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HABITUATION TRAJECTORY DURING EXPOSURE THERAPY:
COMPARING TRAUMA FREQUENCY AND TRAUMA TYPE

by

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A dissertation submitted in partial fulfillment of the requirements
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in the Department of Psychology
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ABSTRACT

The current DSM-5 criteria for Posttraumatic Stress Disorder (PTSD) affords heterogeneous symptom presentations; however, current treatment fails to consider differences in trauma frequency and trauma type. These different symptom profiles that exist within the PTSD framework lead to questions about the generalizability of treatment outcomes from one group to another group. One group of interest is those that experience multiple traumatic events and report multiple index traumas (trauma frequency). The second group of interest is those with a PTSD diagnosis from occupational exposure to traumatic events (trauma type). Appreciation of the reinforcement schedule may be particularly crucial for understanding treatment response. The current study aims to investigate habituation responses during exposure therapy to inform treatment modifications and decisions. Exposure therapy process variables, session variables, and PTSD severity were examined for a sample of 128 participants that sought treatment for combat-related PTSD or occupation-related PTSD, some of whom endorsed multiple index traumas, and some of whom endorsed a single index trauma. Results revealed no significant differences based on trauma frequency or trauma type for within-session and between-session habituation. This suggests that the effectiveness of a flooding approach to extinguish avoidance behavior may overcome the impact of reinforcement schedule on fear habituation. The results of the present study contribute to the growing body of literature that suggests the exclusion of patients based on the pre-treatment characteristics of trauma frequency and trauma type is unfounded.

“We should learn... to do our best for the sake of our communities
and for the sake of those for whom we pave the way.”

-Justice Ruth Bader Ginsberg

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I extend my deepest gratitude to the past, present, and future service members and first responders that allow us to join their team.

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LIST OF ACRONYMS

BSH	Between-Session Habituation
CAPS	Clinician Administered PTSD Scale
CR	Conditioned Response
CS	Conditioned Stimulus
DSM	Diagnostic and Statistical Manual of Mental Disorders
LMER	Linear Mixed Effects Regression
OEF	Operation Enduring Freedom
OIF	Operation Iraqi Freedom
PCL-M	PTSD Checklist – Military Version
PCL-5	PTSD Checklist for DSM-5
PE	Prolonged Exposure
PTSD	Posttraumatic Stress Disorder
SUDS	Subjective Units of Distress Scale
TF	Trauma Frequency
TMT	Trauma Management Therapy
TT	Trauma Type
TWSH	Time to Within-Session Habituation
UCR	Unconditioned Response
UCS	Unconditioned Stimulus
WSH	Within-Session Habituation

INTRODUCTION

In recent years, interest in the psychological and physical effects of job-related stress has grown. Specifically, interest in the consequences of Posttraumatic Stress Disorder (PTSD) in at-risk professions such as firefighting and law enforcement has increased (Fullerton, McCarroll, Ursano, & Wright, 1992; McFarlane & Bryant, 2007). First responders endanger their lives to protect the community, and this profession is considered to be one of the most dangerous and stressful occupations in the United States (Del Ben, Scotti, Chen, & Fortson, 2006; U.S. Department of Labor, 2007). The job of a first responder is multifaceted and places a broad range of demands on each individual. Specifically, the demands include strenuous physical requirements (i.e., carrying, lifting, running, climbing), working under varying environmental conditions (i.e., extreme changes in temperature, fumes, chemicals, hazards), and cognitive demands (i.e., organization, adaptation, decision-making). These demands are often present while the first responder is performing under chaotic emergent situations such as structure fires, motor-vehicle accidents, or mass casualty incidents, all of which can necessitate the provision of basic and advanced life support and exposure to human remains (Marchand, Nadeau, Beaulieu-Prévost, Boyer, & Martin, 2015; Miller, 1995). Some first responders may consider these events traumatic.

Military traumatic events have been defined and categorized by N. R. Stein and colleagues (2012) defined and categorized military traumatic events as, “Life Threat to Self,” “Life Threat to Others,” “Aftermath of Violence,” “Traumatic Loss,” “Moral Injury by Self,” and “Moral Injury by Others.” It is likely that these same definitions and categories can be

applied to events experienced by first responders. In fact, they encounter these traumatic events repeatedly with high frequency as a function of their daily job requirements (Hartley, Sarkisian, Violanti, Andrew, & Burchfiel, 2013; Marmar et al., 2006). During a six-month period, police officers experienced approximately 3.5 traumatic events, and firefighters encountered an average of 6.74 traumatic event exposures per year (Corneil, Beaton, Murphy, Johnson, & Pike, 1999). In comparison, among a sample of 706 Marines, those deployed once reported experiencing an average exposure of 1.76 combat-related traumatic events and Marines deployed twice reported experiencing an average of 2.83 combat-related traumatic events (Phillips, LeardMann, Gumbs, & Smith, 2010). In other words, the number of traumatic event exposures per year for firefighters is 3.82 and 2.38 times greater than the exposure experienced by Marines deployed once and twice, respectively. In addition to the differing frequency of traumatic event exposure the location of traumatic event exposures differs. First responders experience traumatic events in their own communities, whereas, combat-related traumatic events are experienced outside of the United States (Lanza, Roysircar, & Rodgers, 2018)

The extant literature demonstrates that police officers and firefighters develop PTSD resulting from their occupational exposure to traumatic events. Furthermore, first responders diagnosed with PTSD are more likely to take more sick days, retire earlier (Berger et al., 2007; Marmar et al., 2006; North et al., 2002), experience burnout (Asen & Colon, 1995; Bernabé & Botia, 2016), and use alcohol at increased rates (Chae & Boyle, 2013; North et al., 2002; Oehme, Donnelly, & Martin, 2012; Paulus, Vujanovic, Schuhmann, Smith, & Tran, 2017). Prevalence estimates for PTSD in first responders vary from 6.2% to 15% among United States police officers (Hartley et al., 2013; Pietrzak et al., 2014) and from 6.3% to 22% among United States

career firefighters (Bernard, Driscoll, Kitt, West, & Tak, 2006; DeLorme, 2014). Most findings are in excess of the 6.8% rate observed among the general population (Kessler et al., 2005) and are similar to the prevalence rate seen in OEF/OIF veterans (12%, Hoge, Riviere, Wilk, Herrell, & Weathers, 2014).

Given these high prevalence rates and associated risks, providing empirically supported treatments is fast becoming a national call to action. For example, several states, including Colorado, Florida, Texas, and Vermont, have recently passed legislation to include PTSD as a compensable injury under workers compensation. The inclusion of PTSD as a compensable injury provides additional opportunity and access for individuals to seek treatment. Therefore, it is becoming increasingly imperative to understand efficacious treatments for PTSD. It is especially important to determine whether a treatment approach focused largely on military personnel will be equally effective for first responders.

Posttraumatic Stress Disorder: Etiology and Treatment

PTSD: Etiology.

Posttraumatic Stress Disorder (PTSD) represents a cluster of symptoms, lasting longer than a period of one month, resulting from exposure to a traumatic event (i.e., exposure to death, threatened death, actual or threatened serious injury, or actual or threatened sexual violation, occupation-related, media exposure) (American Psychiatric Association, 2013). This exposure may be direct or indirect, which can elicit symptomatic emotional responses that may be immediate or delayed. For instance, exposure to traumatic events can be indirect (e.g., witnessing a child drowning), and the onset of symptoms can arise several months after initial exposure (Bonanno, 2008; Bonanno et al., 2012). Regardless of the type of exposure,

individuals manifest constellations of symptoms that are classified into four clusters. These symptom clusters include: 1) “Intrusion” (e.g., flashbacks, nightmares, upsetting memories); 2) “Avoidance” (e.g., internal cognitive and external physical); 3) “Negative Alterations in Cognitions and Mood” (e.g., restricted affect); and 4) “Arousal and Reactivity” (e.g., hypervigilance, irritability, and difficulty sleeping).

The learning paradigm of fear conditioning can help explain the development of these PTSD symptoms. Learning theory suggests that PTSD is developed and maintained by the malfunction of fear-extinction mechanisms (Bisson, 2009; Pitman, 1989). This malfunction manifests in several brain regions (i.e., hippocampus, amygdala, HPA axis, prefrontal cortex) and behavioral responses related to learning and memory processes (Careaga, Girardi, & Suchecki, 2016; Rao, Suvrathan, Miller, McEwen, & Chattarji, 2009; Yehuda et al., 1995). The fear-conditioning paradigm explains that learning occurs when a response that naturally occurs (unconditioned response, UCR) in the presence of a stimulus (unconditioned stimulus, UCS) can be activated when a previously neutral stimulus (conditioned stimulus, CS) can also evoke that same response (conditioned response, CR) (Pavlov, 1927; Watson & Rayner, 1920). More specifically in the case of traumatic events, the traumatic event is a UCS for the UCR of fear and anxiety that occurred during the trauma. Numerous neutral stimuli that were present before and during the traumatic event are paired with the UCS. Examples of neutral stimuli include garbage receptacles, a house painted blue, holding car keys, wearing sunglasses, among others. For individuals that develop PTSD, following the traumatic event, these neutral stimuli trigger fear and anxiety even in the absence of the traumatic event. What was previously neutral (i.e., blue house and holding car keys) is now a CS. The CS produces a CR (i.e., heavy breathing, muscle

tension, increased heart rate) (VanElzakker, Dahlgren, Davis, Dubois, & Shin, 2014; Watson & Rayner, 1920). The CR is unpleasant; therefore, an individual often escapes from the situation or avoids the situation all together, and their response decreases. The removal of unpleasant symptoms often leads to an increase in the behavioral (reinforcement) of avoidance and emotional numbing (Bisson, 2009; Pitman, 1989). Avoidance is a response that prevents an aversive stimulus from occurring. Avoidance is a maladaptive coping strategy that ultimately serves to maintain the disorder. Efficacious treatment for PTSD targets these maintaining factors in an effort to correct the malfunctioning fear-extinction mechanisms by extinguishing (suppressing the emergence of) the CR (McNally, 2007; VanElzakker et al., 2014).

PTSD: Exposure therapy treatment.

To address PTSD symptoms, the Institute of Medicine (Institute of Medicine, 2007) and the VA/DOD (The Management of Post-Traumatic Stress Working Group, 2010) recommend exposure therapy as a first-line treatment. Learning theory can also help explain the efficacy of exposure therapy. Exposure therapy refers to the process of repeated confrontation to feared stimulus (Abramowitz, Deacon, & Whiteside, 2011). For the treatment of PTSD, the repeated confrontation is to the traumatic memory (UCS) (Foa & Kozak, 1986). This confrontation with reminders can take place by way of imaginal exposure and in-vivo exposure. For some imaginal exposure treatment protocols, the clinician and the patient work together to write a recounting of the traumatic event (imaginal scene; CS). This imaginal scene includes sensory experiences (i.e., smells and sounds of the traumatic event), cognitions (i.e., “I’m not safe,” “I’m never going to see my family again,” “I could be shot at any moment”), and physiological markers (i.e., “my heart was beating harder and harder”) that the patient encountered during the traumatic event.

