$	$z=.55, p=.584$
<i>Time X Emotion</i>	$z=-.52, p=.601$	$z=.95, p=.343$	$z=-.65, p=.516$
EMG-Amplitude Trauma Imagine			
<i>Time</i>	$z=-2.67, p<.01$	$z=-2.67, p<.01$	$z=-3.26, p<.01$
<i>Emotion</i>	$z=-.85, p=.396$	$z=-.38, p=.707$	$z=1.65, p=.098$
<i>Time X Emotion</i>	$z=-.13, p=.898$	$z=-.07, p=.941$	$z=-2.47, p<.05$
EMG-Integral Trauma Imagine			
<i>Time</i>	$z=.66, p=.511$	$z=.68, p=.497$	$z=.67, p=.506$
<i>Emotion</i>	$z=-1.65, p=.098$	$z=.15, p=.878$	$z=.47, p=.638$
<i>Time X Emotion</i>	$z=-1.12, p=.262$	$z=.19, p=.852$	$z=-.45, p=.656$
F-EMG Trauma Script			
<i>Time</i>	$z=-.68, p=.497$	$z=-.64, p=.521$	$z=0.62, p=.532$
<i>Emotion</i>	$z=-2.42, p<.05$	$z=-.64, p=.521$	$z=1.58, p=.114$
<i>Time X Emotion</i>	$z=-1.34, p=.181$	$z=.75, p=.451$	$z=-1.35, p=.178$
F-EMG Trauma Imagine			
<i>Time</i>	$z=-1.27, p=.205$	$z=-1.24, p=.217$	$z=-1.25, p=.213$
<i>Emotion</i>	$z=-2.23, p<.05$	$z=-.60, p=.546$	$z=2.57, p<.01$
<i>Time X Emotion</i>	$z=.44, p=.657$	$z=.03, p=.976$	$z=-1.47, p=.142$
PDS			
<i>Time</i>	$z=-2.75, p<.01$	$z=-2.75, p<.01$	$z=-2.75, p<.01$
<i>Emotion</i>	$z=-.26, p=.795$	$z=2.00, p<.05$	$z=-.68, p=.499$
<i>Time X Emotion</i>	$z=.07, p=.942$	$z=-.19, p=.851$	$z=-1.80, p=.072$

Table 14 Note: Significant effects of Affect Labels are presented in bold font throughout.

Effect Size Calculation

While meta-analyses have provided medium effect size estimates for CBT for PTSD (Hedges' $g=.59$), these comparisons are against a placebo condition (Hofmann & Smits, 2008), which was not included in the current study design. Therefore, the six studies included in the meta-analysis were analyzed for Cohen's d and Hedges' g estimates based on Pre- to Post-differences to allow for direct comparison with the current study (see Table 15). The PDS was selected as the measure for effect size analyses because it was used as an outcome measure in one of the comparison studies and because it is a widely used self-report measure in the PTSD literature. As demonstrated below, the results from the current study were in the medium range for both AL and DL conditions on the PDS, and in the small range for the EO condition. These results suggest benefit of linguistic processing during exposure, but suggest that non-affective linguistic processing was more effective in this sample. While effect sizes were larger for many of the comparison conditions, they were comparable for the McDonagh et al. (2005) trial and Neuner et al. (2004) trials. This is notable given that the McDonagh and colleagues (2005) trial included 38.5 hours of therapy, compared to the 90 minute Training provided in the current study.

Table 15. Effect size estimates for the current study compared to published studies from Hofmann & Smits (2008).

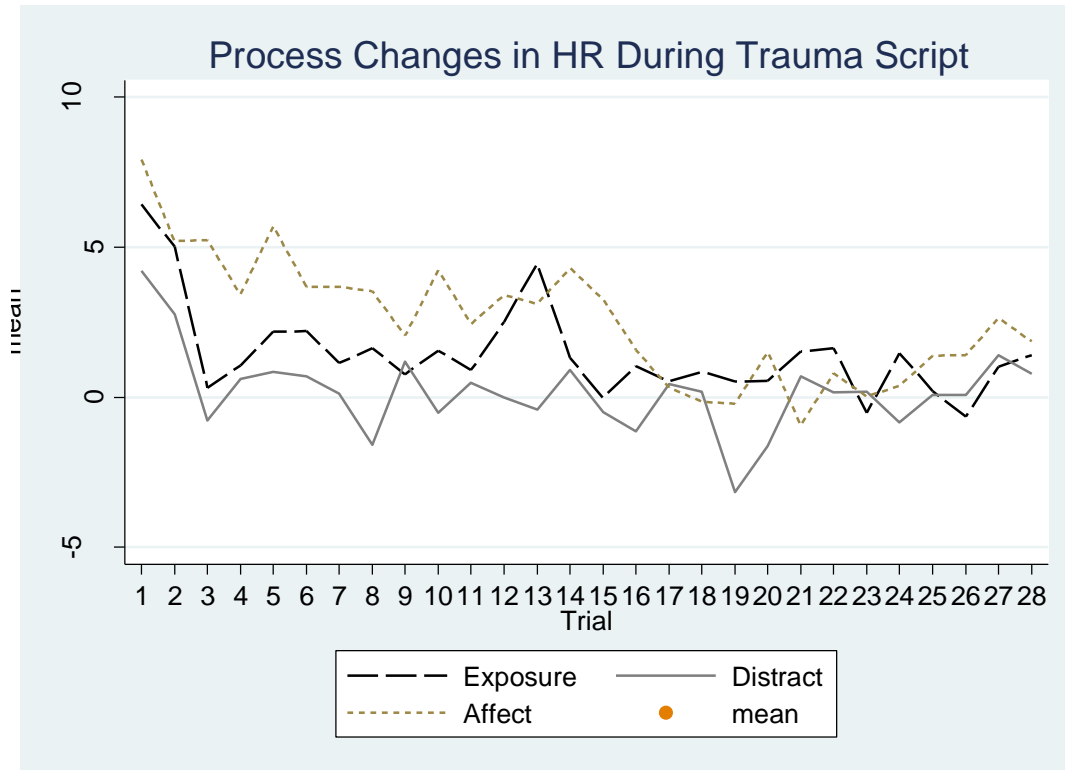
Study	# <i>d</i>	= <i>g</i>	<i>n</i>	# Sessions	Sample
Blanchard et al. (CAPS; 2003)					
CBT	1.78	1.75	27	8-12 sessions, duration not reported	Motor-vehicle Accidents
Bryant et al, 2003 (IES-Intrusion; 2003)					
IE only	0.86	0.8432	20	8, 90 minutes	Civilian Trauma Survivors
IE + Cognitive Restructuring	1.11	1.09	20	8, 90 minutes	Civilian Trauma Survivors
Foa et al. (RAST; 1991)					
PE	1.2	1.05	10	9, 60 minutes	Rape victims
Marks et al. (CAPS; 1998)					
Exposure	1.02	1	20	10, 90 minutes	Civilian Trauma Survivors
Cognitive Restructuring	1.57	1.53	18,19	10, 90 minutes	Civilian Trauma Survivors
Exposure + CR	1.12	1.09	19,20	10, 105 minutes	Civilian Trauma Survivors
McDonagh et al. (CAPS; 2005)					
CBT	0.713	0.703	29	14, first 7: 2 hours, second 7: 1.5 hours	Adult Survivors of Childhood Sexual Abuse
Neuner et al. (PDS; 2004)					
Narrative Exposure Therapy	0.623	0.6084	17	2 sessions, person-dependent until habituation occurred	African Refugee
Current Study (PDS)					
Affect Labeling	0.471	0.462	21	2 sessions, 45 minutes	Civilian Trauma Survivors
Distract Labeling	0.689	0.676	22, 19	2 sessions, 45 minutes	Civilian Trauma Survivors
Exposure Only	0.247	0.241	23, 21	2 sessions, 45 minutes	Civilian Trauma Survivors

Note: Self-report measures of trauma severity were chosen whenever available for closer comparison to the current study. CAPS: Clinician Administered PTSD Severity (Blake et al., 2006); RAST: Rape Aftermath Symptom Test (Kilpatrick, 1988).

Process Analyses

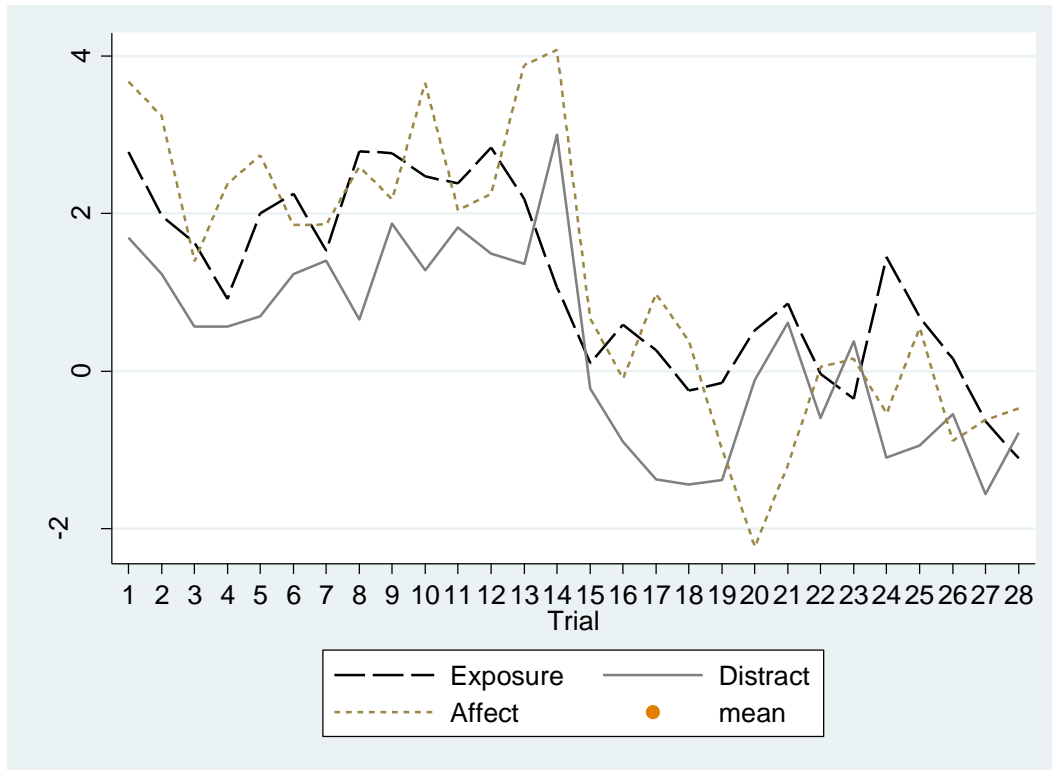
Prior studies have found patterns of non-linear change in gold-standard treatments for PTSD (Nishith et al., 2002). Therefore, these analyses tested the pattern of change in outcome variables across all trials during both SDI-A and SDI-T. For HR during the trauma script there was a significant quadratic effect of Time ($z=4.13, p<.001$, see Figure 29) and an effect of Condition ($\chi^2=7.42, p<.05$) with AL significantly higher than DL ($z= -2.70, p<.01$) but not EO ($z= -1.10, p=.273$), and no differences between DL and EO ($z= -1.66, p=.097$). This difference was likely driven by elevated responding in the AL condition on the first trial of the training compared to both DL ($p<.01$) and EO ($p<.05$; omnibus $F(2,62)=5.96, p<.01$). By trial 2 of the training (trial 4 in the graph below following 2 Pre-SDI-A trials), there are no differences in HR responding to the trauma script by Condition ($F(2,62)=1.33, p=.27$), perhaps suggesting that the baseline difference on the SDI-T reflects some anticipatory anxiety that was not present in the other conditions or in the Pre-SDI-A. There was not a Time X Condition interaction (omnibus $\chi^2=.39, p=.8218$) for HR during the trauma script.

Figure 29. Process changes in HR during trauma script.



