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Amanda Joelle Brown Date

Re-Experiencing Symptom Self-Monitoring as an
Early Step in the Treatment of Posttraumatic Stress Disorder

By

Amanda Joelle Brown
Doctor of Philosophy

Psychology

Linda W. Craighead, Ph.D.
Advisor

Millie C. Astin, Ph.D.
Committee Member

Nancy G. Bliwise, Ph.D.
Committee Member

Bekh Bradley, Ph.D.
Committee Member

Lynne Nygaard, Ph.D.
Committee Member

Accepted:

Lisa A. Tedesco, Ph.D.
Dean of the James T. Laney School of Graduate Studies

Date

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By

Amanda Joelle Brown
A.B., Princeton University, 2006
M.A., Emory University, 2010

Advisor: Linda W. Craighead, Ph.D.

An abstract of
a dissertation submitted to the Faculty of the
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Abstract

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Early Step in the Treatment of Posttraumatic Stress Disorder
By Amanda Joelle Brown

The efficacy of a brief self-monitoring intervention was evaluated as an initial step in the treatment of PTSD. Participants were 137 American combat veterans, diagnosed with PTSD and enrolled in a five-week psychoeducation group at a large Veterans Affairs medical center while waiting to begin individual treatment. Nine groups were designated as active and the other six served as controls. In addition to engaging in the standard group intervention, participants in the active condition were asked to record the number and content of daily nightmares, flashbacks, intrusive thoughts/memories, and physiological and emotional reactions to triggers. Records were collected each week to assess compliance. Symptoms of PTSD and related psychopathology were assessed at the beginning and end of treatment. Data were analyzed for the 88 participants who attended at least two group sessions and completed baseline and final assessments (54 active, 34 controls). The self-monitoring intervention did not significantly reduce symptoms overall, but age moderated response to treatment such that younger veterans in the active group reported a decrease in avoidance symptoms. Compliance with the self-monitoring was low, suggesting that it was experienced as difficult; however, dropout rates did not differ between the conditions. Results indicate that four weeks of intrusion monitoring is not sufficient to effect substantial change in individuals with chronic, severe PTSD. However, it may be an appropriate early step in treatment for individuals with short illness duration and/or exposure to few traumatic events. Follow-up studies

are needed to examine the potential long-term impact of this early intervention on subsequent response to more intensive psychotherapies for PTSD.

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Table of Contents

Re-Experiencing Symptom Self-Monitoring as an Early Step in the Treatment of Posttraumatic Stress Disorder

Posttraumatic stress disorder (PTSD) is a serious mental illness affecting 5-7% of the general population in the United States (Kessler, Berglund, Demler, Jin, & Walters, 2005) and 11-20% of American veterans of the current conflicts in Iraq and Afghanistan (Hoge, Terhakopian, Castro, Messer, & Engel, 2007; Schneiderman, Braver, & Kang, 2008). According to the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2000) the diagnostic criteria for PTSD include three clusters of symptoms: re-experiencing symptoms, avoidance and numbing symptoms, and hyperarousal symptoms. These symptoms develop in the aftermath of a traumatic event, defined as an event that involves actual or threatened death, serious injury, or threat to one's physical integrity, and which is accompanied by subjective feelings of intense fear, horror, or helplessness (see Table 1 for full diagnostic criteria).

While the expression of PTSD symptoms is very common in the immediate aftermath of a trauma, in the majority of cases trauma survivors experience a natural decline in symptoms in the weeks and months following the traumatic event. For example, 94% of rape victims at two weeks post-trauma exhibited symptoms consistent with the diagnosis of PTSD, whereas only 47% met diagnostic criteria at three months post-trauma (Rothbaum & Foa, 1993, as cited in Resick, Monson, & Rizvi, 2008). After three months of persistent symptoms, PTSD is considered "chronic" and symptoms are less likely to decrease in the absence of therapeutic intervention (American Psychiatric Association, 2000; Resick et al., 2008).

Fortunately, PTSD is a treatable condition. A wide variety of intervention strategies have been used successfully to treat this disorder, with skills-focused

treatments, exposure-based treatments, cognitive therapy, and various combination treatments showing the greatest success in empirical studies (Resick et al., 2008). The clinical practice guidelines developed by the International Society for Traumatic Stress Studies identify exposure therapy, cognitive therapy, and interpersonal therapy as the most empirically-supported approaches to the treatment of PTSD (Nemeroff et al., 2006).