The presentation of the CS (imaginal scene) in the absence of the UCS (traumatic event) elicits the physiological and cognitive responses (CR). During subsequent sessions the trauma narrative is recounted, and the patient is asked to immerse him or herself in the event (presentation of CS without US). The aim of the repeated presentations of the CS is to achieve a decline in frequency and magnitude of physiological and cognitive responses (CR) known as habituation (Harris, 1943; Humphreys, 1933; McNally, 2007).

Likewise, in-vivo exposure can begin with the clinician and patient working together to identify traumatic memory cues in the environment. Concurrently, a fear hierarchy of people, places, and things (in the real world) that remind the patient of the traumatic event is created. The patient is instructed to engage in activities (CS) related to the items on the fear hierarchy. For example, a patient may be asked to sit in a crowded area with their back to the exit. Similarly, the aim of the exercise is to achieve habituation and extinction of the physiological and cognitive responses. Over repeated trials, habituation occurs in-session and between sessions. This habituation and subsequent extinction are the result of the presentation of traumatic event related cues (CS) without traumatic event related outcomes (CR). Achievement of habituation and extinction indicate that the exposure therapy process successfully inhibited the underlying malfunctioning fear cues (Abramowitz et al., 2011; Foa, Steketee, & Rothbaum, 1989; McNally, 2007). Within-session habituation (WSH) and between-session habituation (BSH) are commonly tracked by the clinician to measure fear activation and habituation as markers of extinction learning (Beidel, Frueh, Neer, Bowers, et al., 2017; Beidel, Frueh, Neer, & Lejuez, 2017; Foa & Kozak, 1986; Jaycox, Foa, & Morral, 1998; Rauch & Foa, 2006; Tolin & Foa, 1999; van Minnen & Hagedaars, 2002; Wolpe, 1973). During the session, clinicians will

ask patients to provide a subjective verbal rating, using the SUDs (Subjective Units of Distress) scale, to communicate anxiety and discomfort (Wolpe & Lazarus, 1966). While there is considerable debate about the importance of WSH and BSH (Bluett, Zoellner, & Feeny, 2014; Craske et al., 2008; van Minnen & Hagedaars, 2002), both indices continue to be important indicators used by clinicians during exposure therapy sessions. Exposure therapy has demonstrated efficacy for reducing and eliminating symptoms of PTSD for a variety of populations such as adolescents, assault and rape survivors, and combat veterans (Goodson et al., 2011; Haagen, Smid, Knipscheer, & Kleber, 2015; Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010), and has produced large effect sizes for both PTSD symptom severity and secondary symptoms (i.e., depression, quality of life) (Powers et al., 2010).

Multiple Traumatic Events: The Potential Impact on Exposure Therapy

A growing body of literature is concerned with the impact of multiple traumatic events on outcomes of PTSD treatment (Diamond, Lipsitz, Fajerman, & Rozenblat, 2010; Eagle & Kaminer, 2013; Lang & McTeague, 2011; Levy-Gigi, Richter-Levin, Okon-Singer, Kéri, & Bonanno, 2016; McTeague et al., 2010; J. Y. Stein, Wilmot, & Solomon, 2016). Individuals with multiple traumatic events are at greater risk for developing PTSD and increased symptom severity (Green et al., 2000; Ogle, Rubin, & Siegler, 2014; R. J. Turner & Lloyd, 1995), for impaired social and occupational functioning (Keinan, Shrira, & Shmotkin, 2012; Lopes Cardozo, Vergara, Agani, & Gotway, 2000), and for co-morbid diagnoses (Lu, Mueser, Rosenberg, & Jankowski, 2008; McTeague et al., 2010; Suliman et al., 2009; Yalch, Hebenstreit, & Maguen, 2018). Several researchers argue that individuals who endure multiple traumatic events differ from those exposed to single-discrete traumatic events (Diamond et al., 2010; Eagle

& Kaminer, 2013; Kaminer, Eagle, & Crawford-Browne, 2016; Nuttman-Shwartz & Shoval-Zuckerman, 2016). These differences may be attributed to blunted neurophysiological reactivity among individuals exposed to multiple traumatic events (Cuthbert et al., 2003; D'Andrea, Pole, DePierro, Freed, & Wallace, 2013; Hageraars, Stins, & Roelofs, 2012; Lang & McTeague, 2011; McTeague et al., 2010). This lower reactivity to aversive stimuli may be a reflexive reaction to help individuals with multiple trauma exposures to cope with persistent and recurrent stress. Although this hypo-reactivity may provide a physiological safeguard, McTeague and et al. (2010) argue that this blunted reactivity impairs emotional processing of the traumatic event, which reduces the ability to correct the malfunctioning fear-extinction mechanisms. This would imply that individuals with exposures to multiple traumatic events may experience an attenuated response to exposure therapy; however, the extant literature demonstrates that this is not the case. Numerous studies have demonstrated the effectiveness of prolonged exposure therapy (PE) (Foa, Hembree, & Rothbaum, 2007) with individuals exposed to multiple traumatic events (Foa et al., 2005; Hendriks, de Kleine, Broekman, Hendriks, & van Minnen, 2018; Jerud et al., 2017; Schnurr et al., 2007).

Yet, these studies about the effectiveness of PE have focused on general outcomes (e.g., diagnosis remittance), which do not communicate important process variables that reflect change in patient response to trauma (e.g., session length and number of sessions). Further, general outcomes may not be the best way to support or refute the theory that multiple exposures to traumatic events impact treatment efficacy and response due to blunted emotional reactivity. Rather, examination of habituation trajectory between and within sessions may provide better insight into the relationship between multi-trauma exposure and emotional reactivity on

treatment response and outcomes. Several studies have examined treatment response as measured by symptom change across sessions; however, these studies have failed to consider within and between session habituation (Allan, Gros, Myers, Korte, & Acierno, 2017; Clapp, Kemp, Cox, & Tuerk, 2016; Dickstein, Walter, Schumm, & Chard, 2013; Hendriks et al., 2018; Schumm, Walter, & Chard, 2013; N. R. Stein, Dickstein, Schuster, Litz, & Resick, 2012). Only one study to date has aimed to examine McTeague et al.'s (2010) theory that multiple exposure predicts worse distress reduction for exposure-based treatments (Jerud et al., 2017).

Jerud et al. (2017) provided prolonged exposure therapy (PE) (Foa et al., 2007) over the course of 10 weeks to individuals that primarily (78%) experienced interpersonal trauma (i.e., sexual assault, non-sexual assault, childhood sexual abuse). The average time since the index trauma was 12.02 years (SD = 12.25) and the average number of traumatic events was 3.72 (SD = 2.31). Using linear mixed modeling, the researchers found that increased prior trauma exposure did not predict worse distress reduction or PTSD outcomes compared to individuals that experienced a single traumatic event at post-treatment, 3- and 6-month follow-up. Additionally, higher amounts of trauma were not associated with different mean or Peak SUDs across sessions. However, limitations in their study reduce the ability to draw definitive conclusions about their findings.

One such limitation that the findings of within-session habituation are limited by the treatment protocol. The treatment protocol prescribes that during the first active treatment session (session 3), 45- to 60-minutes (of the 90- to 120-minute total session length) is spent engaged in imaginal exposure. Subsequent imaginal exposure sessions (sessions 4-9) engage in imaginal exposure for 30- to 45-minutes (of the 90- to 120-minute total session length). This

prescribed and limited session length does not provide sufficient opportunity to observe the natural habituation period that might have occurred without such time limiting factors (in other words, there is not sufficient time to allow within-session habituation for all individuals).

Second, the findings of between-session habituation may be obscured by the treatment protocol. The traditional PE protocol dictates that sessions be held once-per-week, and between sessions, patients complete homework assignments of listening to an audio recording of the imaginal exposure and engage in in-vivo exposures. As a result, the SUDS ratings collected in-session may not be reflective of the natural progression between sessions but instead augmented by the treatment benefits of homework assignments.

Third, Jerud et al.'s (2017) definition of multiple trauma is likely problematic. They employed a rank order coding scheme for multiple traumas. Participants with one Criterion A event were coded as "0," and individuals with the one Criterion A event plus one additional trauma were assigned the value "1." Individuals with one Criterion A event plus two additional traumas were assigned the value "2," which continued in ascending order of trauma frequency. Therefore, the comparison between individuals that experienced a single trauma and multiple traumas was based on the presence of more than one traumatic event, which fails to capture a more homogenous group of people that experienced multiple traumas.

A fourth limitation of the study is that the type of trauma experienced by individuals in the sample was predominantly of the interpersonal type. Consequently, these findings may not generalize to other types of traumatic events because prevalence, symptom manifestation, and psychopathology outcomes vary by trauma type (J. Y. Stein et al., 2016). As a result of these

limitations, additional information and exploration of the relationship between multiple traumatic events, exposure therapy treatment outcomes, and habituation response are necessary.

Multiple Traumatic Events: Effect on Treatment Outcomes for First Responders

Questions about treatment efficacy are valid considering not all patients that receive exposure therapy experience remittance of PTSD symptoms (Bradley, Greene, Russ, Dutra, & Westen, 2005; Hendriks et al., 2018; Watts et al., 2013). In a study examining treatment outcomes for individuals exposed to multiple traumatic events, 29% did not respond to treatment and continued to report elevated PTSD symptoms (Hendriks et al., 2018). The authors raise the question that extinction learning may not be achievable for some individuals exposed to multiple traumatic events, as evidenced by 29% of their sample not responding to treatment. Therefore, it is necessary to look at studies that examine exposure therapy treatment outcomes for first responders.

Literature that reports on PTSD treatment outcomes for first responders is limited, and published results about exposure therapy treatment efficacy is even more limited (Haugen, Evces, & Weiss, 2012; Papazoglou, 2017). A literature review about PTSD treatment outcome revealed that only 17 of the 845 potentially relevant articles focused on first responders and only two of the 17 were RCTs (Haugen et al., 2012). The authors observed great heterogeneity of frequency, intensity, duration, and intervention type (i.e., EMDR, CBT, Psychodynamic). Each of the reviewed studies demonstrated treatment gains; however, the studies varied in reporting of the gains (i.e., symptom reduction, remission) and measures used to quantify these gains. As evidence of the limited availability of articles about exposure therapy, only four of the 17 studies used exposure therapy. Further, these four studies are limited in the ability to draw conclusions

because of study design. In light of the limitations, these four studies that used exposure therapy are worthwhile to consider given the limited availability of peer-reviewed articles.