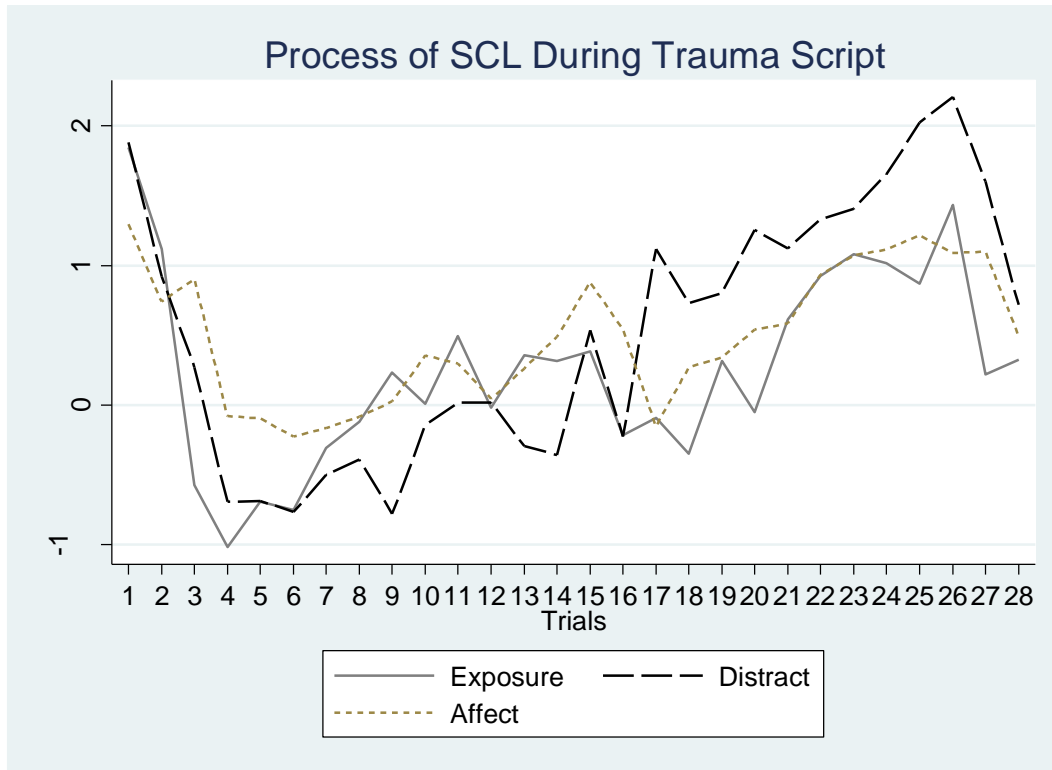
For HR during trauma imagination, there was a significant quintic effect of Time ($z = -5.68, p < .001$, see Figure 30) but not an effect of Condition (omnibus $\eta^2 = 1.80, p = .4065$) or a Time X Condition interaction (omnibus $\eta^2 = .08, p = .9590$).

Figure 30. Process of change in HR during trauma imagination.



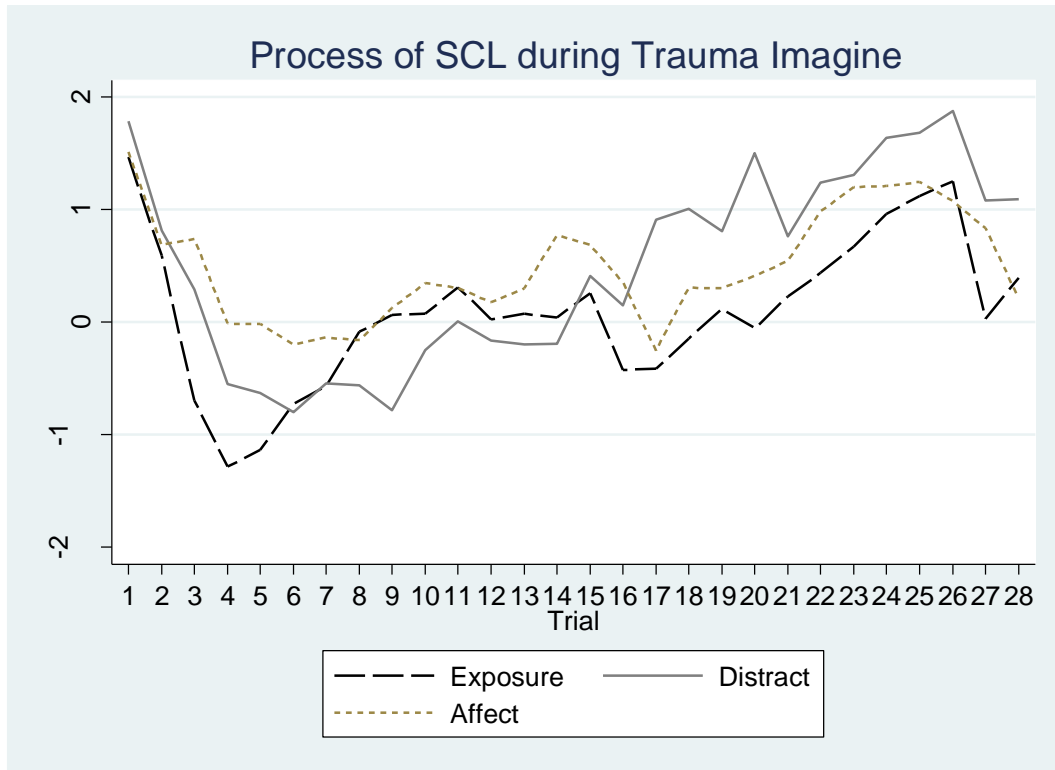
For SCL during the trauma script, there was a significant quintic effect of Time ($z = -5.97$, $p < .001$; see Figure 44) and a quintic Time X Condition interaction ($\eta^2 = 6.14$, $p < .05$), but not a main effect of Condition ($\eta^2 = .37$, $p = .8307$). Tests of simple main effects demonstrated that the Time X Condition interaction was driven by differences between DL and AL ($z = -2.39$, $p < .05$), with no difference between EO and DL ($z = 1.72$, $p = .09$) or DL and AL ($z = -.63$, $p = .53$). While AL was consistently more reactive in the first half of training, differences were reduced in the second half of the training. There was a significant simple slope for EO ($p < .05$) and DL ($p < .05$), but not for AL ($p = .07$), but there were no significant differences in slopes between the groups (all $ps > .05$).

Figure 31. Process of change for SCL during trauma script.



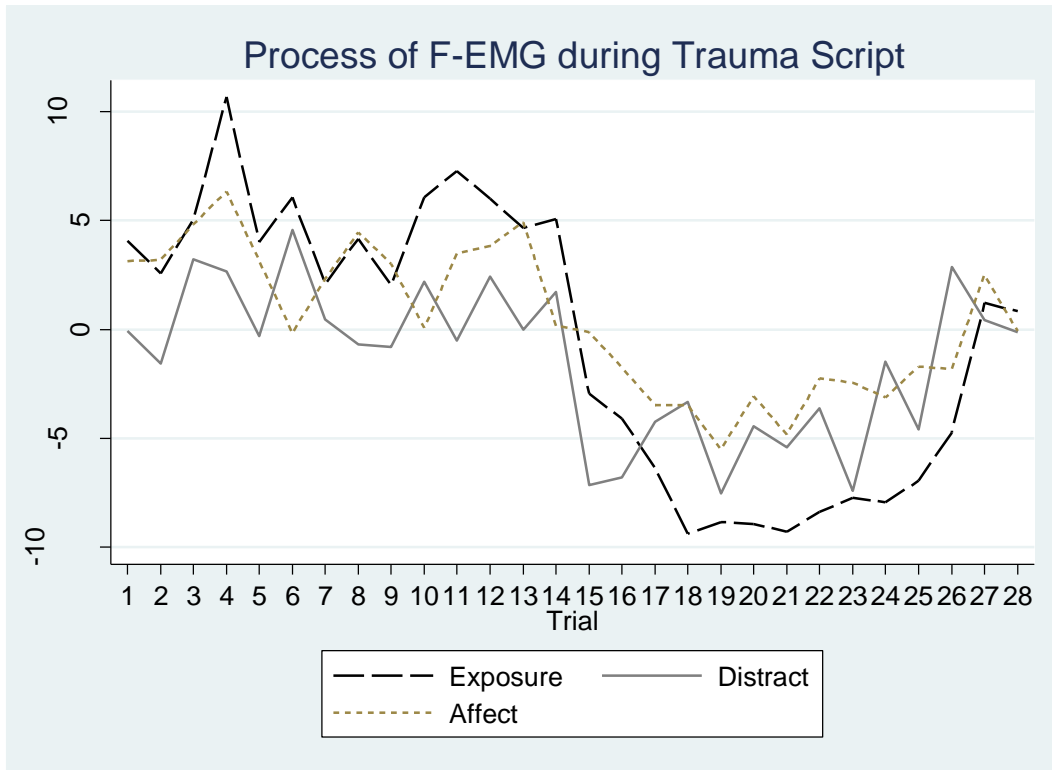
Similarly, for SCL during trauma imagination, there was a significant quintic effect of Time ($z = -5.90, p < .001$, see Figure 32) and a quintic Time X Condition interaction ($\chi^2 = 6.93, p < .05$) but not a main effect of Condition ($\chi^2 = 1.44, p = .4864$). The Time X Condition interaction was driven by differences between EO and DL ($z = 2.63, p < .01$), but there were no differences between EO and AL ($z = 1.38, p = .168$) or between AL and DL ($z = 1.26, p = .209$). Simple slopes were negative for all conditions, but were only significant for EO and AL ($p < .05$; DL: $p = .08$), although there were not differences in simple slope by Condition (all $ps > .05$).

Figure 32. Process of change for SCL during trauma imagine.



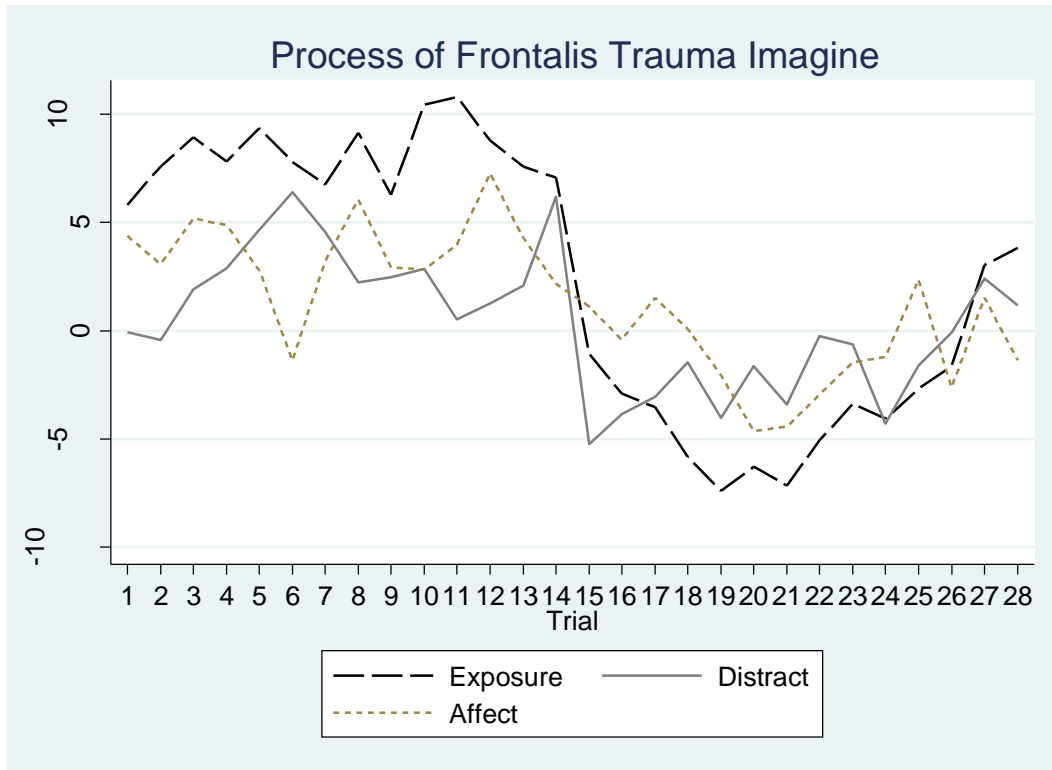
For F-EMG during the trauma script, there was a significant quadratic effect of Time ($z=2.74, p<.01$, see Figure 33) but not a significant effect of Condition ($\eta^2=1.96, p=.3757$) or a quadratic Time X Condition interaction ($\eta^2=4.97, p=.0833$).