Prolonged Exposure Therapy (PE; Foa, Hembree, & Rothbaum, 2007) and Cognitive Processing Therapy (CPT; Resick & Schnicke, 1996) are two manualized treatments for PTSD that have received substantial empirical support and are now widely used to treat victims of rape, combat, and other traumas who are displaying symptoms of PTSD. Sixty to seventy percent of patients receiving either of these treatments show significant symptom remission at the end of treatment (Resick, Nishith, Weaver, Astin, & Feuer, 2002). In 2007, the Office of Mental Health Services initiated a rollout of PE and CPT at Veterans Affairs medical centers across the United States, formally recognizing the efficacy of these treatments. Currently, the demand for specialized PTSD treatment is high among America's combat veterans, and many face long waiting lists for treatment. In addition, PE and CPT require significant contributions of time, energy, and resources on the part of both patient and clinician. In light of these constraints, an empirically-supported stepped care approach may be helpful for effectively and efficiently decreasing symptom severity among individuals with PTSD with minimal associated cost and effort.

The current study evaluates the efficacy of a brief self-monitoring intervention designed to be an early step in the course of treatment for PTSD. Self-monitoring, the observation and recording of one's own physical and mental processes, is useful as both

an assessment and an intervention strategy (Korotitsch & Nelson-Gray, 1999). When used as a treatment approach, self-monitoring requires minimal clinician involvement and has been associated with positive outcome in a number of different disorders (e.g., eating disorders, mood disorders; Ilardi & Craighead, 1994; Korotitsch & Nelson-Gray, 1999; Latner & Wilson, 2002; Thiele, Laireiter, & Baumann, 2002). Four independent studies have found evidence of substantial improvement in PTSD symptoms after four weeks of daily written recording of the frequency and content of re-experiencing symptoms (e.g., nightmares, flashbacks, intrusive thoughts), in the absence of formal treatment (Ehlers et al., 2003; Hardy & Stallard, 2008; Smith et al., 2007; Tarrier, Sommerfield, Reynolds, & Pilgrim, 1999). The theoretical assumption underlying this research project is that self-monitoring of re-experiencing symptoms may decrease cognitive avoidance and encourage emotional processing of the traumatic event. This type of self-monitoring by itself is not expected to be as effective as more structured and intensive treatments such as PE or CPT, but we hypothesized that this minimal intervention would lead to some decrease in the frequency of and distress associated with symptoms of PTSD and related psychopathology, and thus might serve well as a cost-effective initial step in a comprehensive treatment program.

Theoretical Models of PTSD

For decades, clinical researchers interested in mental health problems associated with exposure to traumatic events have proposed theoretical models to account for the development and maintenance of PTSD symptoms. The following discussion aims to provide a theoretical context in which to ground the hypothetical mechanisms of action involved in re-experiencing symptom self-monitoring. Various theoretical models of the

development and maintenance of PTSD are reviewed, beginning with early theories and concluding with current theoretical conceptualizations of the disorder. Each theory's perspective on re-experiencing symptoms is highlighted, as are the implications of each theory for designing effective interventions for PTSD.

Early theories. Well before the term "PTSD" was accepted into the nomenclature of mental illness, clinicians working with victims of trauma grappled to make sense of this complex disorder. Early theories of PTSD grew out of broader perspectives on mental health and disorder. These accounts tended to focus on only a few salient aspects of the disorder and therefore failed to account for the full spectrum of symptoms typically presented by patients with this illness. Nonetheless, these early theories provided the foundation on which later, more complex, theoretical models were built.

Psychoanalytic theories. Early in his career, Sigmund Freud offered an account of post-traumatic "hysterical phenomena," claiming that traumatic experiences (usually sexual in nature) that occurred early in life were dissociated from conscious awareness but exerted a constant pressure on the mind of the victim, which resulted in fixation on the trauma and ultimately accounted for the clinical presentation of these patients (Breuer & Freud, 1893/1955, as cited in van der Kolk, 2007). Years later, after Freud revised this theory in favor of one in which such "early traumas" were recast as childhood sexual fantasies, he offered commentary on what was commonly termed "war neurosis," an early moniker for PTSD subsequent to combat trauma. Freud believed the symptoms of war neurosis were caused by the victim's inability to deal with the overwhelming

intensity of the traumatic event and a consequent “flooding of the mental apparatus” that occurred after the event (Freud, 1920, as cited in van der Kolk, 2007).

Freud was particularly fascinated by patients’ “compulsion to repeat the trauma,” or in modern terminology, re-experiencing symptoms. He suggested that these symptoms resulted from the repression of trauma memories outside of conscious awareness. Freud also noted a disconnect between the frequency and intensity of patients’ re-experiencing symptoms and their difficulty consciously recollecting key components of the event, a puzzling characteristic of PTSD that many clinicians and researchers have continued to grapple with for decades since, but he did not offer a comprehensive theory to account for these seemingly incompatible symptoms (van der Kolk, 2007). Another notable contributor to the psychoanalytic school of thought regarding the psychological effects of trauma was Kardiner (1941, as cited in van der Kolk, 2007), who observed and described such features of “traumatic neuroses” as physiological hyperarousal, chronic irritability, hyperstartle reactions, and increased aggression. The classification system he described in 1941 became the foundation of the formalized definition of PSTD in the DSM-III, published in 1980 (van der Kolk, 2007).