Three of the four are case studies with police officers (Cornelius & Kenyon-jump, 2007; Richards & Rose, 1991; Tolin & Foa, 1999) with Richards and Rose (1991) reporting on two cases, and the fourth study provides a comparative study between virtual reality (VR) assisted exposure therapy and waitlist control. Each of the case studies provided 60 minutes of weekly exposure therapy; however, each followed a different protocol. Cornelius and Kenyon-Jump (2007) provided exposure in a gradual format. Both Tolin and Foa (1999) and Cornelius and Kenyon-Jump (2007) included relaxation training, whereas Richards and Rose (1991) did not include relaxation. The number of sessions differed across studies ranging from four (Richards & Rose, 1991, Case Four) to 15 sessions (Cornelius & Kenyon-jump, 2007). In light of these differences, each case observed positive effects of treatment, which were maintained at 2-month (Cornelius & Kenyon-jump, 2007) and 6-month follow-up (Richards & Rose, 1991; Tolin & Foa, 1999). Furthermore, officers that were relieved of duty before treatment were able to return to full duty after treatment (Richards & Rose, 1991; Tolin & Foa, 1999).

These treatment effects are promising and demonstrate that for police officers, exposure therapy may be a viable treatment option. The fourth study utilized a sample of September 11th World Trade Center (WTC) survivors. The sample included five firefighters that received VR assisted exposure therapy; however, results on the five firefighters are not available because the results included four non-rescue disaster relief workers and four civilians, and of the 13 participants, only 10 completed the treatment protocol (Difede et al., 2014). Participants completed between six and 13 sessions of active VR assisted exposure. At the beginning of

treatment, participants received two sessions of psychoeducation and relaxation training, and at the end of treatment participants engaged in two sessions that focused on reviewing treatment progress and relapse prevention strategies. For the 10 participants that completed the VR protocol clinically significant improvements in CAPS severity scores, which equated to a 39% decrease in CAPS scores, were observed. At the end of treatment, seven of the 10 participants no longer met diagnostic criteria for PTSD, and treatment gains were maintained at 6-month follow-up for those participants available (n = 9).

Despite the limitations of each study, together, the results are promising and indicate first responders are likely able to engage emotionally during the presentation of a stimulus, which some have theorized that first responders would not be able to achieve (Kaminer et al., 2016). Regardless of these promising outcomes, Hagen, Evces, and Weiss (2012) concluded at the end of their review that treatment for PTSD with first responders is inconclusive and more research is needed. Since the 2012 review, publications of first responder treatment articles are scarce. Some articles published since 2012 include one study that used MDMA assisted psychotherapy (Mithoefer et al., 2018), d-Cycloserine assisted exposure therapy (Difede et al., 2014), narrative exposure therapy (Alghamdi, Hunt, & Thomas, 2015), brief eclectic psychotherapy (Plat et al., 2013), and two exposure therapies (Golden, Jones, & Donlon, 2014; Gramlich & Neer, 2018). Overall, treatment for first responders was promising. Firefighters that participated in narrative exposure therapy experienced significant PTSD symptom reduction, but information about PTSD remission was not available (Alghamdi et al., 2015). After brief eclectic psychotherapy 81% of police officers returned to full duty status; however, remission rates were not reported (Plat et al., 2013). Among studies that reported remission rates, the rates were relatively low

compared to those observed in the general population; however, the results specific to first responders are limited because the samples included civilians (28%, Difede et al., 2014) and veterans (57%, Mithoefer et al., 2018).

The case studies, which used exposure-based treatments, demonstrated remission of PTSD diagnosis. The articles published since 2012 do not add substantial amounts of information to help draw conclusions. Further, the lack of literature on exposure-based therapies demonstrates the need for more information. As a result, this warrants the dedication of resources toward more research on treatment outcomes of exposure therapy for PTSD. Additionally, considering the full range of results, attention should be directed toward treatment progress, not just treatment outcomes.

Multiple Traumatic Events: Effect on Treatment Progress for First Responders

To date, the impact that multiple trauma exposures have on treatment outcomes is not well studied. Less is known about those exposed to multiple traumas as a function of their occupation (Bradley et al., 2005; Green et al., 2000; Hendriks et al., 2018; Price, Gros, Strachan, Ruggiero, & Acierno, 2013). Thus, it remains unclear whether more exposure to traumatic events attenuates the habituation response during exposure therapy for PTSD. To better understand the relationship between multiple PTEs, habituation response, and exposure therapy outcomes, it may be beneficial to examine the literature linking fear activation and habituation as markers of extinction learning.

Typically, from a learning theory and conditioning perspective, acquisition of behavior or response is dependent upon reinforcement. The term reinforcement refers to any event that strengthens the likelihood of a behavioral response (Ferster & Skinner, 1957; Grant, Hake, &

Hornseth, 1951; Humphreys, 1939, 1943; Longenecker, Krauskopf, & Bitterman, 1952).

Understanding reinforcement is likely beneficial for understanding exposure treatment effects for anxiety disorders because reinforcement typically impacts extinction (Bouton, 2004; Grant et al., 1951; Hothersall, 1966; Humphreys, 1939, 1943; Longenecker et al., 1952).

The two most commonly discussed schedules of reinforcement are continuous and intermittent reinforcement (Ferster & Skinner, 1957; Humphreys, 1943; Jenkins & Stanley, 1950; Skinner, 1938; Thorndike, 1933). Continuous reinforcement is provided with every response (e.g., 100 percent) to maintain the CR, whereas intermittent reinforcement is provided periodically based upon specified parameters of frequency and pattern (Hothersall, 1966; Jenkins & Stanley, 1950). The third type of reinforcement is partial reinforcement. Unlike intermittent reinforcement, partial reinforcement represents a random pattern (e.g., no parameters of frequency and pattern). Jenkins and Stanley (1950) defined partial reinforcement as "...given at least once but omitted on one or more of the trials or after one or more of the responses in a series" (p. 194).

Appreciation of the reinforcement schedule may be particularly crucial for understanding habituation responses, within- and between-sessions, during exposure therapy for individuals exposed to multiple traumas as a function of their occupation, such as first responders. First responders are exposed to numerous traumatic events throughout their career; however, these exposures, while predictable and likely to happen, are unpredictable in their time and number. Among first responders that develop PTSD, the presence of the CS (i.e., blue house, green garbage receptacle) on their next emergency response can potentially be linked to an entirely new UCS or a very similar UCS, which also triggers the CR. The unpleasant nature of the CR

activates the desire to engage in avoidance strategies (Bisson, 2009; Pitman, 1989). Examples of avoidance strategies can include and are not limited to assuming less prominent roles and responsibilities during a call, utilizing sick and vacation leave, driving alternative routes, using substances while off-shift, among others. Given the nature of the first responder occupation the ability to engage in avoidance strategies and the success of these strategies is random. The ability to engage in any of the strategies is contingent upon several factors and may not always be possible. This suggests that avoidance is on a partial reinforcement schedule. Extant literature demonstrates that resistance to extinction is stronger (e.g., takes more time and trials) when acquisition of the CR was performed under partial reinforcement compared to continuous or intermittent reinforcement (Bouton, 2004; Grant et al., 1951; Hothersall, 1966; Humphreys, 1939, 1943; Longenecker et al., 1952).

If the paradigm is applicable, then habituation between- and within-session during exposure therapy may require more time. Therefore, examination of the feasibility, impact, and habituation response in a treatment-seeking sample of individuals diagnosed with PTSD is warranted. To understand this relationship and provide insight for treatment the following hypotheses were proposed:

1. Participants with multiple index traumas will demonstrate slower between-session extinction rate of change across sessions compared to participants with a single index trauma.
2. Participants with multiple index traumas will demonstrate slower within-session extinction rate of change across sessions compared to participants with a single index trauma.

3. Participants with occupation-related PTSD will demonstrate slower between-session extinction rate of change across sessions compared to participants with combat-related PTSD.
4. Participants with occupation-related PTSD will demonstrate slower within-session extinction rate of change across sessions compared to participants with combat-related PTSD.

The following hypotheses about Exposure Therapy Process Variables were proposed:

5. Participants with multiple index traumas will have higher Peak SUDS ratings during the first and last exposure sessions than participants with a single index trauma.
6. Participants with multiple index traumas will have higher Lowest SUDS ratings during the first and last exposure sessions than participants with a single index trauma.
7. Participants with multiple index traumas will take more time to achieve WSH during the first and last exposure sessions than participants with a single index trauma.
8. Participants with multiple index traumas and participants with a single index trauma will significantly differ for change in time to reach within session habituation between the first and last sessions of exposure therapy
9. Participants with occupation-related PTSD will have higher Peak SUDS ratings during the first and last exposure sessions than participants with combat-related PTSD.
10. Participants with occupation-related PTSD will have higher Lowest SUDS ratings during the first and last exposure sessions than participants with combat-related PTSD.

11. Participants with occupation-related PTSD will take more time to achieve WSH during the first and last exposure sessions than participants with combat-related PTSD.
12. Participants with combat-related PTSD and occupation-related PTSD will significantly differ for change in time to reach within session habituation between the first and last sessions of exposure therapy.

The following hypotheses about Exposure Therapy Session Variables were proposed:

13. Participants with multiple index traumas will engage in significantly longer session times (first session, last session, and total session minutes) compared to participants with a single index trauma, as individuals experiencing multiple traumas will likely require more time to habituate within a treatment session.
14. Participants with multiple index traumas and single index trauma will significantly differ for change in first and last session length of exposure therapy.
15. Participants with multiple index traumas will require more exposure sessions compared to participants with a single index trauma, as individuals experiencing multiple traumas will likely require a greater number of sessions to achieve overall extinction.
16. Participants with occupation-related PTSD will engage in significantly longer session times (first session, last session, and total session minutes) compared to participants with combat-related PTSD, as individuals with occupation-related PTSD will likely require more time to habituate within a treatment session.
17. Participants with combat-related PTSD and occupation-related PTSD will significantly differ for change in first and last session length of exposure therapy.

18. Participants with occupation-related PTSD will require more exposure sessions compared to participants with combat-related PTSD, as individuals with occupation-related PTSD will likely require a greater number of sessions to achieve overall extinction.

The following hypotheses about PTSD Symptom Severity Variables were proposed:

19. Participants with multiple index traumas and combat-related PTSD will enter treatment and be discharged from treatment with greater PTSD symptom severity (as measured by self-report using the PCL-M) than those with a single traumatic event and combat-related PTSD.

20. Participants with multiple index traumas and combat-related PTSD and a single index trauma and combat-related PTSD will significantly differ for change in PTSD symptom severity (as measured by self-report using the PCL-M) over treatment.