Figure 33. Process of change for F-EMG during Trauma Script.



For F-EMG during trauma imagination, there was a significant quadratic effect of Time ($z=-3.04, p<.01$, see Figure 34) but not a main effect of Condition ($\eta^2=3.72, p=.1555$) or a quadratic Time X Condition interaction ($\eta^2=1.58, p=.4529$).

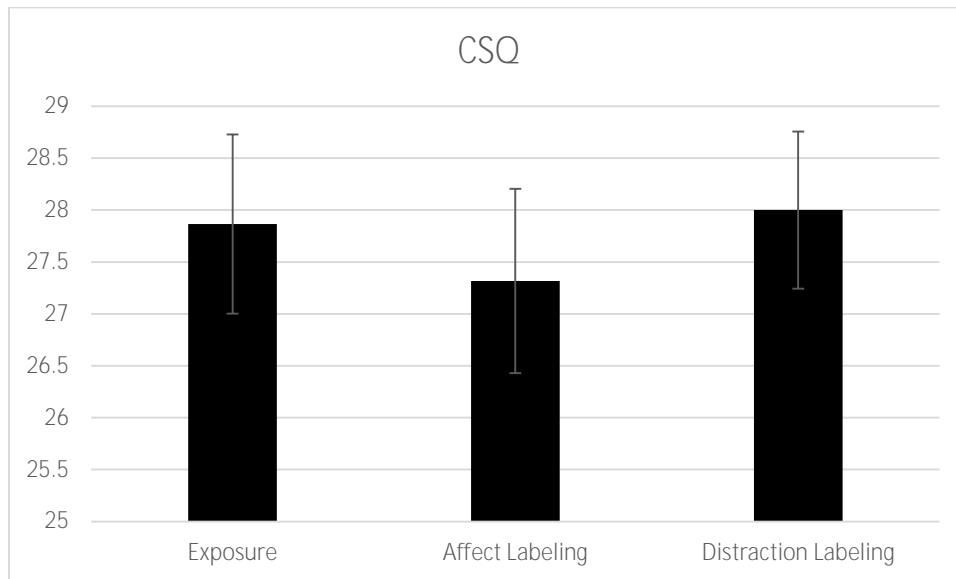
Figure 34. Process of change for F-EMG during trauma imagination.



Participant Satisfaction Ratings

Satisfaction ratings were generally high and there were no differences by Condition in participant satisfaction ($F(2,58)=.18, p=.8320, R^2=.0063$, see Figure 35).

Figure 35. Client satisfaction.



Main Study Discussion

The first goal of the Main Study was to replicate the significant reductions in self-report and physiology found in the Pilot Study in a larger sample recruiting more heavily from community members. Consistent with our hypotheses, there were significant reductions in self-reported distress in terms of trauma severity as measured by two separate self-report measures, the PDS and the IES. There was a trend toward a significant reduction in depression severity, as measured by the BDI, but this effect was not significant. There was also a significant reduction in PDS-Func caused by trauma symptoms from Pre-to Post-Training.

Additionally, there were significant reductions in physiological indices from Pre- to Post-Training. HR response to the trauma script and imagination significantly decreased over time, as did EMG amplitude during trauma imagination. Some of these physiological effects were specific to the trauma memory and were not found during the neutral memory. For instance, there was not a significant reduction over time in HR during the neutral script. This specificity in habituation is consistent with prior research using the SDI procedure, which has demonstrated that following exposure treatment, physiological responding to the exposed trauma memory is lower compared to another non-treated trauma (Shalev et al., 1992). It also demonstrates that participants were not globally less reactive at post-treatment, but that this reduced reactivity was somewhat specific to their trauma reactivity. Future studies should include variations on the trauma memory or memories of multiple trauma to test generalization from the training to non-trained memories.

The second goal of the Main Study was to determine whether Affect Labeling enhances the effect of the Training compared to either Distract Labeling or Exposure Alone. We hypothesized that Affect Labeling would result in lower overall levels of physiological responding at Post-Training. Contrary to our hypothesis, there were no significant main effects of labeling Condition on outcome measures. Similarly, there were no significant interactions between Time and Condition on physiological or self-report measures. However, there were significant negative slopes for both labeling groups but not the Exposure Only group on one index of trauma reactivity, the PDS, and significant negative slopes for only Affect Labeling on another index, the IES. Similarly, there were significant negative slopes for both labeling conditions, but not Exposure Only in EMG Amplitude during trauma imagination, with the Distract Labeling condition receiving the greatest benefit. When the labeling groups were

combined, there was a significant difference in slopes based on Condition, with the Labeling group experiencing a reduction in EMG Amplitude that was not present in No-Label group (i.e., Exposure Only). The interaction between Time and Labeling was significant for EMG amplitude. This suggests that rather than Affect Labeling conferring a specific benefit, linguistic processing more generally was effective at reducing physiological reactivity.

Another goal of the main study was to explore trauma severity as a potential moderator of the relationship between Time and Condition. Trauma severity was a moderator of the relationship between Time and F-EMG during both the trauma script and imagination, such that greater severity was associated with steeper reductions over the course of the Training. These are important findings as they provide justification for studying the application of this Training to more severe populations.

A secondary goal of the Main Study was to explore how word choice within the Affect Labeling Condition moderated outcome. Consistent with our hypotheses, greater use of Anxiety labels was associated with lower trauma reactivity, both in terms of HR and F-EMG. This finding reflects two possibilities. The first is that those who were expressing the most anxious labels were experiencing the greatest attenuation of physiological responding through downregulation of the amygdala and other brain regions associated with these measures. The second is that those who report anxiety emotions are less severe than those reporting more Anger or Sad labels. The latter hypothesis was not supported by relating Anxious words to either Pre- ($p=.367$) or Post-training severity ($p=.298$) in the current sample, but future research should investigate this question within PTSD specifically. There was a significant interaction of anger with Time on HR, and a significant main effect of Sad Words on F-EMG. Perhaps those engaging in emotions of Sadness and Anger are experiencing increases in rumination about the

traumatic experience, which serves as an avoidance tactic (Cribb, Moulds, & Carter, 2006) and prevents the development of inhibitory learning (Pitman et al., 1991). Fear and arousal may be maintained in those with high ruminative coping tendencies because of biased attention toward confirmatory evidence for their fears, like interoceptive cues, to the exclusion of contradictory evidence (Davey, 1995). As discussed in the introduction, those with enhanced trauma reactivity have biased attention toward threat and difficulty disengaging from threatening stimuli (Fani et al., 2012). Alternatively, perhaps those who report greater degrees of Anger or Sadness may have interpersonal traumatic experiences more frequently than those who do not, or perhaps these traumas are characterized by more loss. The current study was not well-suited to address these questions due to collection of emotional word responses within only the Affect Labeling condition, and future research should address these possibilities. However, these findings are consistent with that of prior research demonstrating that greater Anger was associated with attenuated benefit from treatment (Foa et al., 1995). However, there was a significant interaction between Time and Sadness on fear potentiated startle, suggesting that those with greater reports of Sadness had steeper reductions in startle from Pre-to Post-Training. The main effect for Sadness was marginally significant in this analysis ($p=.098$), and suggested a trend toward overall higher reactivity with greater Sadness. Therefore, perhaps a resolution for these seemingly contradictory findings is that those reporting more Sadness have initially higher reactivity that is alleviated throughout the course of the Training.

Every physiological measure included in this study had significant quartic or quintic effects of time. These statistical findings were always confirmed with visual inspection of the data, and were evaluated using backward elimination of non-significant polynomials, as recommended in the literature (Chan, Kwong, Dillon, & Tsim, 2011). Therefore, while caution

is needed in interpreting these process findings due to the limited sample size, the data overwhelmingly support the inclusion of non-linear reductions across all physiological indices.

Further examination of trial by trial differences in physiological responding revealed main effects of Condition or Time by Condition interactions in addition to the significant effects of Time. In HR during the trauma script, those in the Affect Labeling condition demonstrated significantly greater activation compared to those in the Distract Labeling condition (with no differences between Exposure Only in either comparison). This appears to have been driven by responding on the first trial of the Training, which was elevated in the Affect Labeling condition. The instructions provided between the assessment and Training provided the only methodological deviation between the Conditions at this point in the study. Therefore, these findings reflect elevated anticipatory anxiety in the affect labeling condition. Perhaps participants were anxious to verbalize their emotional experience to the experimenter, or perhaps they were nervous about being mindful of their emotional experience. Another possibility is that the instruction to report on emotional experiencing throughout trauma imagination primed participants in this Condition that the task would be aversive. Alternatively, perhaps individuals in the Distract Labeling condition anticipated that the Training would be less triggering because of engaging in a distracting task. These differences corrected by the second trials, suggesting that the manner of introducing the tasks has an important impact on the Training, but this impact is negligible once the participant has completed the task.

In the process analysis, there were also some significant effects of the Time X Condition interaction. For SCL during the trauma script, there were differences between Affect and Distract Labeling. While Affect Labeling was consistently more reactive in the first half of training compared to Distract Labeling, this pattern reversed in the second half of training. There

were significant simple slopes for Distract Labeling (and Exposure Only) that were not present in Affect Labeling, although there were not significant differences in simple slopes by Condition. As mentioned above, perhaps participants experienced elevated anticipatory anxiety about identifying and expressing their emotional reactions which eventually subsided. For SCL during trauma imagination, there were significant slopes for Exposure and Affect Labeling, but not Distract Labeling. However, there were not significant differences in simple slope by Condition. These results suggest that perhaps the process of change during Training trials varies based on the type of training.

The somewhat contradictory results between HR and SCL are not specific to this study. Decades of research have demonstrated differential patterns of psychophysiological responding between HR and SCL based on the experimental manipulation (Campos & Johnson, 1966). Therefore, some differences in patterns of responding are to be expected. However, it remains unclear why the Distract Labeling condition experienced an increase in SCL reactivity to the trauma script throughout the second day of the training. Future research should replicate this finding before firm conclusions are drawn.

Both labeling conditions performed comparably to two of the six studies included in the Hofmann & Smits (2008) meta-analysis. This is important given the limited session numbers included in this study, less severe sample (and therefore, less potential for change), lack of therapist contact, and reliance on self-report rather than clinician-rated symptom levels. Across all participants, a 50% reduction in symptom severity according to the PDS was detected in 75% of completers. Considering only those who began above the clinical cutoff, 44% no longer met criteria for PTSD according to the PDS. This is consistent with a meta-analysis of treatment response following CBT for PTSD, which reported 47% of patients that had clinically significant

improvement (Bradley et al., 2005). Further, of this more severe sample 72% reported a 50% reduction in symptom severity from pre-to post-training. Therefore, this training was not only able to produce medium effect sizes in symptom reduction, it was also successful in helping some participants transition to remission. It will be important to determine whether there is a dose/response relationship between the training in the current study and outcome, and whether outcome could resemble that of formal CBT with one or two additional sessions. Importantly, whereas the effect size estimates for both Affect Labeling and Distract Labeling were in the medium range, estimates for Exposure Only were in the small range.

Relatedly, there was minimal attrition for participants who began the Training. Out of 66 participants in the Main Study, only 5 discontinued prematurely. As reported above, three of these participants were in the Distract Labeling condition (citing personal events and symptom increase, with one dropping before the first training trial), and two were in Exposure Only (citing a scheduling conflict or not providing a rationale). No participants discontinued prematurely from the Affect Labeling condition. Across all Conditions, 7.6% of participants discontinued prematurely. This is important given that attrition rates for general CBT for anxiety disorders range from 15-30% (Haby, Donnelly, Corry, & Vos, 2006b) and have been reported as problematic in trials of combined emotion regulation training and exposure for PTSD (Bryant et al., 2013).