Other analytically-minded clinicians and theorists commented on features of traumatized veterans in the aftermath of World War II, and many tried to enhance analytic treatment for these patients by incorporating lessons learned from Kardiner’s astute observations (van der Kolk, 2007). Hypnosis was reintroduced into treatment as a means of uncovering repressed memories of the traumatic experience, and group-based interventions were developed to treat soldiers who had been exposed to the same traumatic events. Krystal (1968, as cited in van der Kolk, 2007), a psychoanalyst who

studied posttraumatic responses in concentration camp victims, suggested that anxiety and hyperarousal after a trauma evolves over time into inhibition of emotional and behavioral responses, ultimately leading to the loss of the ability to grasp the personal meaning of emotional states. While awareness of the complexity of posttraumatic reactions increased during this time, the overall theoretical perspective offered by the psychoanalytic school of thought remained relatively unchanged (van der Kolk, 2007).

***Behavioral models*.** The dominance of the psychoanalytic perspective on mental health and disorder began to weaken in the mid-19th century due to the increasing influence of behaviorism, which sought to define and explain psychological disorders solely in terms of observable behaviors and laws of behavior change that resembled mathematical and scientific postulates. The language of the analysts, with its emphasis on the unconscious mind, was substituted by such behavioral terms as conditioning, association, reinforcement, and stimulus generalization. This shift in theoretical orientation cast a new light on understanding the development and maintenance of all mental disorders, PTSD included(Hunt, 2009).

Early behavioral models of PTSD conceptualized the disorder according to Mowrer's two-factor learning theory (Mowrer, 1951), which explains fear acquisition in terms of both classical conditioning and associative learning. Behaviorists proposed that during a traumatic event, a wide range of neutral stimuli become associated with the unconditioned stimulus (a hurricane, enemy soldier, rapist, etc.). Following the trauma, these previously neutral stimuli elicit a fear response similar to that experienced at the time of the trauma, and cognitive and behavioral avoidance of these trauma-related stimuli prevents natural extinction of these incorrect associations. The avoidance itself is

reinforced by a reduction in fear, leading to the maintenance of the disorder (Foa, Steketee, & Rothbaum, 1989).

Behavioral models of PTSD suggest that re-experiencing symptoms are triggered by stimulus-response associations that develop after a trauma. Keane et al. (1985) pointed out that nightmares, flashbacks, and other re-experiencing symptoms act as "spontaneous exposures" that could serve a therapeutic purpose if allowed to run their course. However, these exposures tend to be incomplete and of short duration, so they are generally ineffective. Keane and colleagues (1985) suggested that in order for re-experiencing episodes to assist with the extinction of maladaptive stimulus-response connections, they must contain a wider range of trauma-related cues, last for a longer time, and evoke affect that matches that experienced at the time of the trauma. However, avoidance of these episodes and the stimuli likely to trigger them is negatively reinforced by a decrease in negative affect, which maintains the cycle of re-experiencing, fear, and avoidance.

Cognitive models. The so-called cognitive revolution of the 1960s reintroduced non-observable mental processes into the realm of experimental psychopathology (Hunt, 2009). Early cognitive models centered around the idea that the way in which traumatic events are represented in memory is both unique and important to understanding the development and maintenance of PTSD symptoms. These "information processing" theories can be classified as either schema-based or associative network-based, depending on their underlying assumptions about the nature of memory and cognition. Overall, these models have added to the behavioral perspective by considering not only

the learned associations between trauma-related stimuli and aversive reactions, but also the role of adaptive and maladaptive cognitive processes.

Schema-based models. In the language of cognitive psychology, a schema is a way of mentally representing knowledge that allows for information to be organized at various levels of abstraction(Dalgleish, 2004). Schemas develop over time by observing commonalities and regularities of experience, and they serve as blueprints into which future experiences may be fitted. Schemas also serve as filters through which new experiences are processed; information that is inconsistent with existing schemas requires assimilation (organization into an existing schema) or accommodation (a change in the nature of a schema that allows it to account for the new information). Schema-based models of PTSD account for posttraumatic difficulties by pointing to the challenge of assimilating trauma-related information into pre-existing schemas or accommodating the information by updating those schemas (Dalgleish, 2004).