21. Participants with multiple index traumas and occupation-related PTSD will enter treatment and be discharged from treatment with greater PTSD symptom severity (as measured by self-report using the PCL-5) than those with a single traumatic event and occupation-related PTSD.

22. Participants with multiple index traumas and occupation-related PTSD and a single index trauma and occupation-related PTSD will significantly differ for change in PTSD symptom severity (as measured by self-report using the PCL-5) over treatment.

METHOD

Data were extracted from two sources to create a secondary dataset. The first source was extracted from a completed randomized controlled trial for combat-related PTSD. The second source was extracted from a dataset of first responders who presented to a community clinic seeking treatment for occupation-related PTSD.

Participants

The sample consisted of treatment-seeking individuals with combat-related PTSD ($n = 102$) and occupation-related PTSD ($n = 26$). The 128 participants with a mean age of 38.1 years old ($SD = 9.45$) were mostly White (70.3%), identified as male gender (93.8%), completed some college (63.8%), and married (52.0%) at the time of treatment. Table 1 presents work-related (i.e., service branch) and personal demographic characteristics. Work-related and personal demographic characteristics were compared based on trauma frequency (TF; multiple index traumas vs. single index trauma) and trauma type (TT; combat-related PTSD vs. occupation-related PTSD) (Table 2). Aggregated by TF, significant differences for the personal demographic characteristic of race/ethnicity was observed ($p = .039$). No other personal and no work-related demographic characteristics were significant for TF. Aggregated by TT, participants with occupation-related PTSD ($M = 41.8$, $SD = 9.94$) were older than participants with combat-related PTSD ($M = 37.1$, $SD = 9.13$) ($t(126) = 2.29$, $p = .024$, $d = 0.50$). No other personal demographic characteristics were significant. Additionally, a significant difference for the work-related demographic characteristic of employment status was observed ($p < .001$) for TT.

Participants with combat-related PTSD consisted of veterans and active duty personnel who served in Operation Enduring Freedom (OEF) and Operation Iraqi Freedom (OIF). Participants with occupation-related PTSD consisted of currently employed (i.e., active duty) and retired (i.e., veteran) first responders. All participants completed a three-week exposure-based treatment protocol called Trauma Management Therapy (TMT) (Beidel, Frueh, Neer, Bowers, & Rizzo, 2014). TMT included daily exposure sessions and group therapy; however, the group components differed slightly between participants with combat-related PTSD and occupation-related PTSD (see treatment protocol below for descriptions). Individuals were included in the treatment protocol if they met a current clinician-determined diagnosis of combat-related PTSD. Individuals were excluded from the treatment protocol if they had significant cardiac event history, non-stabilized medication history, acute substance use disorder, or if they met criteria for antisocial personality disorder. Although screened for history of Traumatic Brain Injury (TBI), participants were not excluded from treatment. Previous literature, with this sample, indicated that history of TBI did not impact the exposure process (Ragsdale, 2014; Ragsdale et al., 2018). See Beidel et al. (2017) for CONSORT 2010 Flow Diagram.

All participants met the Diagnostic and Statistical Manual of Mental Disorders criteria for PTSD as assessed by a clinician administered semi-structured interview. Specifically, participants enrolled in the randomized controlled trial for combat-related PTSD met DSM-IV-TR criteria for PTSD and participated in the Clinician Administered PTSD Scale (CAPS; Blake et al., 1995). Participants that presented for occupation-related PTSD met DSM-5 criteria for PTSD and participated in the Clinician Administered PTSD Scale for DSM-5 (Weathers et al., 2017).

Treatment Protocol.

Combat-related PTSD treatment protocol.

A detailed description of the treatment protocol can be found in the publication by Beidel, Frueh, Neer, and Lejuez (2017). After completing a telephone screener that assessed study eligibility, individuals participated in diagnostic interviews and completed self-report measures. Eligible participants joined a closed cohort of four to seven participants. For the duration of treatment, participants were housed in a hotel located close to the clinic. Treatment was provided by licensed clinical psychologists, post-doctoral clinicians, or clinical psychology doctoral students. All treatment providers were trained in the theory and implementation of TMT and were closely supervised.

Session one of the treatment involved construction of the imaginal scene based on the individual's index trauma. Beginning with session two, individuals engaged with daily exposure therapy in the morning and group therapy in the afternoon. During individual exposure therapy, the clinician queried about distress using the SUDS Likert Scale every five minutes. The exposure continued until WSH was achieved as indicated by a minimum 50% reduction from in-session "Peak SUDS" and the clinician's behavioral observations. Imaginal exposure was conducted for up to 14 sessions and was terminated after evidence of BSH as indicated by the absence of SUDS increase following imaginal scene exposure.

Group therapy modules were co-led by two clinicians. During the 90-minute session, the clinicians facilitated discussions, modeling of skills, behavioral rehearsals, and feedback. The topics of the group therapy modules were Social Reintegration (five sessions), Anger Management

(five sessions), and Brief Behavioral Activation (four sessions). Participants completed daily assignments related to session content.

Occupation-related PTSD treatment protocol.

Participants contacted the clinic and completed a telephone screen to assess appropriateness for treatment participation (i.e., first responder and traumatic event exposure). Next, the first responder participated in an intake session with a licensed clinical psychologist, master's level social-work clinicians, post-doctoral clinicians, or clinical psychology doctoral students. All treatment providers were trained in the theory and implementation of TMT and were closely supervised. A licensed clinical psychologist supervised all sessions and cases were discussed during individual and group supervision meetings. During the intake session, patients participated in diagnostic interviews and completed self-report measures (see first responder assessment section). Eligible patients joined a closed cohort that comprised a combination of other first responders, active duty military, and military veterans. The number of group members and group composition varied depending upon those seeking treatment at any one time. For the duration of treatment, participants were housed in a hotel located close to the clinic.

After the assessment process, the next session involved a collaborative effort between the clinician and the patient to construct an imaginal exposure scene. Beginning with session 2, individuals engaged with daily exposure therapy in the morning and group therapy in the afternoon. During individual exposure therapy, the clinician queried about distress using the SUDS Likert Scale every five minutes. The exposure continued until within-session habituation (WSH) was achieved as indicated by a minimum 50% reduction from in-session "Peak SUDS" and the clinician's behavioral observations. Imaginal exposure was conducted for up to 14

sessions and was terminated after evidence of between-session habituation (BSH) as indicated by the absence of SUDS increase following imaginal scene exposure.

The group component of TMT-IOP is based on the protocol described in Beidel et al. (2017). The topics of the group therapy modules were modified in session content and a module was added. The modules in the TMT-IOP group component included Social Reintegration (three sessions), Anger Management (four sessions), Brief Behavioral Activation (four sessions), Sleep Hygiene (two sessions), and Relapse Prevention (one session). Group therapy modules were co-led by two clinicians. During the 90-minute session, the clinicians facilitated discussions, modeling of skills, behavioral rehearsals, and feedback. Participants completed daily assignments related to session content.

Measures

Group comparison variables, exposure therapy process variables, exposure therapy session variables, and PTSD symptom severity variables were used to evaluate the proposed hypotheses. Data collection of variables was consistent across the two dataset sources.

Group Comparison Variables.

The secondary dataset represented the combination of patients that sought treatment for combat-related PTSD and occupation-related PTSD. Two independent variables (IV) each with two levels were created to investigate the proposed hypotheses. The first IV aggregated participants by the number of index traumas they reported during the intake process and the second IV aggregated participants by the type of trauma for which the participant sought treatment.

Trauma frequency.

A variable was created to dichotomize the number of index traumas into the categories of single and multiple. To identify the appropriate trauma frequency label, manual searches of the Criterion A sections of CAPS and CAPS-5 were performed. Additionally, the researcher performed manual searches of the patients' clinical file, and the exposure therapy trauma narratives were reviewed.

Trauma type.

A variable was created to dichotomize participants by type of trauma. The two categories were combat-related PTSD and occupation-related PTSD. Additionally, a manual search of the patient file for participants identified as occupation-related PTSD was performed to confirm the patient's place of employment and occupation.

Exposure Therapy Process Variables.

The exposure therapy process variables were all created based on Subjective Units of Distress (SUDS). SUDS ratings are self-report ratings of the participants' subjective fear and anxiety experienced during the imaginal exposure. SUDS ratings were provided on a 9-point Likert scale that ranged from 0 (no distress) to 8 (extreme distress). SUDS ratings were collected at the start of the exposure (baseline) and queried every five minutes during the exposure. Specific therapy process variables examined in this study include Peak SUDS ratings, time to within-session habituation, and extinction rate (between-session and within-session). Operational definitions are largely based on prior conceptualizations (see Craske et al., 2008; S. M. Turner, Beidel, Long, & Greenhouse, 1992).

Peak SUDS.

Highest reported SUDS rated during the exposure session.

BSH extinction rate.

Slope of ‘Peak SUDS’ ratings over time, by participant. Primary measure of interest used to evaluate if more exposure to traumatic events attenuated the between-session habituation response during exposure therapy for PTSD.

Time to WSH (TWSH).

The amount of time (in minutes) that lapsed until WSH was identified during the session. WSH was identified by a minimum 50% reduction from in-session Peak SUDS rating *or* a return to baseline SUDS rating, whichever occurred first.

WSH extinction rate.

Slope of ‘Time to WSH’ over time, by participant. Primary measure of interest used to evaluate if more exposure to traumatic events attenuated the within-session habituation response during exposure therapy for PTSD.

Lowest SUDS.

Lowest reported SUDS rated during the exposure session, excluding the baseline SUDS rating.

Exposure Therapy Session Variables.

Length of sessions.

The sum of session duration (in minutes), as defined by the total time the participant engaged in an exposure therapy session, across sessions.

Number of sessions.

The total number of exposure therapy sessions that the participant attended.

PTSD Symptom Severity Variables.

PTSD severity variables were created separately for participants with combat-related PTSD and occupation-related PTSD. This was necessary because of the change in DSM criteria for PTSD.

PTSD symptom severity variables for combat-related PTSD.

The PTSD Checklist-Military Version (PCL-M; Weathers, Litz, Herman, Huska, & Keane, 1993) was completed by participants from the randomized controlled trial. This version of the PCL-M was used because the study was initiated prior to the release of the DSM-5. The PCL-M is a 17-item self-report measure of PTSD symptoms. The measure asked about “stressful military experiences,” and each item corresponded with DSM-IV-TR PTSD diagnostic criteria. Participants completed the PCL-M at pre-treatment, post-treatment, and 3-month and 6-month follow-up. Additionally, participants completed the PCL-M at the beginning of each week of treatment and assessed their symptoms “over the past week.” Items were rated from 1 “not at all” to 5 “extremely.” Severity scores range from 17 to 85. The current study used the PCL-M scores from pre-treatment and post-treatment.