General Discussion

PTSD is a costly disorder in terms of psychological health, physical health, and economic burden (Solomon & Davidson, 1997). While empirically supported treatments, such as Prolonged Exposure (Foa et al., 1999; Foa & Kozak, 1986), Cognitive Processing Therapy

(Resick & Schnicke, 1992), and traditional CBT (Hofmann & Smits, 2008) are promising forms of treatment, many who complete these treatments remain symptomatic (with some estimates up to 50%; Schottenbauer, Glass, Arnkoff, Tendick, & Gray, 2008), many who are interested in these treatments have financial or logistical barriers preventing access to them (Davis, Ressler, Schwartz, Stephens, & Bradley, 2008), and many are not interested in these treatments due to the stigma associated with them (Ouimette et al., 2011). There is a clear need for further development and dissemination of efficient treatments for individuals with elevated trauma reactivity. The studies included herein demonstrate the promise of brief computerized imaginal exposure for this population.

The Pilot Study had a variety of baseline differences in self-reported and physiological trauma severity prevented compelling conclusions, but it provided initial support for the finding that brief imaginal exposure results in significant reductions in self-reported depression and physiology. Importantly, this training was independently operated with no interference from the researchers. The significant main effects of the timing of labeling demonstrated that engaging in labeling During imagination confers greater benefit than labeling After imagination. Given that amygdala activation is likely to be higher During rather than After imagination, perhaps engaging in linguistic processing During imagination results in greater downregulation of the amygdala (and corresponding reductions in self-report and physiological distress), though this study did not include a brain imaging component to test this hypothesis. This is consistent with the theory that Affect Labeling improves outcome through modulation of amygdala activity by the vIPFC (Lieberman et al., 2007). There were few differences in the Pilot Study by Task. This was a surprising finding, but should be interpreted conservatively given that it did not emerge for any other outcome variables. Finally, the Pilot Study also provided evidence that greater choice

of negative emotion words, relative to neutral emotion words, were predictive of more pronounced improvements in physiological responding.

The Pilot Study inspired several important methodological changes that were adopted in the Main Study. These included randomization stratified by trauma severity, (which successfully reduced baseline differences by Condition), significant changes to the format of Affect Labeling, provision of Affect Labeling choices only During imagination, and the inclusion of an Exposure Only Condition. Given that the Affect Labeling Condition did not enhance outcome in the Pilot Study using a simple forced choice format, the Main Study included more flexibility in emotional responding. Consistent with prior research, participants were provided with the opportunity to freely choose one negative word to describe their emotional experience, and one negative word to describe the event. The Distraction Labeling condition was also re-formatted to allow for free responding to a distraction cue. These more flexible tasks provide the opportunity for enhanced variability in responding, provide more accurate emotional reflections, and allow for linguistic analysis using LIWC software.

The main study demonstrated that 90 minutes of a computerized, user-operated training over the course of three weeks can significantly improve both self-reported and physiological trauma reactivity. Significant reductions in self-reported trauma distress were observed across two measures, as were reductions in impairment resulting from traumatic distress, and a non-significant trend toward reduction in depression. Further, there were significant reductions in physiological responding to both the trauma script and trauma imagination across both heart rate and fear-potentiated startle responding. Importantly, this reduction in responding was largely specific to the trauma memory, as some physiological measures did not habituate to the neutral script whereas they did habituate during the trauma script.

Surprisingly, in the Main Study there were no significant effects of Condition on the Pre- to Post-Training analyses. It is possible that power to detect difference between Conditions was reduced by small sample sizes. Given the novelty of this Training and the baseline differences that precluded strong statistical comparison in the Pilot Study, power analyses were not conducted prior to recruitment. Several researchers argue that post-hoc power analyses are biased and do not provide an accurate estimate of true power, and instead these analyses reflect information already provided with effect size estimates and significance levels (Goodman & Berlin, 1994; Hoenig & Heisey, 2001; Levine & Ensom, 2001; Zumbo & Hubley, 1998). Effect sizes from the Main Study will leverage power analyses for subsequent studies.

Other samples that have demonstrated benefits of Affect Labeling have used physical or visual cues during exposure, such as images of spiders (Tabibnia et al., 2008), live spiders (Kircanski et al., 2012), or delivery of a speech in front of an audience (Niles et al., 2015). Perhaps Affect Labeling is too cognitively demanding to be a successful augmentation strategy during the already-demanding imaginal exposure involved in PTSD treatment. Participants were instructed to cease imagining while engaging in Affect Labeling, yet perhaps this is an unrealistic task demand causing some participants to simultaneously visualize their trauma while attempting to generate a descriptive sentence. This added cognitive demand might have potentially reduced any added benefit of the training. The literature on detrimental costs of multitasking suggests that dual performance in any two cognitively demanding tasks typically results in reduced performance compared to performance of a single task (Finley, Benjamin, & McCarley, 2014), providing further support for this possibility.

However, these studies collectively demonstrate benefits for general (i.e., non-affective) labeling relative to no labeling. Prior research has also found benefit of labeling in an

unexpected direction, in that irrelevant negative labels are effective at reducing physiological responding relative to relevant negative labels, neutral labels, and exposure only labels in terms of both HR and skin conductance response (Tabibnia et al., 2008). In addition, categorizing distressing images along a “Natural” or “Artificial” dimension results in downregulation of the amygdala, recruitment of the bilateral ventral PFC, and lower SCL relative to shape-match (Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003). Further, linguistic processing writing interventions have demonstrated efficacy across clinical populations for decades (Pennebaker, 1997). It is likely that trauma stimuli are more distressing than other previously studied stimuli, and this may have changed the effect of Affect Labeling. The results of these studies should be replicated in larger populations before strong conclusions are drawn, but these findings collectively suggest that linguistic processing may improve the outcome of exposure-based Training.

Another possibility for the lack of significant findings for Affect Labeling relative to other populations may be related to the reduced ability of trauma-exposed individuals to identify, categorize, and express their emotions. PTSD is highly associated with the presence of alexithymia (Zlotnick et al., 2001), or the inability to detect and experience emotions, and degree of alexithymia is related to trauma severity (Zlotnick et al., 2001). Alexithymia was not measured in the current sample, but based on the prior literature, at least some proportion of participants in the current studies might have had difficulty with emotion identification. This deficit likely includes challenges with categorizing emotional experiences based on cognitions, physiological sensations, and behavioral urges. Therefore, perhaps additional psychoeducation around nuanced emotional experiencing and expression might compound the benefit of this computerized training. There is a precedent for providing skills training in emotion

identification/regulation prior to exposure therapy for PTSD, and these studies typically result in improved outcomes (Bryant et al., 2013). Therefore, perhaps the provision of psychoeducation around emotional experiencing and expression prior to engagement in Affect Labeling during imaginal exposure might enhance outcomes in the future.

Emotional expression with the context of PTSD is as highly varied as are the kinds of trauma one can encounter. Some individuals with PTSD over-rely on the expression of secondary emotions, such as anger, relative to primary emotions, such as fear and sadness (Fitzgibbons, 1986; Riggs et al., 1992) because the primary emotions increase feelings of vulnerability. Other investigations have noted the theoretical possibility that activation of anger inhibits the susceptibility of an excitatory fear association to extinction because of the ability of anger to inhibit fear expression (Riggs et al., 1992). Therefore, perhaps the emotional expression task included in this study was too flexible in that participants could choose to engage in only secondary emotional expression for each trial. Psychoeducation on the function behind primary and secondary emotions might limit this tendency in future studies, as might more specific prompts that encourage vulnerable emotion expression. Further, models that encourage attention allocation toward primary emotions or reinforce the expression of more vulnerable genuine emotions may enhance outcomes. The expression of more vulnerable emotions might predispose participants to habituation, whereas the expression of anger-related emotions might predispose individuals to experience enhanced physiological activation (Everson, Goldberg, Kaplan, Julkunen, & Salonen, 1998). Between-subject variability in emotional expression might have washed out any benefit of affect labeling. A final consideration on this topic is that while the experimenter was not involved in the training, the participant was being observed from behind throughout the training to ensure safety and to record labeling choices. This may have created an

opportunity for socially desirable emotional expression that might not have occurred had the participant been sitting alone. Future studies should provide the opportunity for participants to report their emotional experiences privately to avoid this confound.

Trauma severity and impairment were both important moderators of outcome in a direction consistent with our hypothesis. Those who had higher severity in terms of trauma distress and impairment benefitted the most from the training. Clinicians are frequently concerned about the potential for enhancing distress through exposure therapy, and they may feel that this concern is especially justified for their most severe patients (Becker, Zayfert, & Anderson, 2004). These results justify continued study of the effects of a computerized training for trauma using the SDI procedure in a more severe population.

The use of negative emotional word choice was an important predictor of outcome in the current study. Greater choice of anxiety words predicted lower physiological reactivity overall, although there was not a significant interaction with time. Relatedly, those who reported more sadness and anger had worse outcomes across a variety of measures. There was a significant interactions of anger with Time effect and significant main effects of Anger on physiological reactivity.

Prior research on the naturalistic course of PTSD symptoms suggest that while symptom distress is remarkable stable over time, with some participants continuing to report distress up to 50 years post-trauma (Gold et al., 2000), there are fluctuations in degree of distress related to PTSD at any given time (O'Donnell et al., 2007). In treatment studies for PTSD, non-linear change is a common phenomenon (Nishith et al., 2002). Additionally, basic experimental research highlights differences in within-session habituation, or reductions in fear from the

beginning of one phase to the end of that phase, and between-session habituation, or reductions in initial fear activation from the beginning of one extinction phase to the beginning of the next (Plendl, 2010). These principles of extinction learning naturally allow for increases in distress at the beginning of an extinction phase compared to the end of the prior phase (Plendl, 2010), though the fear level throughout extinction training typically attenuates. Therefore, non-linear change throughout future studies of similar training will likely be non-linear in nature.

Given the sensitive nature of the study population, the experimenter took extra precautions to ensure the safety and comfort of participants. These included programming a “pause” function into the training/assessment protocol that could allow participants to stop and start the experiment as needed. This function was only used for one participant who decided to stop the experiment prior to the first training trial. Further, a crisis training protocol was developed to handle extreme reactions of duress to the experiment. This procedure was not needed at any point in either study. Therefore, whereas some clinicians might have concerns about the potential to worsen symptoms through a brief exposure training in the absence of supportive therapy, the training significantly improved symptom distress. This finding, if replicated with a more severe population, provides further support that despite clinician reluctance to provide exposure therapy for fears of worsening symptoms (Cook et al., 2004), even extremely time limited exposure that did not allow for full habituation was not iatrogenic.

Rather than the training worsening symptoms as some might have hypothesized, it resulted in effect size estimates comparable to that of recent meta-analyses of gold-standard treatments for PTSD. As mentioned in the introduction, Hedges’ g estimates combining CBT for PTSD are around .62 (Hofmann & Smits, 2008) from pre-to post-treatment (compared to placebo) following ten 90 minute sessions on average. The effect sizes demonstrated herein are

comparable to two of the six studies included in the Hofmann & Smits (2008) meta-analysis, further supporting the potential benefit of this Training.

Even in the event that the SDI Training requires the same number of sessions as traditional CBT to achieve comparable outcomes, the format of the Training enhances its accessibility. There is a clear need to expand evidence-based treatment options outside of major metropolitan areas and computer-delivered trainings provide this possibility. While computer-assisted treatments have been developed and tested for PTSD, some have not resulted in significant reductions in trauma severity, whereas the same protocol resulted in significant reductions in all other anxiety disorders studied (Craske et al., 2011) though this may have been due to reduced power as effect size estimates were comparable across anxiety disorders by follow-up. Furthermore, the sample in the Craske and colleagues (Craske et al., 2011) sample was more severe than the current sample. Other protocols have been developed that have used smartphone application technology to supplement in-person prolonged exposure (Reger et al., 2013) or other outpatient treatments for PTSD (Erbes et al., 2014), though the efficacy of these supplemental techniques has not yet been reported. Proposals have been submitted for conducting formal reviews of computerized and internet-delivered PTSD treatment compared to traditional delivery formats, but these results are still in progress (Lewis, Roberts, Bethell, & Bisson, 2015). Therefore, while other computerized supplemental approaches for CBT are in development, the current study is novel in its use of an efficacious stand-alone computerized Training without therapist involvement.