Using schema-based information processing theory as the foundation of his model, Horowitz (1974) described “stress response syndromes” in terms of incongruities between new and old mental representations of the world that need to be worked through conceptually and emotionally in order to achieve integration(Horowitz, 1974). He proposed that mental representations of the traumatic event are stored in active memory rather than assimilated into the individual’s current schemas because of the incompatibility between the traumatic event and pre-existing representations of the world. Furthermore, Horowitz (1974) posited that in an attempt to reduce arousal, individuals actively defend against trauma-related memories. This defensive position (i.e., cognitive and behavioral avoidance) further impedes processing and integration,

which causes difficulties for the individual over time because of the conflict that arises between the high levels of distress surrounding schema-incompatible trauma memories and individuals' innate drive to assimilate new information into stable, coherent schemas.

Horowitz's schema-based model explains re-experiencing symptoms as indications that the trauma memory has not yet been successfully integrated into the victim's schematic representations of the world. He proposed that the active form of memory in which unassimilated trauma memories are stored has a tendency toward repeated representation, which triggers involuntary recollections until processing is completed. Horowitz (1974, 1978) noted that trauma victims often describe oscillation between episodes of intrusive memories or images and periods of profound cognitive avoidance, denial, dissociation, and other indications of efforts to keep the trauma information out of conscious awareness. He suggested that avoidance and numbing symptoms serve as defensive responses to the intrusive repetitions, and they impede the essential process of schematic assimilation and accommodation(Horowitz, 1978).

The goal of psychotherapy for PTSD, according to Horowitz's theory, is to facilitate a synthesis between the victims' cognitive schemas about themselves and the world and information about the traumatic event. The most common technique used to complete this task is detailed discussion in therapy of the details of the trauma and their meaning for the person(Horowitz, 1974). Horowitz (1974) wrote, "The problem in therapy is to provide tolerable doses of awareness because knowledge of the discrepancies between desire and reality leads to painful emotional responses" (p. 772). In other words, therapy offers a safe space in which trauma victims can recount their experiences and work toward integrating these experiences into their overarching mental

representations of the world. The active reconstruction of traumatic events in memory and subsequent discussion of the meaning of the event for the person is a key element of current exposure-based treatments and cognitive therapies for PTSD.

Another schema-based cognitive model of PTSD was proposed by Janoff-Bulman (1989). She identified three major schemas that are impacted by trauma: perceived benevolence of the world, perceived meaningfulness and controllability of the world, and perceived worthiness of the self(Janoff-Bulman, 1989). According to this model, traumatic events severely challenge these deeply embedded, fundamental schemas and thereby disrupt the trauma survivor's ability to objectively interpret new information (e.g., tending to misattribute danger to ambiguous stimuli) and to anticipate the future (e.g., becoming hopeless and pessimistic; having a sense of foreshortened future). She further posited that less flexibility or "viability" of these schemas may be associated with greater difficulty coping with trauma.

Janoff-Bulman (1989) adopted Horowitz's theory regarding intrusive symptoms and added that these symptoms may serve the purpose of facilitating active processing of trauma-related information. She proposed that repeatedly thinking about one's traumatic experience could contribute to the eventual assimilation of these new data into preexisting schemas. However, like Horowitz, she noted that denial, avoidance, dissociation, and similar maladaptive coping strategies often interrupt this process.

Associative network models. A different approach to understanding the nature of trauma memories and the etiology of PTSD draws on cognitive psychology research suggesting that information is processed and stored in associative networks that link together relevant thoughts, images, actions, and emotions. According to this view,

cognition involves the activation of information processing networks at many points in a distributed network, and learning results in the formation of new nodes within the network or in the formation of new connections among existing nodes. Furthermore, psychological disorders are hypothesized to result either from failures of these networks to operate appropriately or from the correct action of inappropriate networks(Dalgleish, 2004).

Foa and Kozak (1986) presented an information processing theory of PTSD based on an associative network view of memory and cognition. They posited that after a traumatic event, trauma-related emotions and cognitions are organized into information structures that guide individuals' reactions to stimuli. These "fear networks" include information about the feared situation; information about verbal, physiological, and overt behavioral responses to the situation; and interpretative information about the meaning of the stimulus and response elements of the structure. Fear structures therefore encode information about the level of danger associated with a stimulus and about the individual's preparedness for escape from the stimulus (Foa & Kozak, 1986). Foa and Kozak (1986) suggest that three things distinguish PTSD from other anxiety disorders: the pervasiveness of trauma-related stimuli, the intensity of the physiological and behavioral responses to the trauma, and the low threshold for activation of the fear structure, all of which render PTSD particularly disruptive to daily functioning.