PTSD severity variables for occupation-related PTSD.

The PTSD Checklist for DSM-5 (PCL-5; Weathers et al., 2013) was completed by participants that presented to the community clinic and identified as first responders. The PCL-5 is a 20-item self-report measure. Each item corresponded with the 20 symptoms listed in the DSM-5. The PCL-5 can be scored by calculating a total severity score (range = 0 - 80) by summing all items. Cluster scores can also be calculated by summing the items within a cluster (Cluster B, items 1-5; Cluster C, items 6-7; Cluster D, items 8-14; Cluster E, items 15-20). Items were rated from 0 “not at all” to 4 “extremely.” Participants completed the PCL-5 at pre-treatment, post-treatment, and 3-month and 6-month follow-up. Additionally, participants completed the PCL-5 at the beginning of each week of treatment and assessed their symptoms “over the past week.” The current study used the PCL-5 scores pre-treatment and post-treatment.

Statistical Strategy

Data was analyzed in jamovi (2018) and ‘RStudio’ (2016). Hypotheses one through four assessed the trajectory of habituation response (BSH and WSH) over the course of treatment. Linear Mixed Effects Regression (LMER) was used because of its ability to overcome missing data across time-points, overcome assumptions related to covariance, independence, and homogeneity of regression slopes (Laird & Ware, 1982; Singer & Willett, 2003; West, 2009; West, Welch, & Galecki, 2014). The LMER data analyses followed the recommendations proposed by Singer and Willett (2003). First, descriptive analyses with empirical growth plots were produced. Second, data were fit for an unconditional means model (model A). Third, data were fit for an unconditional growth model (model B). Lastly, the data were fit separately for the uncontrolled effects of trauma frequency and trauma type (model C). Finally, models were compared to identify

the best fitting model. Hypotheses five through 26 assessed exposure therapy session variables and PTSD severity variables using parametric and non-parametric statistical analyses.

RESULTS

Trauma frequency

Hypothesis 1: Between-Session Habituation.

An unconditional means model (Model A-BSH), unconditional growth model (Model B-BSH), and a model for the uncontrolled effects of trauma frequency (Model C-BSH TF) were constructed to determine if participants with multiple index traumas experienced a slower rate of between-session habituation (BSH) (Table 3). Rejection of the null hypothesis ($p < .001$) for Model A-BSH confirmed that the average Peak SUDS was non-zero; therefore, variance exists both within and between participants. Specifically, 22.64% (ICC) of the total variation in Peak SUDS ratings is attributed to differences among participants. Model B-BSH revealed that the relationship between true rate of change in Peak SUDS rating and session number is negative and moderate ($r = -0.34$). As displayed in Table 3, the average initial Peak SUDS rating is approximately 8 and over time the Peak SUDS rating decreased by an average of 0.48 SUDS rating. However, there was no difference in the average initial Peak SUDS rating, and there was no difference over time for a decrease in Peak SUDS ratings between participants with multiple index traumas and a single index trauma (Model C-BSH TF). The results failed to support the hypothesis that there would be a difference in BSH by trauma frequency. The best fitting model, of the significant models, was the unconditional growth model (Model B-BSH) ($\chi^2(3) = 1230.3$, $p < .001$)).

Hypothesis 2: Within-Session Habituation

An unconditional means model (Model A-WSH), unconditional growth model (Model B-WSH), and a model for the uncontrolled effects of trauma frequency (Model C-WSH TF) were constructed to determine if participants with multiple index traumas experienced a slower rate of within-session habituation (WSH) (Table 4). Rejection of the null hypothesis ($p < .001$) for Model A-WSH confirmed that the average time (in minutes) to within-session habituation was non-zero; therefore, variance exists both within and between participants. Specifically, 30.89% (ICC) of the total variation in time to within-session habituation (TWSH) is attributed to differences among participants. The relationship between true rate of change in TWSH and session number is negative and moderate ($r = -0.43$). As displayed in Table 4, the average initial TWSH was 66 minutes and over time TWSH decreases by an average of 2.57 minutes (Model B-WSH). There was no difference in the average initial TWSH, and there was no difference over time for TWSH between participants with multiple index traumas and a single index trauma (Model C-WSH TF). These results indicate there is no difference in within-session habituation between participants with multiple index traumas and a single index trauma. The results failed to support the hypothesis that there would be a difference in WSH by trauma frequency. The best fitting model, of the significant models, was the unconditional growth model (Model B-WSH) ($\chi^2(3) = 183.56, p < .001$).

Trauma Type

Hypothesis 3: Between-Session Habituation.

An unconditional means model (Model A-BSH), unconditional growth model (Model B-BSH), and a model for the uncontrolled effects of trauma type (Model C-BSH TT) were constructed to determine if participants with occupation-related PTSD experienced a slower rate

of between-session habituation (BSH) than combat-related PTSD (Table 3). For hypothesis three, results for the unconditional means model (Model A-BSH) and unconditional growth model (Model B-BSH) are identical to those results observed for hypothesis one. The results do not differ because the models are fit using the same level-1 and level-2 predictors. Model C-BSH TT differs from Model C-BSH TF because different predictors were used to estimate the rate of change. The results indicated there is no difference in between session habituation (BSH) between participants with occupation-related PTSD and combat-related PTSD (Model C-BSH TT). The results failed to support the hypothesis that there would be a difference in BSH by trauma type.

Hypothesis 4: Within-Session Habituation.

An unconditional means model (Model A-WSH), unconditional growth model (Model B-WSH), and a model for the uncontrolled effects of trauma type (Model C-WSH TT) were constructed to determine if participants with occupation-related PTSD experienced a slower rate of within-session habituation (WSH) (Table 4) compared to participants with combat-related PTSD. The results for the unconditional means model (Model A-WSH) and unconditional growth model (Model B-WSH) are identical to those results observed for hypothesis two. The results do not differ because the models are fit using the same level-1 and level-2 predictors. Model C-WSH TT differs from Model C-WSH TF because different predictors were used to estimate the rate of change. The results indicated there is no difference in within-session habituation (WSH) between participants with occupation-related PTSD and combat-related PTSD (Model C-WSH TT). The results failed to support the hypothesis that there would be a difference in TWSH by trauma type.

Exposure Session Process Variables

Trauma frequency.

Hypothesis 5: Peak SUDS.

During the first session, those with multiple index traumas ($Mdn = 8.0$) reported one SUDS rating score higher than those with a single index trauma ($Mdn = 7.0$; $U = 1127$, $p = .029$). At the last session, participants with multiple index traumas ($Mdn = 3.0$) did not differ from participants with a single index trauma ($Mdn = 3.0$) on their reported Peak SUDS rating ($U = 1335$, $p = .338$).

Hypothesis 6: Lowest SUDS.

During the first imaginal exposure session, participants with multiple index traumas ($Mdn = 4.0$) did not report Lowest SUDS ratings that were higher than participants with a single index trauma ($Mdn = 4.0$; $U = 1286$, $p = .240$). Likewise, although participants with multiple index traumas ($Mdn = 2.0$) reported one SUDS rating score rating higher than participants with a single index trauma ($Mdn = 1.0$), this difference was not significant ($U = 1198$, $p = .107$).

Hypothesis 7: TWSH first and last session.

Among individuals that achieved WSH, those with multiple index traumas took more time to achieve WSH during the first session compared to those with a single index trauma ($p = .007$, one-tailed; Table 5). During the last imaginal exposure session, participants with multiple index traumas did not take more time to achieve within-session habituation than participants with a single index trauma ($p = .402$, one-tailed; Table 5).

Hypothesis 8: TWSH change.

RM-ANOVA results displayed in Table 6 depict there was a significant difference in the time to reach within-session habituation between the first and last imaginal exposure session; however, the amount of time did not differ based on trauma frequency ($p = .153$).

Trauma Type

Hypothesis 9: Peak SUDS.

Participants diagnosed with occupation-related PTSD ($Mdn = 8.0$) did not report significantly higher SUDS ratings during the first imaginal session than participants diagnosed with combat-related PTSD ($Mdn = 8.0$; $U = 1175$, $p = .296$). Similarly, at the last session, participants with occupation-related PTSD ($Mdn = 3.0$) did not differ from participants with combat-related PTSD ($Mdn = 3.0$) on their reported Max SUDS ($U = 1197$, $p = .437$).

Hypothesis 10: Lowest SUDS.

During the first imaginal exposure session, participants with occupation-related PTSD ($Mdn = 4.0$) did not report Lowest SUDS ratings that were higher than participants with combat-related PTSD ($Mdn = 4.0$; $U = 1172$, $p = .355$). Likewise, during the last session, although participants with occupation-related PTSD ($Mdn = 2.0$) reported one SUDS score rating higher than participants with combat-related PTSD ($Mdn = 1.0$), this difference was not significant ($U = 1233$, $p = .570$).

Hypothesis 11: TWSH first and last session.

In comparison to participants with combat-related PTSD, participants diagnosed with occupation-related PTSD did not take longer to achieve WSH during the first ($p = .400$) or last ($p = .944$) sessions of imaginal exposure (see Table 5).

Hypothesis 12: TWSH change.

RM-ANOVA results (see Table 6) represent there was a significant difference in the time to reach within-session habituation between the first and last imaginal exposure session; however, the amount of time did not differ based on trauma type ($p = .743$).

Exposure Session Therapy Variables

Trauma frequency.

Hypothesis 13: Session length.

As displayed in Table 5, independent samples t-test for the length of the first imaginal exposure session was significant ($p = .027$). Participants with multiple index traumas engaged in a significantly longer first imaginal exposure session than participants with a single index trauma. However, participants with multiple index traumas did not engage in significantly longer last imaginal exposure sessions, nor did the overall total minutes engaged with imaginal exposure.

Hypothesis 14: Change in session length.

Table 7 depicts results from the Repeated-Measures Analysis of Variance (RM-ANOVA) for change in session length. There was a significant difference between the length (in minutes)

of imaginal exposure session during the first session compared to the last session; however, the amount of time did not differ based on trauma frequency ($p = .544$).

Hypothesis 15: Session number.

The nonparametric (Mann-Whitney U) test for the number of imaginal exposure sessions was not significant. Participants with multiple index traumas ($Mdn = 10.0$) did not require significantly more imaginal exposure sessions than participants with a single index trauma ($Mdn=10.0$; $U = 1,188$, $p = .202$).

Trauma Type

Hypothesis 16: Session length.

As displayed in Table 5, independent samples t-tests for the length of the first imaginal exposure session, last imaginal exposure session, and total minutes of imaginal exposure was not significantly different. Participants with occupation-related PTSD did not engage in longer first and last sessions of imaginal exposure, or the total minutes of imaginal exposure.