All experimental conditions reported high satisfaction ratings. These ratings collectively reflect quality of service, the service meeting participants' needs, willingness to recommend the program to friends, and satisfaction with the amount of help received. Importantly, satisfaction

ratings did not differ based on experimental condition. These findings attest to the acceptability and face validity of the training, and provide initial evidence to support the feasibility of this Training in similar samples.

There was low attrition across both studies. In general, attrition can be due a variety of factors that vary in their relation to the treatment provided. Some examples include logistical barriers, lack of treatment response, symptoms worsening, dislike of treatment providers/research staff, or reduced motivation to participate. It is also common for participants to not provide a reason for study drop out and to simply not return for appointments. The reasons cited in this study included both logistical concerns, such as a scheduling conflict and personal events that precluded continued participation, concerns with treatment response, including an increase in nightmares and an unwillingness to begin Training trials, and unstated reasons. While few participants dropped out overall, no participants in the Affect Labeling condition discontinued. This provides tentative evidence that the Affect Labeling condition might have been more acceptable compared to Distraction or Exposure Only. The reduced attrition might have been attributable to the inclusion of participants without diagnoses of PTSD, therefore reducing the overall severity of the sample. Alternatively, the delivery format of this Training might have been more manageable given the limited session duration and frequency. A final possibility is that the computerized format of the Training, the efficiency of imaginal exposure, or some combination might increase participant acceptability.

The current studies included all intent to treat participants, regardless of attrition status. This is the standard recommendation for clinical trials, as these provide more conservative estimates of effect size (Armijo-Olivo et al., 2009; Gupta, 2011). Even with this more conservative estimate, significant effects were found in both studies across a variety of measures.

One consideration about the sample included in both studies is that participants were not treatment seeking and may have had motivations other than managing trauma reactivity for participating. As with many studies, these motivations include the provision of course credit or minimal payment for compensation. The promising data provided herein warrants continued exploration of the feasibility of delivering the training in a treatment seeking population. However, the significant response of the training in participants who were not treatment-seeking raises some interesting possibilities. One possibility is that response will be inflated in treatment-seeking samples due to improved outcome expectancies. In other words, participants who are interested in receiving an empirically-supported treatment for PTSD will likely generate larger effect sizes relative to participants who are not interested in treatment. In anxiety disorders treatments, improvements in outcome expectancy are predictive of improved symptom (Brown et al., 2014). Another possibility is that response rates will be reduced given that participants will be more severe and present with more intractable symptoms, or they might be seeking treatment after having not responded to traditionally provided services, again representing a more challenging treatment course. All of the participants in the current studies scored above the clinical cutoff on the Posttraumatic Checklist, indicating that they were experiencing elevated levels of distress tied specifically to their trauma. Unfortunately, these studies did not collect treatment history data, though willingness to participate in a research program about trauma suggests the possibility that current or past treatments offered did not fully resolve distress. These findings highlights the severity and range of trauma experienced in undergraduate and community participants who might not independently seek treatment, and provides justification for further outreach about empirically-supported treatments for trauma survivors in a college population.

There are several important limitations of this study. Firstly, whereas all participants reported trauma reactivity above a clinical cutoff on the PTSD Checklist (Blanchard et al., 1996), only 40% of participants met criteria for PTSD on a clinician-rated diagnostic scale. Therefore, this is a less severe population compared to other training trials. However, this provides suitable pilot data suggesting that the training significantly improves trauma reactivity in people who are reporting distress about their trauma. Another limitation of this study is that resources did not permit the inclusion of a longer-term follow-up. As mentioned in the introduction, return of fear following extinction training or exposure therapy is more the rule than the exception, with some estimates in randomized controlled trials in the range of 19-62% for even the gold-standard treatments for anxiety disorders (Craske & Mystkowski, 2006). Future research should include a follow-up to determine whether gains are maintained, enhanced, or reduced by this Training. It is not clear whether the inhibitory learning trained in this study will generalize to other related trauma memories, cues that were not included in the trauma scripts, or other trauma memories. Clinical trials have demonstrated that exposure therapy for one category of feared stimuli commonly reduces distress and arousal about other stimuli (Craske et al., 2007). These findings suggest that resources should be invested in treating one content area in individuals with multiple feared stimuli rather than dividing resources between the stimuli. The small sample size that included mostly college students included in this study warrant caution in translating the results broadly, though these results are promising and justify additional research.

There are many important clinical implications from this project. This study suggests that 24 1-minute trials of imaginal extinction Training are suitable for reducing self-reported and physiological trauma reactivity. At the least, the promising findings should alleviate some long-standing clinician concerns about the potential for traditional exposure therapy to worsen

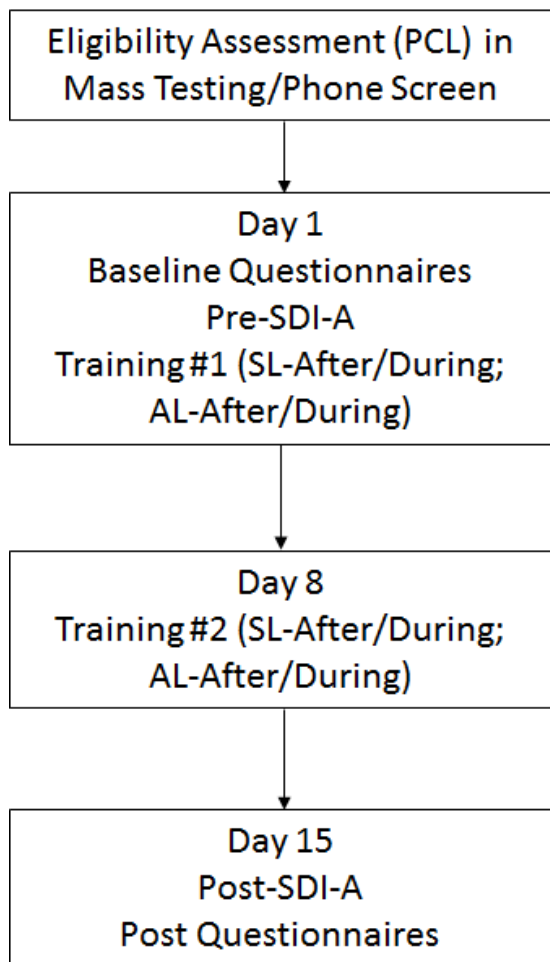
symptom distress. A survey of 207 psychologists from a variety of settings, mostly in private practice, revealed that 72% reported feeling “not at all comfortable” with the use of imaginal exposure in PTSD, and 24% reported being “not at all familiar” with this treatment (Becker et al., 2004). Collectively, these clinicians cite concerns about lack of training, fears of patient decompensation, and a dislike of manualized treatments, among others. Similarly, many therapists who incorporate exposure into clinical practice continue to operate under the principles originally prescribed by Emotional Processing Theory (Foa & Kozak, 1986). This theory purports that long-term fear reduction requires that patients remain in the feared situation until fear subsides. However, an overwhelming number of recent studies (Baker et al., 2010; Brown, LeBeau, Chat, & Craske, in press; Culver, Stoyanova, & Craske, 2012), including the current study, dispute this claim. Traditional Prolonged Exposure (PE) imaginal exposures extend therapy sessions to 1.5 hours in order to allow for habituation. In contrast, this study provided 1-minute extinction training regardless of fear level. Future analyses will explore a variety of indices of inhibitory learning, including within session habituation, between session habituation, and inter-trial variability in responding (an index of expectation violation; Brown et al., under review) throughout the training as a predictor of performance at post-training.

While this study provides promising findings about the feasibility and efficacy of the SDI Training, future research is necessary to expand the impact of the training. Firstly, more severe populations should be recruited in the next iteration of this project to determine whether the Training remains acceptable and effective. Secondly, psychoeducation in emotional experiencing, particularly as it relates to primary and secondary emotional experiencing, should be provided in the next phase of experimentation. Finally, a longer-term follow-up should be included to see whether any benefit from the training is maintained.

In sum, these studies suggest that very brief imaginal exposure Training is effective at reducing symptom levels in a sample of undergraduate and community participants with trauma exposure and distress. More severe participants received the most benefit from the training, as did those who expressed more emotions related to anxiety throughout the training. A lack of consistent findings demonstrating improved performance with Affect Labeling supplemental training was apparent across both studies, however, Labeling in general had benefit over No Labeling, suggesting that linguistic processing improves inhibitory learning outcomes.

Appendices

Appendix I. Pilot Study Diagram.



Appendix II. Placement of psychophysiology electrodes.

Part A: SCL electrode placement.

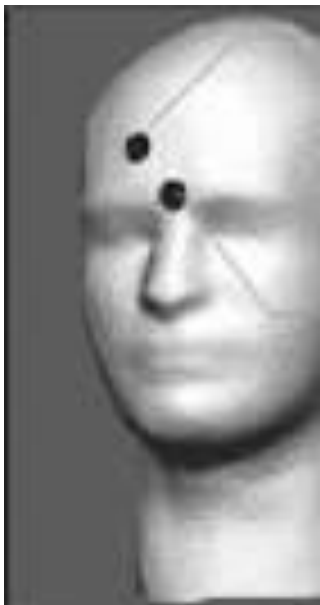


Note: This image was borrowed from Dawson, Schell, and Filion (2000).

Part B: Startle blink EMG Placement



Part C: F-EMG Placement



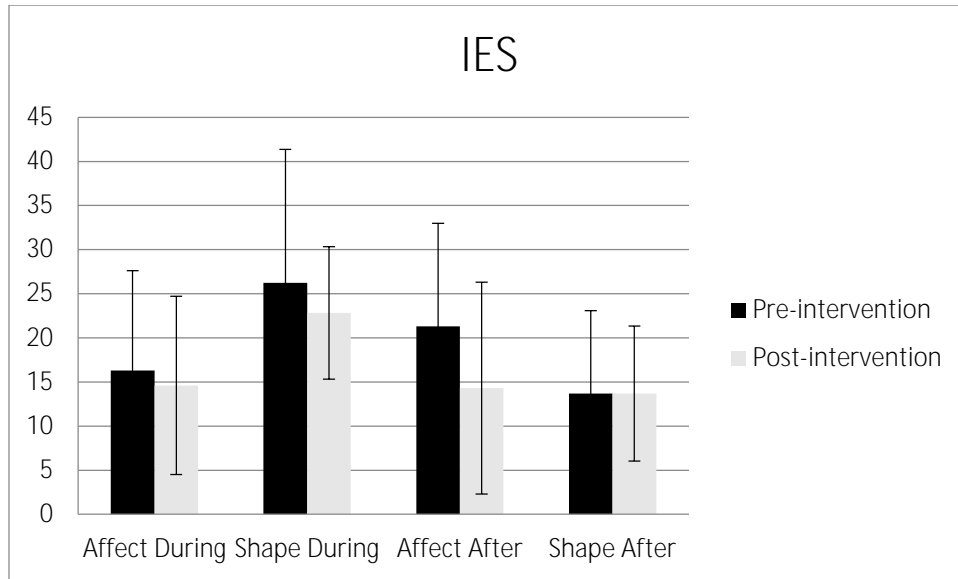
Note: This image was borrowed from Chin, Barreto, Cremades, & Adjouadi (2008).