Chemtob and colleagues (Chemtob, Roitblat, Hamada, Carlson, & Twentyman, 1988) also described a theoretical approach to PTSD that draws upon associative network models of information processing. Their theory, termed "cognitive action theory," posits that following a trauma, exceedingly strong connections are formed among threat-arousal

nodes, and associations between nodes representing constructs present during the trauma and other nodes signaling safety are inhibited. Because of this imbalance in the associative memory networks, activation of any trauma-related node – even those that had previously been associated with safety – will activate the fear network. In addition, the aversiveness of threat-arousal activation renders this network generally inhibited in daily life, which Chemtob et al. (1988) suggest may account for the difficulty many trauma survivors have in deliberately accessing trauma-related memories. Like Foa and Kozak (1986), these authors suggest that symptoms of PTSD are a direct result of faulty information processing resulting from the formation of maladaptive associative networks in memory.

Associative network models of PTSD offer specific hypotheses about the role of re-experiencing symptoms in PTSD. According to these theories, intrusive memories, nightmares, and flashbacks are caused by spreading activation from threat-arousal nodes to related nodes representing information about related situations and experiences. Because of the size and intensity of fear structures in memory, many stimuli can activate the behavioral, cognitive, and affective components of the structure, causing memories to come to mind involuntarily (Chemtob et al., 1988; Foa, Steketee, & Rothbaum, 1989). Furthermore, individuals with PTSD have higher "resting levels" of threat potentiation, tend to interpret neutral evidence as threatening, and have higher thresholds for the activation of arousal-dampening mechanisms (Chemtob et al., 1988), all of which increase the likelihood of activating threat-arousal nodes and facilitate the occurrence of re-experiencing episodes.

Adopting an associative network perspective, Creamer, Burgess, & Pattison (1992) tested the validity of a longitudinal model of PTSD using path analyses and found that re-experiencing symptoms were associated with two divergent outcomes. For some patients, intrusive symptoms preceded and predicted cognitive and behavioral avoidance, and such avoidance interfered with symptom improvement and led to poorer outcomes over the course of the study. In contrast, Creamer et al. (1992) found that high levels of intrusive symptoms powerfully predicted global symptom severity at a single point in time but were associated with a reduction in symptom levels some months later. The authors concluded from these findings that re-experiencing events may facilitate cognitive processing via modification of the fear network, but only if they prompt reflection and coping; if avoided or suppressed, modification of the network cannot take place and symptoms are maintained.

Creamer et al.'s (1992) findings and interpretation are in accord with associative network theorists' perspective on the key factors involved in the treatment of PTSD. These theoretical models propose that recovery from PTSD is facilitated by accessing the fear network and incorporating new information that is incompatible with the maladaptive components of the network into new, less pathological, associative structures in memory. Exposure-based treatment approaches such as PE (Foa et al., 2007) draw heavily on associative network theories of PTSD. Both imaginal and in vivo exposure techniques require individuals to confront stimuli that activate their fear structure and to continue the exposure to these stimuli long enough for habituation to occur. The decrease in arousal characteristic of habituation weakens the links between nodes in the

fear network and strengthens new, inhibitory connections between the previously feared stimuli and safety information (Foa et al., 2007).

Recent theories. More recent theoretical models of PTSD have moved away from choosing a single perspective on information processing and mental representation and toward more integrated approaches to understanding how trauma memories are processed and stored and how, in consequence, PTSD may be developed and maintained. There currently exists no single, comprehensive theory that dominates the field of PTSD research; rather, the following four theoretical models offer cogent and compelling explanations of the etiology, development, and maintenance of PTSD and account for much, if not all, of the empirical research on the disorder that has been conducted to date.

Emotional processing theory. Recent theoretical contributions by Foa and colleagues (e.g., Foa & Riggs, 1993; Foa & Rothbaum, 1998; Foa et al., 2007) have incorporated ideas from schema-based approaches into the associative network model of Foa and Kozak (1986) and Foa et al. (1989), creating a more comprehensive cognitive model known as emotional processing theory. The first of the three core components of emotional processing theory harkens back to Foa and colleagues' earlier associative network models. This aspect of the theory deals with the nature of trauma memories, which are hypothesized to contain a wider range of physiological, behavioral, and affective response elements compared with non-trauma memories. In addition, trauma victims' perception, attention, and memory for information about the traumatic event are posited to be systematically biased by the extreme emotions experienced at the time of the event's occurrence. Because of this, the fear networks associated with trauma-related stimuli tend to be larger and more disorganized than typical associative networks.

The second component of Foa and colleagues' updated theory incorporates schema-based accounts of information processing in PTSD. According to the theory, symptoms of PTSD may result either from a discrepancy between the experience of trauma and pre-existing schemas about oneself and the world (e.g., a sense of security and justice in the world) or from the activation of a maladaptive schema that is concordant with the experience of trauma (e.g., a sense of oneself as incompetent or the world as extremely dangerous). They suggest that PTSD develops when a negative self-schema (e.g., self as incompetent) interacts with a negative world-schema (e.g., the world as a dangerous place).