Hypothesis 17: Change in session length.

Table 7 depicts results from the RM-ANOVA for change in session length. There was a significant difference between the length (in minutes) of imaginal exposure session during the first session compared to the last session; however, the amount of time did not differ based on the trauma type ($p = .800$).

Hypothesis 18: Session number

The nonparametric (Mann-Whitney U) test for the number of imaginal exposure sessions was not significant. Participants diagnosed with occupation-related PTSD ($Mdn = 9.0$) did not

require significantly more imaginal exposure sessions than participants with combat-related PTSD ($Mdn = 10.0$; $U = 1,041$, $p = .956$).

PTSD Symptom Severity Variables

Different measures of PTSD symptom severity were completed by participants; therefore, comparisons solely on the basis of Trauma Type were not performed. PTSD symptom severity was analyzed by comparing the differences between multiple and single index trauma among occupation-related PTSD and multiple and single index trauma among combat-related PTSD, separately. Results are displayed in Table 8 and Table 9.

Hypothesis 19: PCL-M.

Participants diagnosed with combat-related PTSD and multiple index traumas did not enter treatment ($p = .295$, one-tailed) or discharge from treatment ($p = .113$, one-tailed) with higher self-report PTSD symptoms than participants diagnosed with combat-related PTSD and a single traumatic event (Table 8). RM-ANOVA for change from pre- to post-treatment (see Table 9) demonstrated that PCL-M scores significantly decreased from intake to discharge ($p < .001$); however, there was no interaction effect for trauma frequency ($p = .279$).

Hypothesis 20: PCL-5.

Participants diagnosed with occupation-related PTSD and multiple index traumas did not enter treatment ($p = .804$, one-tailed) or discharge from treatment ($p = .315$, one-tailed) with higher self-report PTSD symptoms than participants diagnosed with occupation-related PTSD and a single traumatic event (Table 8). RM-ANOVA for change from pre- to post-treatment

demonstrated that PCL-5 scores significantly decreased from intake to discharge ($p < .001$); however, there was no interaction effect for trauma frequency ($p = .201$; Table 9).

DISCUSSION

The current DSM-5 criteria for PTSD affords heterogeneous symptom presentations. There are approximately 636,120 different combinations of symptom presentations that would satisfy PTSD diagnostic criteria (Galatzer-Levy & Bryant, 2013). The number of possible presentations is even higher because the above number does not take into consideration trauma frequency (single index trauma, multiple index traumas), type of trauma (i.e., combat, occupational, interpersonal), delayed onset, or the presence of dissociative symptoms. These different symptom profiles that exist within the PTSD framework lead to questions about the generalizability of treatment outcomes from one group to another group. As our understanding of PTSD etiology grows so too does the need to employ treatment strategies that will maximize the benefits of treatment (Armour, Fried, Deserno, Tsai, & Pietrzak, 2017; Friedman, Resick, Bryant, & Brewin, 2011; Maples-Keller, Price, Rauch, Ger-Ardi, & Rothbaum, 2016; Seligowski, Rogers, & Orcutt, 2016). These treatment maximization strategies may encompass specific treatment recommendations that align with certain PTSD diagnosis subtypes. Conversely, these treatment strategies will not be needed because treatment is efficacious regardless of PTSD diagnosis subtype.

Two of the most apparent presentation differences are that of trauma frequency (TF) and trauma type (TT). There is some concern that empirically-based treatments for PTSD may be differentially effective for different subtypes. For example, extant literature questions the efficacy of exposure therapy for individuals with exposure to multiple traumatic events and certain types of traumatic events (Eagle & Kaminer, 2013; Hendriks et al., 2018; Jerud et al., 2017). It has been suggested that exposure to more traumatic events and certain types of traumas

predict worse distress and symptom reduction for exposure-based treatments (Green et al., 2000; Jerud et al., 2017; McLaughlin et al., 2013; Price et al., 2013; Suliman et al., 2009).

Two likely theoretical underpinnings can help explain why treatment would and would not be equally effective among PTSD subtypes. An underlying principle of learning theory explains that extinguishing one conditioned stimulus (CS) also extinguishes another CS (Pavlov, 1927). This principle has direct applications for practitioners interested in the efficacious treatment for PTSD. Specifically, the seminal literature observed that a flooding approach is efficacious for behaviors that have been acquired gradually (i.e., multiple index traumas) and those acquired during a single trial (i.e., single index trauma) and regardless of the type of trauma (Baum, 1970; Marks & Tobeña, 1990; Mineka, 1979; Pavlov, 1927). In terms of treatment, this would reason that exposure treatment delivered using a flooding approach would produce favorable outcomes in any scenario.

Additionally, a flooding approach rapidly extinguishes avoidance (Baum, 1970; Marks & Tobeña, 1990; Mineka, 1979; Sherman, 1970). Among individuals with anxiety and PTSD, avoidance impedes contact with the feared stimuli thereby serving as negative reinforcement (e.g., reducing symptoms of anxiety) (Dymond, 2019; Lovibond, Mitchell, Minard, Brady, & Menzies, 2009; Mineka, 1979; Zuj & Norrholm, 2019). The process of flooding during exposure therapy precludes an individual from engaging in an avoidance response, thereby extinguishing their previously used maladaptive strategy for managing anxiety symptoms. Within the avoidance learning paradigm, this process is known as *unavoidable aversive event + response prevention*, which is an effective extinction procedure (Dymond, 2019). For treatment, this similarly reasons that flooding during exposure therapy serves as an extinction of the avoidance

response and suggests that individuals with multiple index traumas could benefit from exposure therapy.

On the other hand, learning theory explains that acquisition of a behavior or a response is dependent upon reinforcement, and the type of reinforcement impacts the extinction of that behavior (Bouton, 2004; Grant et al., 1951; Hothersall, 1966; Humphreys, 1939, 1943; Longenecker et al., 1952). Individuals that report multiple index traumas and individuals that report their occupation as a first responder likely experience traumatic incidents on a partial reinforcement schedule. Practitioners should be concerned about the effects of partial reinforcement on treatment because under this type of reinforcement schedule, resistance to extinction is stronger (e.g., takes more time and trials) (Bouton, 2004; Grant et al., 1951; Hothersall, 1966; Humphreys, 1939, 1943; Longenecker et al., 1952). This understanding lends support to the hypothesis that both individuals with multiple index traumas and occupation-related PTSD will not respond well to exposure therapy.

Prior investigations have not directly examined the process variables, which provide a better indication of the ability to engage in treatment, critical for exposure therapy. The present study represents the first comparative examination of exposure therapy process variables on the ability to engage in exposure therapy. In other words, this is the first study in which exposure therapy process variables were examined to elucidate differences by trauma frequency (single index trauma vs. multiple index traumas) and trauma type (combat-related PTSD vs. occupation-related PTSD).

Examination of the exposure therapy process variables by trauma frequency and trauma type revealed similar results. The analyses examining TF revealed that individuals with a single

index trauma and multiple index traumas experienced similar rates of change in BSH and WSH. This finding is in direct contrast with the proposed hypotheses that individuals with multiple index traumas would exhibit a slower rate of decline in BSH and WSH. The notion that individuals with multiple index traumas do not habituate more slowly over the course of treatment has theoretical implications about our understanding of Pavlovian fear conditioning for PTSD. If the current results were to support the Pavlovian fear conditioning parameter that fear acquisition bears significant weight on the effectiveness of treatment, then the results would demonstrate that individuals with multiple index traumas have smaller units of change in Peak SUDS and TWSH across sessions, as illustrated by flatter slopes. For clinicians, the practical implications would include the need to assess the number of index traumas, allocate more treatment sessions, and schedule longer treatment sessions.

The current results do not support the Pavlovian fear conditioning parameter that fear acquisition bears significant weight on the effectiveness of treatment. Specifically, a partial reinforcement schedule, or for that matter, any acquisition schedule of the conditioned response (CR), did not attenuate BSH and WSH habituation during exposure therapy. Instead, the results lend initial support that a flooding approach is a type of *unavoidable aversive event + response prevention* and that this mechanism for extinction may be stronger than the effects that a partial reinforcement schedule during fear acquisition has on fear habituation. This finding has several treatment implications. First, it is critical that the clinician understands the principles of flooding. Second, the clinician must work with the patient to craft a trauma narrative that floods the patient, which can include the use of multiple sensory stimuli (olfaction, tactile, auditory, visual). Third, the clinician must take note of any observable avoidance tactics the patient is

using and ask the patient if they are using any overt or covert avoidance strategies. Fourth, the clinician must understand the importance and rationale for motivating the patient to persist and not use previously learned techniques (overt or covert) to manage their anxiety.

Similarly, in this study, analyses examining TT do not support that fear habituation is attenuated by fear acquisition. Specifically, individuals with combat-related exposure and occupation-related exposure experienced similar rates of change in BSH and WSH. This finding is in direct contrast with the proposed hypotheses that trauma type would bear significance on the rate of decline in BSH and WSH. The notion that individuals with occupation-related exposure do not habituate more slowly throughout treatment has theoretical implications about our understanding of Pavlovian fear conditioning for PTSD. To support the weight fear acquisition bears on the effectiveness of treatment, then the current results would have depicted flatter slopes for participants with occupation-related PTSD on the outcomes of Peak SUDS and TWSH. For clinicians, the practical implications would include the need to extensively interview the patient about the different types of trauma, target treatment for each type of trauma, and allocate more treatment sessions, and longer treatment sessions. The results of these analyses lend initial support that a flooding approach is effective regardless of the type of trauma frequency.

Together, linear mixed effects regression analyses indicated that trauma frequency and trauma type did not attenuate habituation (BSH and WSH). Individuals with multiple index traumas or first responders diagnosed with occupation-related PTSD do not engage in exposure therapy differently or less effectively than individuals with a single index trauma or military personnel diagnosed with combat-related PTSD.

Analyses of exposure session variables only revealed significant differences during the first imaginal exposure session. Participants with multiple index traumas reported higher Peak SUDS ratings (1-point), a longer session (8.2-minutes), and longer time to achieve WSH (13.8-minutes). The clinical significance of this is not known; however, this difference may suggest that individuals with multiple index traumas may experience greater fear activation during the first imaginal exposure session, which contributed to needing more time to achieve WSH, which impacted the length of the session. These differences observed during the first imaginal exposure session for trauma frequency may lend support to the theory that the reinforcement schedule for fear acquisition impacts treatment. However, these same differences were not observed for trauma type. The lack of significant findings for TT suggests the number of index traumas not the type of trauma (combat-related vs. occupation-related) is important for fear acquisition.