Appendix III. Intraclass correlation for Pilot Study

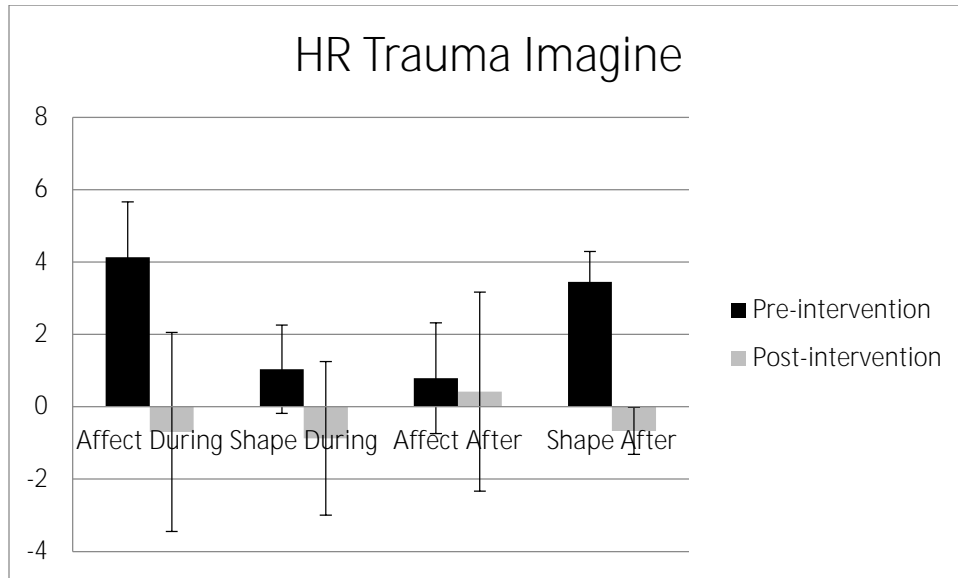
	ICC	SE
BDI	0.7	0.091
HR Trauma Script	0.167	0.182
SCL Trauma Script	0	0
SCL Trauma Imagine	0.047	0.199
SCL Neutral Script	0	0
SCL Neutral Imagine	0.065	0.199
EMG Neutral Integral	0.555	0.124
EMG Neutral Amplitude	0.695	0.094
Frontalis Trauma Script	0.377	0.177
Frontalis Trauma Imagine	0.234	0.21
Frontalis Neutral Script	0.422	0.174

Note: ICC indicates intraclass correlation, and SE indicates standard error of intraclass correlation. For the two variables with very low ICC estimates, these models were re-run using repeated measures ANOVs, though no differences were found.

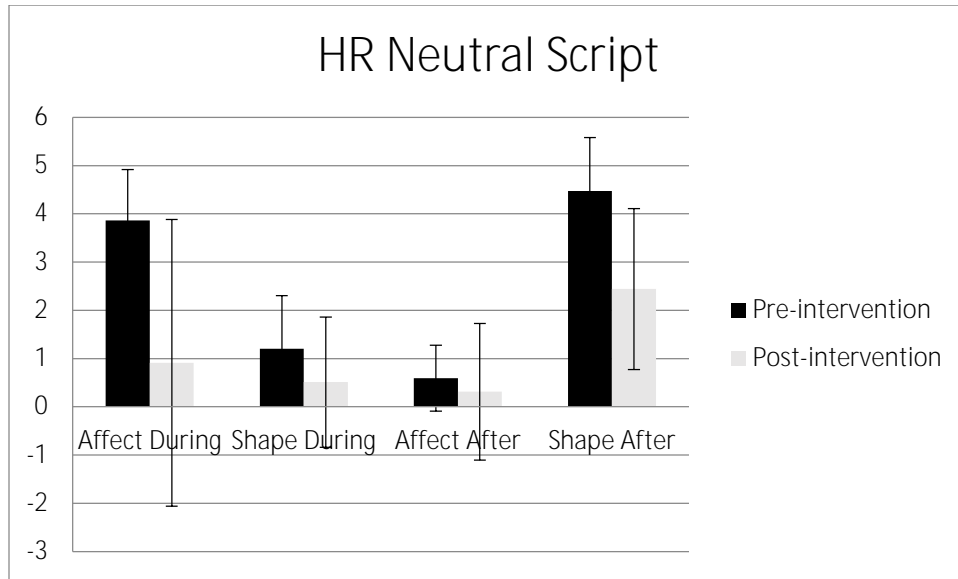
Appendix IV. Pilot Outcome for IES



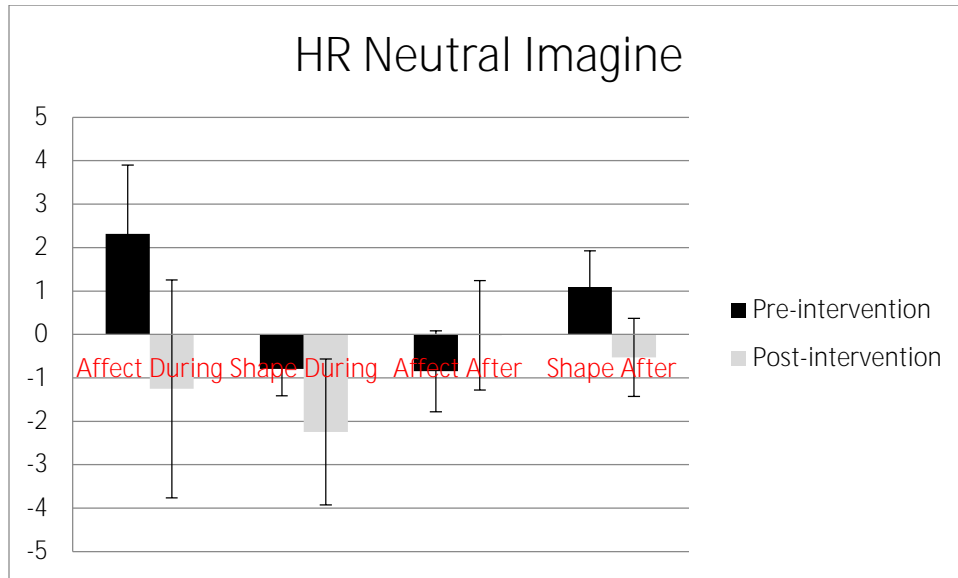
Appendix V. Pilot HR Trauma Imagine.



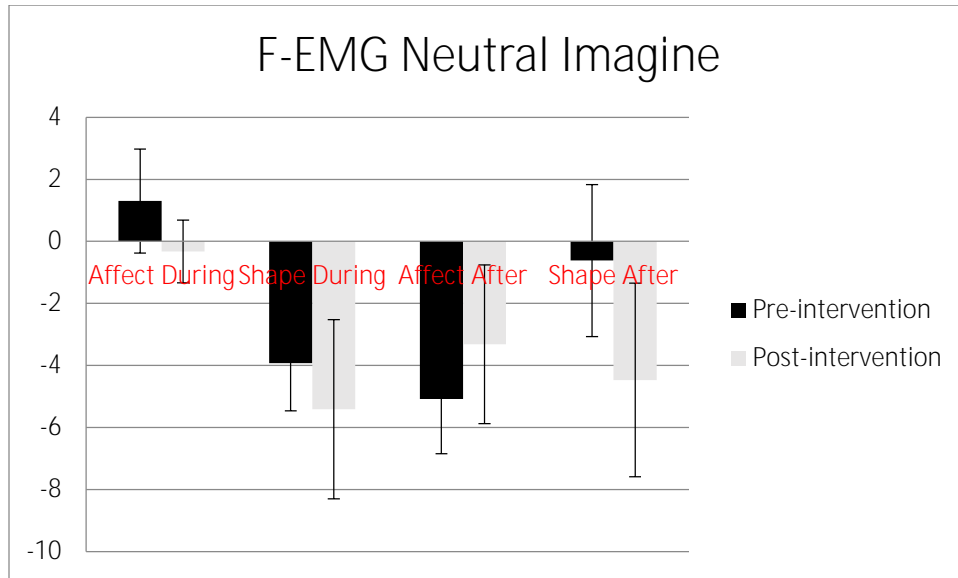
Appendix VI. Pilot Study HR Neutral Script



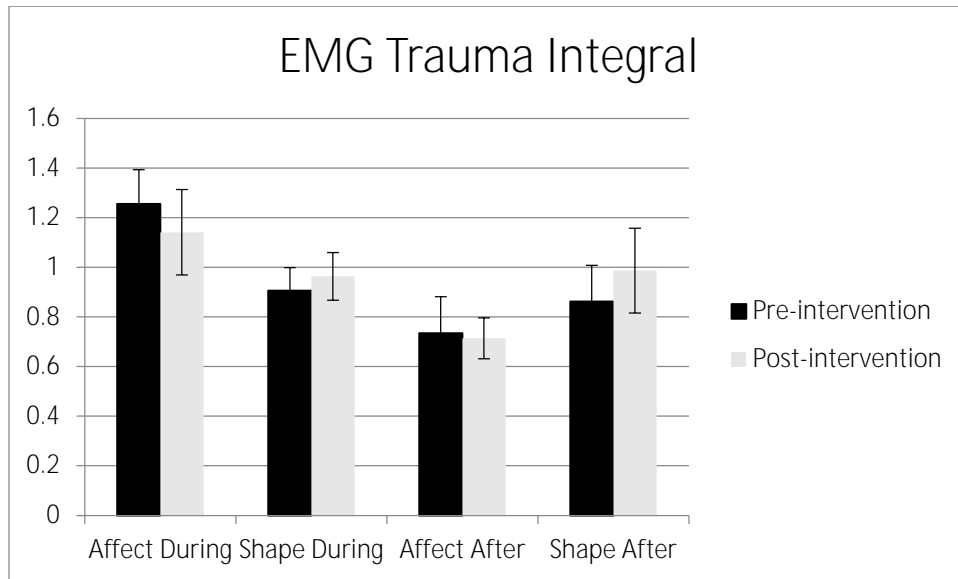
Appendix VII. Pilot HR Neutral Imagine.



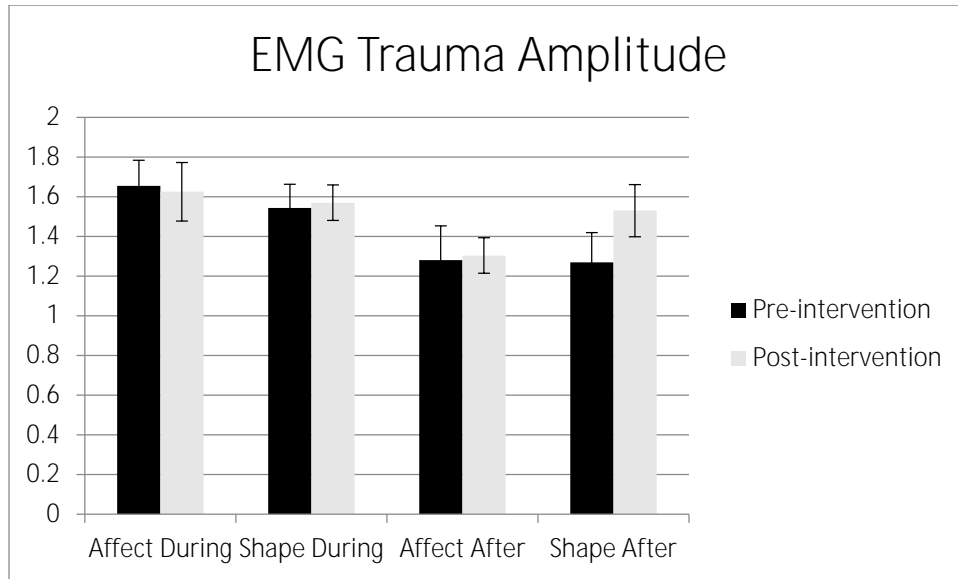
Appendix VIII. Pilot F-EMG Neutral Imagine.



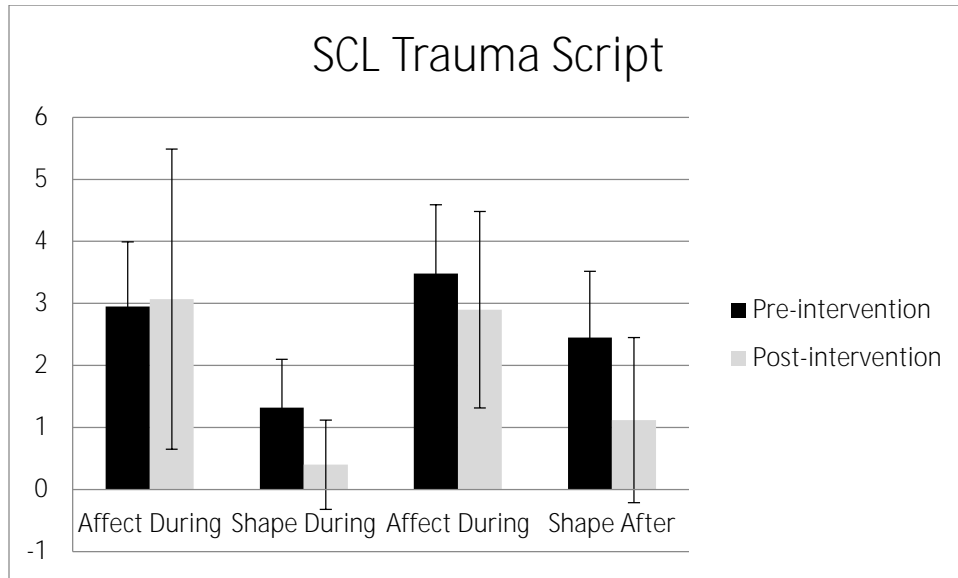
Appendix XVI. Pilot EMG Trauma Integral.