The third core component of emotional processing theory is the nature of post-trauma reactions experienced by the traumatized individual and by significant others in his or her life. The individual's interpretation of the event is hypothesized to play a key role in the development of PTSD and may account for many individual differences in risk and resilience. One victim may view the trauma as an unfortunate accident while another interprets it as an indication that no one in the world can be trusted. The reactions of others may intensify or undercut such interpretations, as unsympathetic, blaming, or disbelieving friends and family members may negatively impact the individual's appraisal of the event.

According to emotional processing theory, on which the highly effective treatment PE is based (Foa et al., 2007), symptom remission requires changes both in schematic representations of the meaning of the trauma and in the trauma memory records themselves. Both imaginal exposure to trauma memories and in vivo exposure to trauma reminders are used in PE to incorporate safety information into the trauma

memory network, while cognitive restructuring techniques are used during the processing of imaginal exposure to facilitate schema-level changes in how the individual views themselves and the world. Avoidance and emotional numbing are targeted directly through exposure practices, with the goal of activating the fear network and weakening maladaptive associations between neutral stimuli and the fear response.

Dual representation theory. Presenting another updated information processing model of PTSD, dual representation theory, Brewin, Dalgleish, and Joseph (1996) suggested that two processes in memory contribute to the unique characteristics of PTSD symptoms. The first is the verbally accessible memory (VAM) system, which represents the conscious experience of the traumatic event. VAM memories can be deliberately retrieved and edited and may include information about the sensory features of the event, the emotional and physiological reactions experienced at the time of encoding, and the perceived meaning of the event. The second memory system, situationally accessible memory (SAM), cannot be accessed deliberately or edited. SAM representations may be accessed automatically when the person is in a context that somehow relates to the traumatic event, whether that context is external (e.g., hearing a sharp clap of thunder) or internal (e.g., experiencing an emotion that was felt at the time of the trauma). SAM memories contain vivid sensory, physiological, and motor aspects of the traumatic event, and these aspects may be involuntarily activated by exposure to trauma-related cues, resulting in the experience of flashbacks and other re-experiencing symptoms.

Brewin et al. (1996) view emotional processing as a largely conscious process in which representations of past and future events are actively manipulated within working memory. They claim that emotional processing of traumatic events requires activation of

SAM representations, which supply sensory details and physiological information about the event, and a conscious search for meaning that ultimately translates the SAM memories into VAM and restores a sense of safety and control. They argue that premature inhibition of emotional processing through avoidance keeps trauma memories locked into SAM, where they continue to be re-experienced. Recovery is achieved when a coherent representation of the trauma and its meaning can be represented, accessed, and manipulated in VAM.

SPAARS model. The Schematic, Propositional, Analogue, and Associative Representational Systems (SPAARS) model (Dalgleish, 2004; Power & Dalgleish, 1997, 1999) was originally formulated as a model of normal, everyday emotional experiences that was later applied to the domain of psychopathology, including PTSD (Dalgleish, 2004). According to this model, mental representation occurs at four levels, each coding a different type of information and representing different working memory spaces where active information can be manipulated. PTSD is related to dysfunction at one or more levels of representation.

The first level of mental representation occurs in the analogical system, which codes and stores information and memories in the form of sensory "images." This system is similar to Brewin et al.'s (1996) SAM system, as it encodes information nonverbally. The output from the analogical processing system then feeds into three representation systems that operate in parallel. The associative representation level represents the connectivity between related images, analogous to the associative networks discussed earlier. At the propositional level of representation, information is encoded in verbal form, similar to the VAM system in dual processing theory (Brewin et al., 1996).

Power and Dalgleish (1999) argue that there is no direct route from propositional representation to emotional experience; instead, propositional representations feed into either the associative representations or are subsumed by appraisals that exist at the schematic model level of representation. At the schematic representational level, information is organized into abstract, general ideas that have emotional import. Schema-based theories of PTSD focus primarily on this level of representation.

Two routes to the generation of emotions exist within the SPAARS model. The first is data-driven or "bottom-up," meaning that the emotion is evoked by the relationship between past experiences and innate emotional responses. This idea relates to Mowrer's (1951) two-factor theory of learning, which assumes that the association between an event and a subsequent emotional response is responsible for learning. The data-driven route mainly relies on associative representations, while the second route, appraisal-driven emotional generation, relies more heavily on the schema level of representation. Appraisal-driven emotional responses are more cognitive in nature and are often related to the individual's goals and values (Dalgleish, 2004). The SPAARS model assumes that at any given time, information is organized, activated, and inhibited as a function of dominant schematic representations and emotional responses, which the authors claim may "hijack the system" to achieve a particular end (Dalgleish, 2004).