In light of the significant differences observed for TF during the first imaginal exposure session, these differences did not impact the overall habituation trajectory. Further examination of the first imaginal exposure session revealed that 78.1% of participants achieved WSH. When aggregated by single index trauma (79.3%) versus multiple index traumas (77.3%), no significant difference emerged ($\chi^2(1) = 0.051, p = .821$). Similarly, no significant difference emerged between combat-related PTSD (79.4%) and occupation-related PTSD (73.1%; $\chi^2(1) = 0.487, p = .485$). The theoretical implications of these findings suggest initial support that avoidance extinction occurs during flooding for TF and TT, and that fear acquisition may only impact the first session, but not subsequent sessions and not for trauma type. Treatment implications of these findings suggest that clinicians should be prepared to schedule a longer first

imaginal exposure session with patients that report multiple index traumas, but longer sessions may not be necessary for certain types of trauma.

As previously stated, there were several non-significant findings. Both groups for TF and TT reported a median Peak SUDS of 3.0 during the last session. Similarly, the median lowest SUDS ratings during the first session for both TT and TF groups was 4.0 and continued to decrease to reported median SUDS of 2.0 (multiple index traumas and occupation-related PTSD) and 1.0 (single index trauma and combat-related PTSD) during the last exposure session. These were consistent with the findings of the LMER analyses, and this suggests that all participants were able to experience fear habituation. If fear acquisition contributed to a differential exposure therapy experience, it would be expected that participants that reported multiple index traumas and participants that reported occupation-related PTSD would report higher SUDS ratings during the last session and higher lowest SUDS ratings during the first and last sessions. The results do not suggest that fear acquisition resulted in groups experiencing a lesser degree of fear habituation. These findings were in contrast with the proposed hypotheses and lent support to the notion that a flooding approach serves as an extinction of the avoidance response.

The extinction of the avoidance response is further highlighted by the lack of significant results for TF and TT in the difference for the amount of time to achieve WSH between the first and last session; the lack of differences during the first and last sessions in the amount of time to achieve WSH; the total session length across all sessions; the length of the first session and length of the last session; and the total number of exposure sessions. The results of these analyses also lent initial support that a flooding approach is effective and that this mechanism for avoidance extinction may overcome a partial reinforcement schedule described by Pavlovian fear

conditioning. Treatment implications of these results indicate that although there was no statistically significant difference for the total session length in minutes for both TF and TT, the amount of time corresponds with an additional 1-hour and 10-minutes of treatment for individuals with multiple index traumas and an additional 1-hour and 30-minutes of treatment for combat-related PTSD. These variations in time can be important for clinicians to consider when scheduling patients and planning for adequate avoidance extinction.

The limitations for the measurement of PTSD symptom severity variables required the results to be separated by trauma type. To support the hypothesis that multiple exposures to traumatic events predict worse distress reduction (Jerud et al., 2017; McTeague et al., 2010) the difference in PTSD symptom reduction from pre- to post-test should be significant. Additionally, if the hypothesis about blunted neurophysiological reactivity among individuals exposed to multiple traumatic events (Cuthbert et al., 2003; D'Andrea et al., 2013; Hagenaars et al., 2012; Lang & McTeague, 2011; McTeague et al., 2010) were to hold, we would expect participants with multiple index traumas to enter treatment with lower PTSD symptoms and discharge from treatment with higher PTSD symptoms than participants with a single index trauma. The findings of the current study did not support these hypotheses.

The results of the current study suggested that multiple index traumas do not impair the ability to engage in treatment as measured by PTSD symptom severity. Specifically, no differences were observed across sessions, and participants with multiple traumatic events did not enter treatment with lower PTSD symptom severity or discharge from treatment with greater PTSD symptom severity. At the time of discharge, 92% of participants with occupation-related PTSD were below the PCL-5 cut-off (score = 33) for probable PTSD, and 67% of participants with

combat-related PTSD were below the PCL-M cut-off (score = 50) for probable PTSD. Ninety-two percent of participants with occupation-related PTSD and 68% of participants with combat-related PTSD experienced clinically significant change, as calculated using the Jacobson and Truax (1991) method. Further, the average score decreases on the PCL-5 and PCL-M were 30 points ($SE = 2.37, t(25) = 12.63, p < .001$) and 19.7 points ($SE = 1.99; t(93) = 9.87, p < .001$), respectively. The current results indicate that participants were capable of participating in exposure therapy and experiencing the benefits of reduced PTSD symptoms.

Limitations

The current study offered initial support for additional understanding of Pavlovian fear conditioning, habituation, and avoidance extinction; however, several limitations exist. First, the current study was not well suited to draw conclusions about the extinction of the avoidance response described above, and there was no direct measure of avoidance; however, the results are in initial support of this relationship. Future research is needed to elucidate this relationship, and Dymond (2019) proposes strategies for studying the avoidance extinction learning paradigm. Also, it is important to consider that the current results may be entangled within the evidence that massed exposure sessions result in greater decrements in anxiety and avoidance (Baum, 1970; Foa, Jameson, Turner, & Payne, 1980), rather than the hypothesis that a reduction in avoidance leads to positive treatment outcomes.

Despite the treatment protocol criterion of WSH, some sessions were terminated before achieving WSH. Descriptive analyses revealed that of all the 1,286 sessions attended by participants in this study, 72.7% were documented to have achieved WSH habituation. When aggregated by trauma frequency, the percent of participants that achieved WSH approached

significance, and when aggregated by trauma type, the results were significant. Among participants with multiple index traumas, 74.0% achieved WSH, and 68.4% of participants with a single index trauma achieved WSH ($\chi^2(1) = 3.43, p = .064$). Among participants diagnosed with combat-related PTSD, 76.1% achieved WSH and 57.9% of participants achieved WSH ($\chi^2(1) = 32.50, p < .001$). The results of the analysis for trauma type revealed that fewer than expected participants with occupation-related PTSD achieved WSH; more than expected participants diagnosed with combat-related PTSD achieved WSH; more than expected participants with occupation-related PTSD did not achieve WSH; and fewer than expected participants diagnosed with combat-related PTSD did not achieve WSH. It is unclear the source of these discrepancies given that the imaginal exposure treatment protocols were similar. This difference may be due to clinician adherence to the treatment protocol. For example, adherence to the treatment protocol may have been lower for participants with occupation-related PTSD because treatment was not part of a randomized controlled trial.

Furthermore, there was no standard protocol for the query of multiple index traumas; therefore, it is possible that for each patient, not every index trauma was captured. Examination of the trauma narratives revealed that of the 68 trauma narratives located for participants that endorsed exposure to multiple index traumas, 22 participants had trauma narratives that included multiple index traumas. Forty-six participants that endorsed exposure to multiple index traumas had trauma narratives that included exposure to only one index trauma. It is unclear how the presence of multiple index traumas in the trauma narrative impacted habituation; however, this may provide additional support for generalization of fear habituation and the avoidance extinction. Also, the designation of two or more index traumas labeled as the presence of

multiple index traumas may be arbitrary. The current analyses do not provide information about the difference between one and two index traumas or five and seven index traumas.

Similarly, although 73% of participants with multiple traumatic events no longer met self-report cut-off score for probable PTSD, the current study did not perform behavioral tests to the other identified index traumas to confirm habituation. Lastly, a larger sample size of participants diagnosed with occupation-related PTSD would allow greater generalization of results.

Future Directions

The current study raises interesting questions for future examination. There is a significant debate in the extant literature about the importance of WSH (Bluett et al., 2014; Craske et al., 2008; Sripada & Rauch, 2015; van Minnen & Hageraars, 2002). The results of the current study suggest that an examination of trajectory differences between those that achieved WSH during the first session and those that did not may provide additional understanding. Consideration for the role of self-efficacy and its relationship to first session WSH should be explored. Patients that achieve first session WSH may learn that they are capable of successfully engaging with previously avoided stimuli and experience a reduction in their anxiety response. Researchers could hypothesize that participants who achieve WSH during the first session will experience a more rapid habituation trajectory than participants who did not achieve WSH during the first session. Similarly, although the current study revealed no differences in the habituation trajectory based on TF and TT, it may be essential to compare the trajectories to treatments that do not include WSH in their protocol. This examination may provide additional

clarity about the relationship between a flooding procedure and avoidance extinction and the role of self-efficacy.

Further, more investigation is needed to elucidate the interplay of fear acquisition and avoidance extinction. The current study alludes to the ability for avoidance extinction to overcome the effects of fear acquisition on fear habituation; however, there is much room to grow. Growth in the understanding of learning mechanisms for avoidance extinction needs to take place within the experimental and clinical psychopathology arenas and the intersection of these two arenas.

Conclusion

Together, these results support the notion that the effectiveness of a flooding approach to extinguish avoidance behavior may overcome the impact of reinforcement schedule on fear habituation. Similarly, this may reason that a flooding approach overcomes any difference in the type of trauma to which a person was exposed (e.g., combat-related and occupation-related). The results of the present study contribute to the growing body of literature that suggests the exclusion of patients based on the pre-treatment characteristics of trauma frequency and trauma type is unfounded (Ragsdale et al., 2018; Schottenbauer, Glass, Arnkoff, Tendick, & Gray, 2008; Trachik, 2016; van Minnen, Arntz, & Keijsers, 2002).