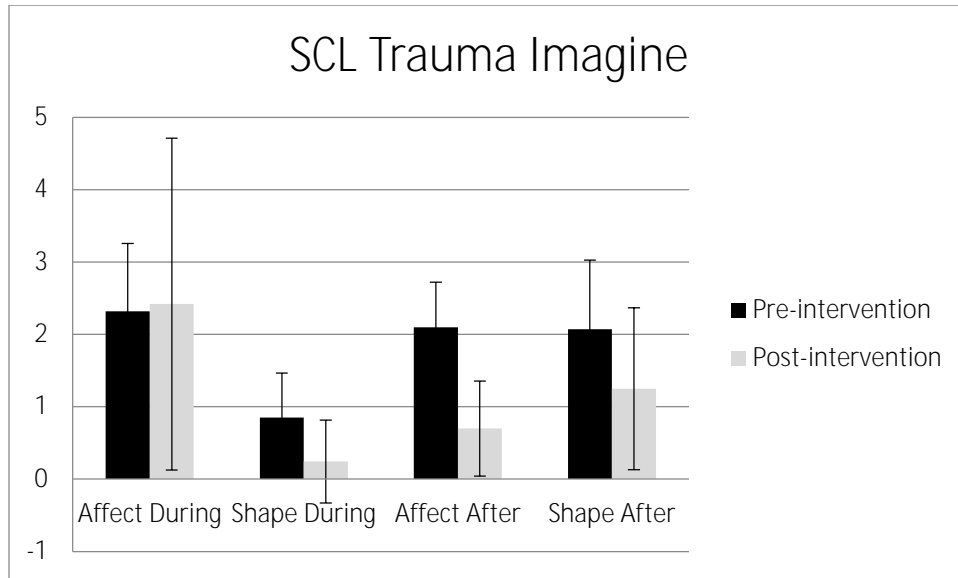
Appendix IX. Pilot F-EMG Trauma Amplitude.



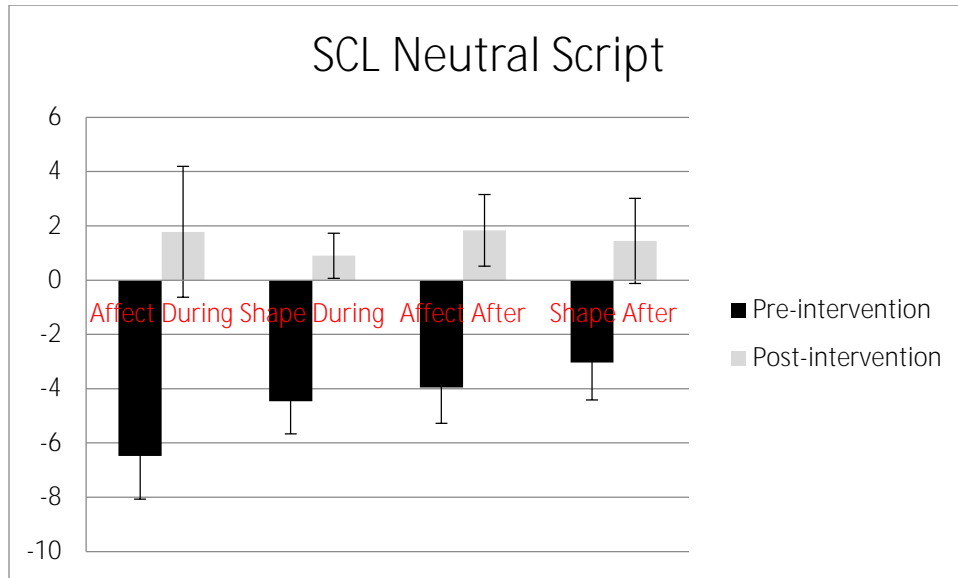
Appendix XI. Pilot SCL Trauma Script.



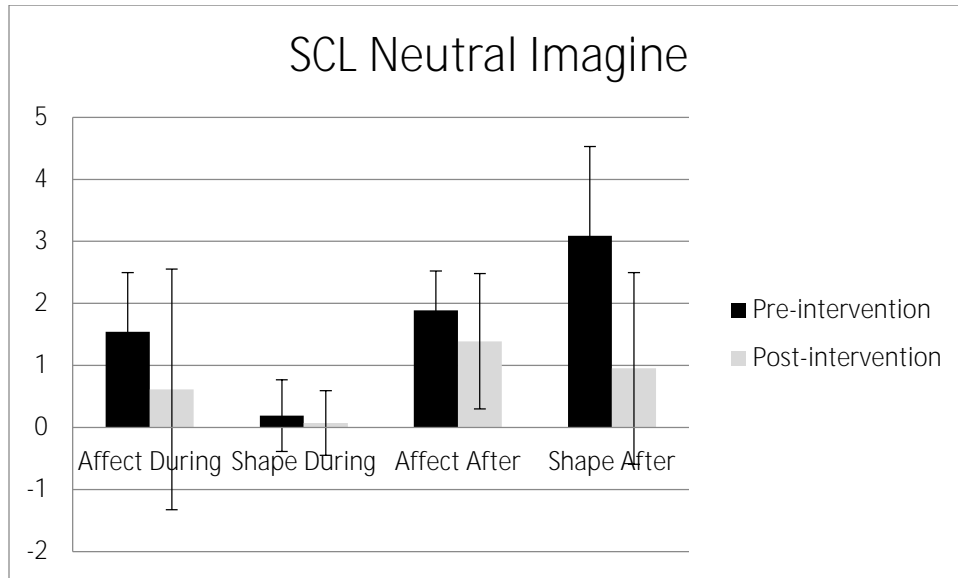
Appendix XII. Pilot SCL Trauma Imagine.



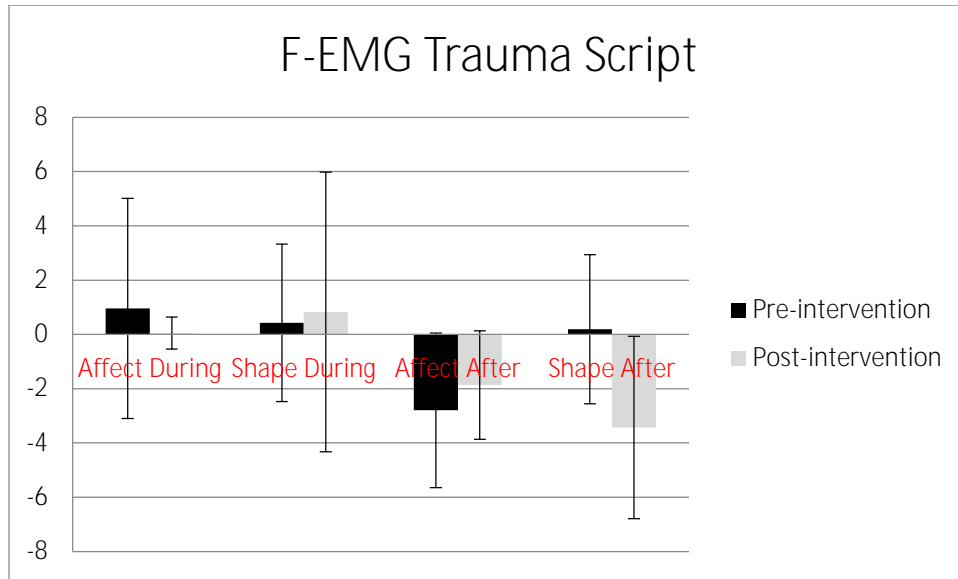
Appendix XIII: SCL Neutral Script.



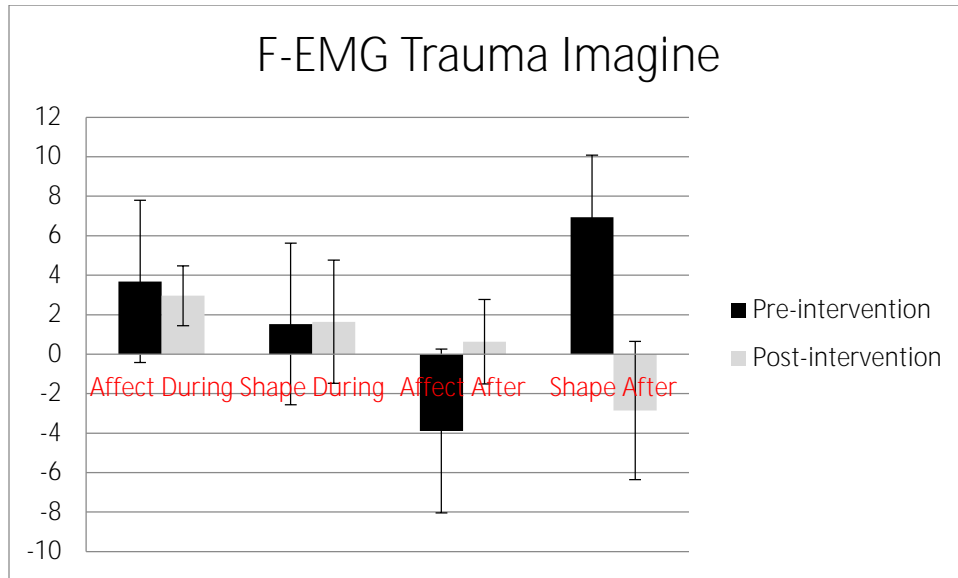
Appendix XIV. SCL Neutral Imagine.



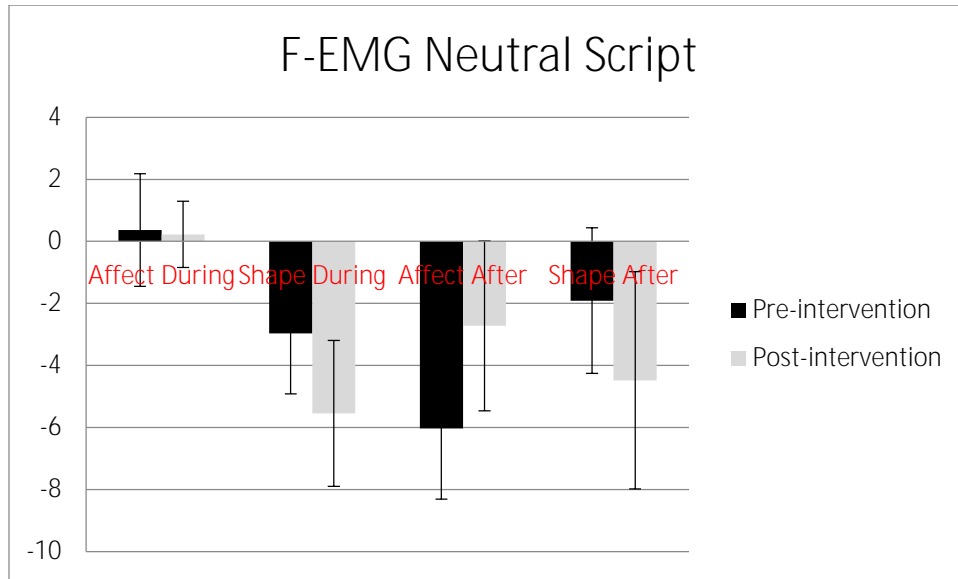
Appendix XV. Pilot F-EMG Trauma Script.