PTSD, according to the SPAARS model, develops as a result of the parallel encoding of associative, propositional, analogical, and schematic representations of the traumatic event, which links aspects of the event to each other and to their related emotions (Dalgleish, 2004). In addition to evoking innate emotional responses such as fear and horror, trauma also elicits intense appraisal-driven fear as the goal of personal

survival is threatened. In the aftermath of trauma, processing resources are allocated to an assimilation process whereby trauma-related information is continuously activated, leading to chronic activation of the fear mode (resulting in autonomic hyperarousal) and intrusion of trauma memories into consciousness (re-experiencing symptoms). Furthermore, cognitive processing biases cause trauma-related cues to be selectively processed, which increases the likelihood of trauma-related information stored in memory to become activated and intrude into consciousness.

Sharing many similarities with Brewin et al.'s (1996) dual representation theory, the SPAARS model suggests that recovery from PTSD requires the translation of trauma-related information existing at the analogical level of representation (similar to SAM) into propositional representations (similar to VAM) which can then be assimilated into schematic representations. Until this process is complete, unassimilated trauma memories will retain high levels of internal cohesion, facilitating the re-activation of these memories in the form of flashbacks, nightmares, and intrusive thoughts. The SPAARS model makes few claims about the role of avoidance in the maintenance of other PTSD symptoms, but it can be assumed that cognitive and behavioral avoidance interferes with the process of assimilation, thereby extending the symptomatic period.

Cognitive appraisal model. The final comprehensive model of PTSD combines an information processing approach with a cognitive therapy perspective on the role of negative appraisals and interpretations in maintaining PTSD symptoms. Ehlers and Clark (2000) posited that two key processes are responsible for the development and maintenance of PTSD symptoms: appraisal of the trauma and its sequelae, and the relationship between the trauma memory and other autobiographical memories. They

suggested that negative appraisals of the event and the subsequent cognitive and behavioral reactions to the event produce a sense of current threat and an inability to see the trauma as a time-limited event that does not have global negative implications for the future. In terms of the role of the trauma memory itself, Ehlers and Clark (2000) suggested that high arousal during encoding of trauma memories causes poor elaboration, inadequate integration into autobiographical memory, the creation of very strong stimulus-response associations, and a reduced perceptual threshold for the activation of these memories. These unique characteristics of the memory are responsible for many symptoms of PTSD, including re-experiencing symptoms.

According to the cognitive appraisal model, there is a reciprocal relationship between the nature of the trauma memory and the associated appraisals related to the traumatic event. For example, recall of the trauma is biased by negative appraisals, increasing the likelihood of retrieving appraisal-consistent information and decreasing the retrieval of information that is less compatible with the appraisal. This bias prevents change in the negative, maladaptive appraisals. Also, the "here and now" nature of trauma memories may contribute to the sense of ever-present danger and other negative appraisals of the event. The authors point out that maladaptive behavioral strategies and cognitive processing styles may increase or even directly produce PTSD symptoms (e.g., thought suppression leading to increased intrusive thoughts) and may also prevent change in the negative appraisals that maintain PTSD (e.g., engaging in safety behaviors prevents disconfirmation of the belief that the feared consequence will necessarily occur).

Ehlers and Clark (2000) also suggested that intrusive symptoms might be a result of activation of the trauma memory by relevant cues. They note that the triggers for re-

experiencing events typically have a sensory, not semantic, relationship to the original trauma, and they are experienced as if occurring in the present. Ehlers and Clark (2000) discussed the fact that trauma survivors often experience "affect without recollection," meaning that physiological sensations and emotions associated with the trauma are often re-experienced without conscious recollection of the event. They proposed that there are two routes to the retrieval of autobiographical information, the first being through higher-order, meaning-based strategies and the second being through direct triggering by cues that are physically similar to those present before, during, or immediately after the trauma.

The cognitive appraisal model suggests that in order to recover from PTSD, affected individuals need to elaborate their trauma memories and integrate them into the context of their preceding and subsequent experience. Modification of problematic appraisals of the trauma and post-trauma sequelae must also occur, and dysfunctional coping strategies need to be substituted by positive coping. Ehlers and Clark (2000) outlined a specific treatment approach, which involves brief psychoeducation about the cognitive model of PTSD and then targets negative appraisal through both imaginal exposure with cognitive restructuring and in vivo exposure to trauma reminders.