APPENDIX A: TABLES

Table 1. Work-related and personal demographic characteristics

Work-Related Demographic		Personal Demographic	
Characteristic	n(%)	Characteristic	n(%)
Occupation at Treatment		Gender	
Military (Active Duty and Veteran)	102(79.7%)	Male	120(93.8%)
First Responder (Active Duty and Veteran)	26(20.3%)	Female	6.3%)
Current or prior military service		Race/Ethnicity	
Yes	105(82%)	Black	13(10.2%)
No	23(18%)	White	90(70.3%)
Employment Status		Hispanic	17(13.3%)
Active Duty	57(45.6%)	Other	8(6.3%)
Veteran/Retired	68(54.4%)	Education	
Trauma frequency		High School	18(14.2%)
Combat-PTSD	102(79.7%)	Some College	81(63.8%)
Occupation-PTSD	26(20.3%)	Bachelors	19(15.0%)
Served as Military or Civilian First Responder		Masters-Doctoral	9(7.1%)
Yes	42(32.8%)	Relationship Status	
No	86(67.2%)	Married	66(52.0%)
Number of Index Trauma		Separated	15(11.8%)
Single	29(23.0%)	Divorced	22(17.3%)
Multiple	97(77.0%)	Single	24(18.9%)
Occupational Specialty		Reported Past Sexual Trauma	
LEO	6(4.7%)	Yes	22(17.3%)
FF	19(14.8%)	No	105(82.7%)
EMS	1(0.8%)		

Table 1. Work-related and personal demographic characteristics

Work-Related Demographic		Personal Demographic	
Characteristic	n(%)	Characteristic	n(%)
Army	74(57.8%)		
Marine	11(8.6%)		
Air Force	9(7.0%)		
Navy	7(5.5%)		
Coast Guard	1(0.8%)		

Table 2. Comparisons of Demographic Characteristics Based on Trauma Frequency and Trauma Type

Characteristic	Trauma Frequency		Trauma Type	
	χ^2 (df)	<i>p</i>	χ^2 (df)	<i>p</i>
Gender	1.01(1)	.315	1.56(1)	.212
Race/Ethnicity	8.37(3)	.039	5.61(3)	.132
Education	0.572(3)	.903	4.16(3)	.245
Relationship Status	3.00(3)	.392	5.07(3)	.166
Employment Status	0.707(1)	.401	13.0(1)	<.001
Past Sexual Trauma	1.37(1)	.242	0.156(1)	.693
Served as Military or Civilian First Responder	0.00880(1)	.925	NA	
Number of Index Trauma	NA		0.0659(1)	.797
Occupational Specialty	7.09(7)	.420	NA	

Table 3. Linear Mixed Effects Regression for Between-Session Habituation (BSH)

		Parameter	Model A- BSH	Model B- BSH	Model C- BSH TF	Model C- BSH TT
Fixed Effects						
Initial Status, π_{0i}	Intercept	γ_{00}	5.76*** (0.09)	8.11*** (0.09)	7.89*** (0.41)	7.55*** (0.43)
		γ_{01}	-	-	0.13 (0.22)	0.31 (0.23)
Rate of Change, π_{1i}	Intercept	γ_{10}	-	-0.48*** (0.03)	-0.57** (0.12)	-0.51** (0.12)
		γ_{11}	-	-	0.05 (0.07)	0.02 (0.07)
Variance Components						
Level 1	Within- person	σ_{ε}^2	2.76 (1.66)	0.77 (0.8773)	0.76 (0.87)	0.77 (0.88)
Level 2	In initial status	σ_0^2	0.81 (0.90)	0.71 (0.8448)	0.71 (0.84)	0.70 (0.84)
	In rate of change	σ_1^2	-	0.08 (0.28)	0.08 (0.28)	0.08 (0.28)
Pseudo R ² Statistics and Goodness-of-fit						
σ_{ε}^2 Pseudo R ²			-	0.72	-	-
σ_0^2 Pseudo R ²			-	0.12	-	-
σ_1^2 Pseudo R ²			-	-	-	-
Deviance			5124.8	3894.5	3832.6	3891.7
AIC			5130.8	3906.5	3848.6	3907.7
BIC			5146.3	3937.5	3889.7	3949.0

Notes. * Standard errors are in parentheses. $p < .05$, ** $p < .01$, *** $p < .001$; Model A-WSH (Unconditional Means Model); Model B-WSH (Unconditional Growth Model); Model C-WSH TF (Uncontrolled Effects of Trauma Frequency); Model C-WSH TT (Uncontrolled Effects of Trauma Type).

Table 4. Linear Mixed Effects Regression for Within-Session Habituation (WSH)

		Parameter	Model A- WSH	Model B- WSH	Model C- WSH TF	Model C- WSH TT
Fixed Effects						
Initial Status, π_{0i}	Intercept	γ_{00}	51.62*** (1.29)	66.06*** (1.87)	60.36*** (8.3329)	67.46*** (4.45)
		γ_{01}	-	-	3.19 (4.55)	-1.85 (4.90)
Rate of Change, π_{1i}	Intercept	γ_{10}	-	-2.57*** (0.23)	-2.58** (1.08)	-2.27*** (0.59)
		γ_{11}	-	-	0.1050 (0.58)	-0.34 (0.64)
Variance Components						
Level 1	Within- person	σ_{ε}^2	346.50 (18.61)	248.11 (15.73)	248.567 (15.77)	248.16 (15.75)
Level 2	In initial status	σ_0^2	154.9 (12.45)	274.12 (16.56)	267.143 (16.35)	271.40 (16.47)
	In rate of change	σ_1^2	-	2.17 (1.47)	2.209 (1.49)	2.14 (1.46)
Pseudo R ² Statistics and Goodness-of-fit						
σ_{ε}^2 Pseudo R ²			-	0.28	-	-
σ_0^2 Pseudo R ²			-	-0.77	-	-
σ_1^2 Pseudo R ²			-	-	-	-
Deviance			8280.9	8097.4	7999.1	8096.1
AIC			8286.9	8109.4	8015.1	8112.1
BIC			8301.4	8138.4	8053.7	8150.8

Note. Standard errors are in parentheses. *p < .05, **p < .01, ***p < .001; Model A-WSH (Unconditional Means Model); Model B-WSH (Unconditional Growth Model); Model C-WSH TF (Uncontrolled Effects of Trauma Frequency); Model C-WSH TT (Uncontrolled Effects of Trauma Type).

Table 5. Independent Samples t-test for TWSH (Top) and Session Length (Bottom) by TT and TF

		<i>M(SD)</i>	<i>t(df)</i>	<i>p</i>	<i>d</i>
TWSH					
First Session					
TF	S	50.6(21.3)	-2.505(87)	<.01	-0.66
	M	64.4(20.9)			
TT	O	57.0(23.2)	0.85(88)	.400	0.02
	C	62.2(21.4)			
Last Session					
TF	S	34.6(21.1)	-0.248(96)	.402	-0.06
	M	35.7(19.3)			
TT	O	35.8(20.1)	0.07(98)	.944	0.24
	C	35.4(19.8)			
Session Length					
First Session					
TF	S	68.8(23.9)	-1.94(124)	.027	-0.41
	M	77.0(18.6)			
TT	O	74.2(18.9)	0.25(126)	.599	0.06
	C	75.3(20.4)			
Last Session					
TF	S	46.0(18.0)	-1.20(124)	.117	-0.25
	M	50.9(19.7)			
TT	O	50.0(18.5)	0.07(126)	.472	0.02
	C	49.7(19.6)			
Total					
TF	S	621.6(299.5)	-1.44(124)	.076	-0.30
	M	705.6(268.8)			
TT	O	613.1(211.3)	1.49(126)	.930	0.33
	C	703.3(289.9)			

Note. Mean and standard deviation reported in minutes. One-tailed p-values. TWSH = Time to Within Session Habituation. TF = Trauma Frequency; S = single index trauma; M = multiple index traumas; TT = Trauma Type, O = occupation-related PTSD; C = combat-related PTSD.

Table 6. Repeated Measures ANOVA for TWSH by TF (Top) and TT (Bottom)

	<i>SS</i> (df)	<i>MS</i>	<i>F</i>	<i>p</i>	η^2
Trauma Frequency					
Within Subjects Effects					
First-Last	12146(1)	12146	35.48	<.001	0.16
First-Last * TF	713(1)	713	2.08	.153	0.01
Residual	24991(73)	342			
Between Subjects Effects					
TF	1948(1)	1948	3.77	.06	0.03
Residual	37696(73)	516			
Trauma Type					
Within Subjects Effects					
First-Last	25761.9(1)	25761.9	80.83	<.001	0.21
First-Last * TT	20.5(1)	20.5	0.06	.800	0.00
Residual	40159.1(126)	318.7			
Between Subjects Effects					
TT	6.94(1)	6.94	0.01	.902	0.00
Residual	57823.69(126)	458.97	.		

Note. *SS* = Sum of Squares; *MS* = Mean Squares; First-Last = First-Last Session TWSH (minutes); TF = Trauma Frequency; TT = Trauma Type.

Table 7. Repeated Measures ANOVA for Session Length by TF (Top) and TT (Bottom)

	SS (df)	MS	<i>F</i>	<i>p</i>	η^2
Trauma Frequency					
Within Subjects Effects					
First-Last	26572(1)	26572	82.35	<.001	0.21
First-Last * TF	120(1)	120	0.37	.544	0.00
Residual	40013(124)	323			
Between Subjects Effects					
TF	1904(1)	1904	4.25	.04	0.02
Residual	55533(124)	448			
Trauma Type					
Within Subjects Effects					
First-Last	12241.7(1)	12241.7	35.27	<.001	0.16
First-Last * TT	37.7(1)	37.7	0.11	.743	0.00
Residual	25687.8(74)	347.1			
Between Subjects Effects					
TT	509(1)	509	0.94	.336	0.01
Residual	57823.69(126)	458.97	.		

Note. SS = Sum of Squares; MS = Mean Squares; First-Last = First-Last Session length (minutes); TF = Trauma Frequency; TT = Trauma Type,

Table 8. PTSD Severity Scores at Intake and Discharge

		Combat-Related PCL-M				Occupation-Related PCL-5					
		<i>M</i> (<i>SD</i>)	<i>t</i> (<i>df</i>)	<i>p</i>	<i>d</i>			<i>M</i> (<i>SD</i>)	<i>t</i> (<i>df</i>)	<i>p</i>	<i>d</i>
PCL- Intake	S	62.50(8.66)	-0.54(100)	.295	-0.13	S	61.40(10.90)	3.41(24)	.999	1.70	
	M	63.90(11.70)				M	45.60(8.98)				
PCL- Discharge	S	41.60(16.41)	-0.95(92)	.171	-0.23	S	24.0(19.60)	1.12(24)	.863	0.56	
	M	45.20(15.40)				M	17.40(9.59)				

Note. One-tailed *p*-values; S = single index trauma; M = multiple index traumas;

Table 9. Repeated Measures ANOVA for PTSD Symptom Severity Scores by TT

	SS (df)	MS	F	p	η^2
Occupation-Related PCL-5					
Within Subjects Effects					
First-Last	8687(1)	8687	125.73	<.001	0.57
First-Last * TF	171(1)	171.3	2.48	.128	0.01
Residual	1658(24)	69.1			
Between Subjects Effects					
TF	1017(1)	1904	4.25	.04	0.02
Residual	3811(24)	159			
Combat-Related PCL-M					
Within Subjects Effects					
First-Last	13690.2(1)	13690.2	72.56	<.001	0.44
First-Last * TF	26.7(1)	26.7	0.14	.707	0.00
Residual	17357.5(92)	188.7			
Between Subjects Effects					
TF	253(1)	253	1.40	.240	0.01
Residual	16645(92)	181			

Note. SS = Sum of Squares; MS = Mean Squares; First-Last = First-Last Session PTSD symptom severity scores; TF = Trauma Frequency; TT = Trauma Type.

APPENDIX B: IRB APPROVAL LETTER



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

Approval of Human Research

From: UCF Institutional Review Board #1
 FWA00000351, IRB00001138
To: Deborah Casamassa Beidel
Date: September 18, 2014

Dear Researcher:

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

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

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

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

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

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

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

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

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

A handwritten signature in black ink, appearing to read "J. B. and".

IRB Coordinator

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