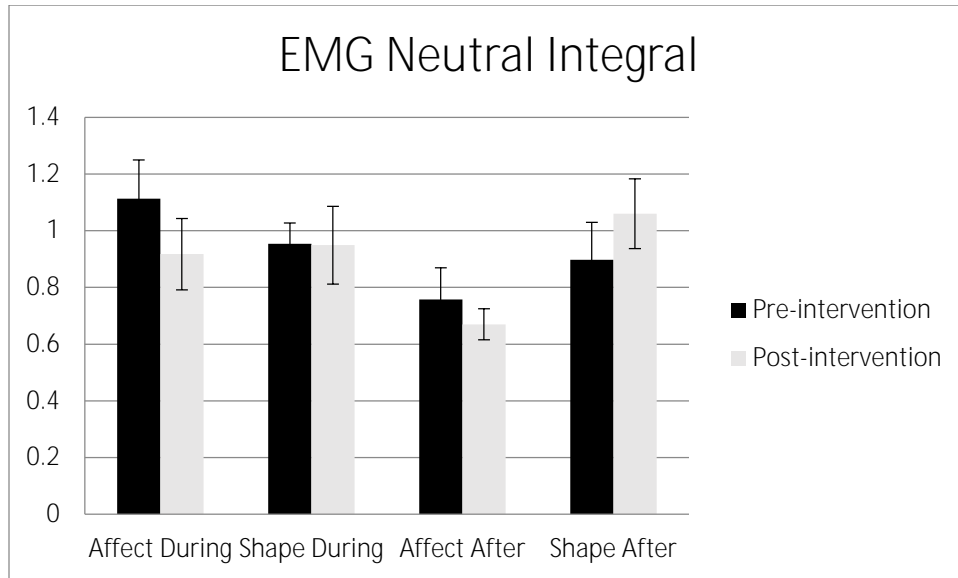
Appendix XVI. Pilot F-EMG Trauma Imagine.



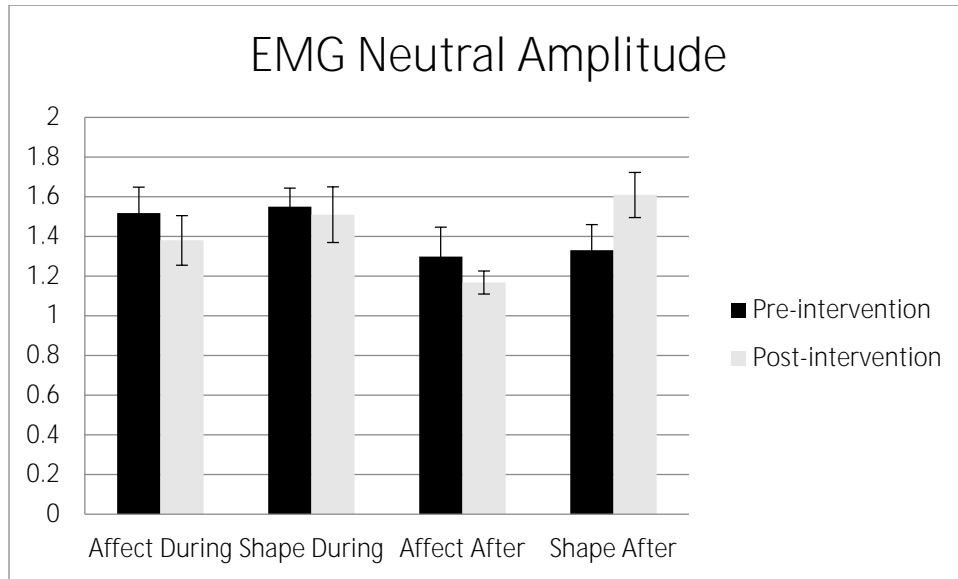
Appendix XVII. Pilot F-EMG Neutral Script.



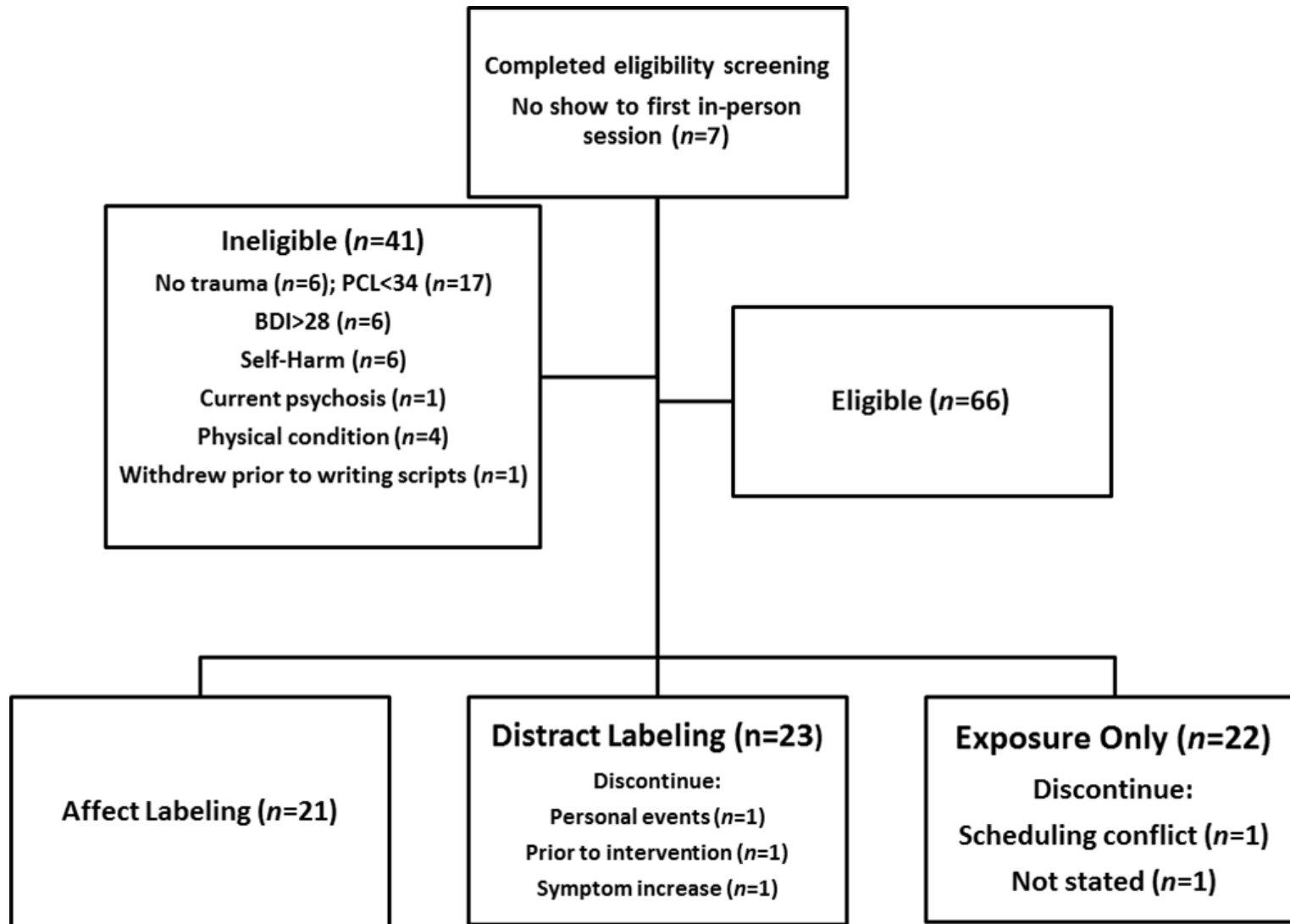
Appendix XVIII. Pilot EMG Neutral Integral.



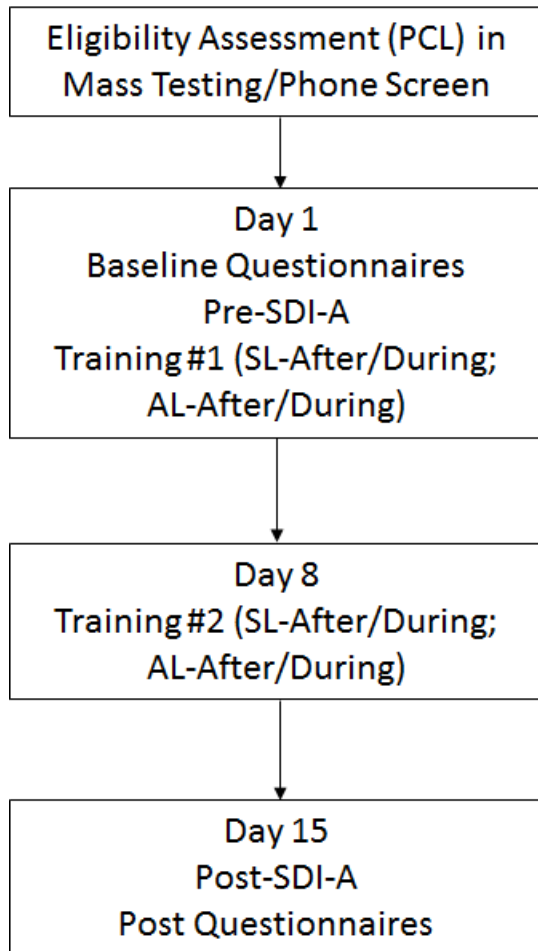
Appendix XIX. EMG Neutral Amplitude



Appendix XX. Consort diagram for main study.



Appendix XXI. Main study diagram.



Appendix XXII: Types of Trauma in Main Study

	AL (%)	DL (%)	EO (%)	Total (%)
Serious Accident	6.06	4.55	3.03	13.64
Assault by Someone Known	1.52	3.03	3.03	7.58
Assault by Stranger	1.52	0.00	0.00	1.52
Sexual Assault by Someone Known	7.58	3.03	6.06	16.67
Sexual Assault by Stranger	1.52	1.52	3.03	6.06
Life-threatening Illness	4.55	6.06	0.00	10.61
Natural Disaster	0.00	0.00	1.52	1.52
Other				
Witnessed/Learned about Someone Killed/Murdered	3.03	4.55	1.52	9.09
Witnessed Sexual Assault	3.03	1.52	0.00	4.55
Domestic Violence	1.52	1.52	6.06	9.09
Sudden Death of Someone Close	4.55	4.55	4.55	13.64
Physical Safety of Self/Others Threatened	1.52	3.03	1.52	6.06

Appendix XXIII: ICC for Main Study

	ICC	SE
PDS	0.595	0.082
IES	0.593	0.083
BDI	0.658	0.072
HR Trauma Script	0.214	0.129
HR Trauma Imagine	0.046	0.126
HR Neutral Script	0	0
HR Neutral Imagine	0.218	0.121
SCL Trauma Script	0.226	0.125
SCL Trauma Imagine	0.159	0.128
SCL Neutral Script	0.24	0.121
SCL Neutral Imagine	0.165	0.124
EMG Trauma Integral	0.425	0.105
EMG Trauma Amplitude	0.331	0.117
EMG Neutral Integral	0.349	0.114
EMG Neutral Amplitude	0.323	0.116
F-EMG Trauma Script	0.24	0.131
F-EMG Trauma Imagine	0.184	0.142
F-EMG Neutral Script	0	0
F-EMG Neutral Imagine	0.111	0.187

Note: For ICC that were very low (HR Neutral Script and F-EMG Neutral Script), these analyses were rerun using repeated measures ANOVA with no differences in outcome.

Appendix XXIV. ICC from Main Study: Process Analyses

Outcome Measure	ICC	SE
HR Trauma Script	0.229	0.036
HR Trauma Imagine	0.355	0.044
SCL Trauma Script	0.367	0.0144
SCL Trauma Imagine	0.419	0.045
F-EMG Trauma Script	0.274	0.039
F-EMG Trauma Imagine	0.389	0.045

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ⁱ Random effects of time were excluded for the analyses using PDS, IES, and HR as the standard error were not estimated for the random effect in these models. They were included for BDI, PDS-Func, SCL, EMG Amplitude, and Frontalis during the neutral script. All models except PDS-Func model would not converge with correlated random effects, and therefore the default independent random effect setting was used in these cases.