Summary and contextualization of present study. The theoretical perspectives on PTSD outlined above differ mainly in emphasis and scope; despite these differences, they share many assumptions about the etiology of specific symptom clusters and often advocate for similar treatment strategies. In terms of re-experiencing symptoms, three commonalities emerge from among the viewpoints. First, the idea that intrusive symptoms represent unprocessed trauma-related information that is stored in active

memory, sensory memory, or an analogical representation memory system is shared among the psychoanalytic, schema-based, emotional processing, dual representation, and SPAARS theories. Schema-based cognitive theory and emotional processing theory suggest that this type of storage occurs because the memories are not yet assimilated into preexisting cognitive schemas, whereas dual representation theory and the SPAARS model posit that trauma memories are stored in a raw sensory form until they are translated into linguistic form.

The second common viewpoint on re-experiencing symptoms among various theories of PTSD is that these symptoms often occur as a result of being triggered by the wide range of stimuli that become associated with the fear response during and after a traumatic event. Behavioral, associative network, and cognitive appraisal theories all share this view, though they each emphasize different pathways to activation of the trauma memory. The behavioral view focuses on the development of stimulus-response associations as a result of classical and operant conditioning during and after the trauma. The associative network view discusses "spreading activation" throughout the fear network that causes re-experiencing on account of the strong associations between the various nodes in the fear structure. The cognitive appraisal model posits that cues with sensory, rather than semantic, relationships to actual trauma cues are most likely to trigger re-experiencing episodes, likely due to the meaning associated with those cues.

The third perspective shared by many theoretical models is that re-experiencing symptoms themselves serve a therapeutic purpose and may facilitate emotional processing if allow to run their course (i.e., are not obstructed by cognitive or behavioral avoidance). The treatment recommendations espoused by the majority of the theoretical

models, particularly the four newer models, are in line with this viewpoint. Exposure to trauma-related cues and incorporation of safety information, elaboration of trauma memories and translation of these memories from the sensory domain to the linguistic domain, and cognitive restructuring around the meaning or interpretation of the traumatic event are the main treatment techniques advocated for by one or more of the theoretical models reviewed above. Furthermore, these techniques are at the core of the two most empirically supported treatments for PTSD, PE (Foa et al., 2007) and CPT (Resick, Galovski, et al., 2008; Resick & Schnicke, 1992).

Re-experiencing Symptom Self-monitoring

The intervention technique evaluated in the present study, re-experiencing symptom self-monitoring, represents a logical outgrowth of these summarized views on the nature of intrusive recollections and the role of both exposure techniques and cognitive processing in the treatment of PTSD. Individuals with PTSD are asked to keep a daily record of the frequency, intensity, and content of their nightmares, flashbacks, intrusive thoughts and images, and psychological and physiological reactions to trauma reminders. By encouraging confrontation of trauma memories and impeding cognitive avoidance, it is hypothesized that intrusion monitoring facilitates cognitive and emotional processing, thereby decreasing symptom severity and beginning the process of recovery in the absence of formalized treatment.

Although re-experiencing symptom monitoring is not currently included in any current manualized treatment for PTSD, other applications of self-monitoring are used throughout the course of PE (Foa et al., 2007). Early in treatment, patients are introduced to the Subjective Units of Distress Scale (SUDS), which they use in the remainder of the

sessions to describe and contextualize their subjective experience of anxiety, fear, and other negative emotions. Patients are asked to observe and record their SUDS ratings while engaging in both in vivo and imaginal exposures. By monitoring their subjective distress in this way, patients' attention is directed towards important changes in their emotional state, particularly the decrease in their anxiety that results from repeated exposure to trauma-related stimuli. Put another way, self-monitoring of their internal experience raises patients' awareness of the benefits of resisting avoidance and helps them tolerate their emotional distress as they begin to realize that it will eventually subside.

Intrusion monitoring applies these basic concepts to the domain of habitually avoided distressing cognitions. Patients with PTSD fear re-experiencing the trauma and develop powerful avoidance strategies to keep these symptoms from being triggered. However, just as the use of SUDS monitoring benefits patients by drawing their attention to reductions in their distress and negative emotions, monitoring re-experiencing events allows patients to observe that while these symptoms feel terrifying and overwhelming, they are not associated with any physical danger or harm. This awareness itself may weaken patients' fear of intrusive symptoms, which may translate into greater cognitive and emotional processing of the remembered event. It is for this reason that re-experiencing symptom self-monitoring is proposed in this study as a first step in a treatment regimen; perhaps by weakening the fear response and decreasing cognitive avoidance, the work of integrating the meaning of the event into one's representation of themselves and the world can be completed with greater ease.

Self-monitoring as a therapeutic technique. Self-monitoring, the collection and utilization of information about one's own internal and external states and actions, has been a keystone of treatment for mental and behavioral disorders for decades (Thiele et al., 2002; Wilson & Vitousek, 1999). Currently, self-monitoring is a core component of treatments for conditions ranging from pain disorders and somatic complaints to mood and anxiety disorders, alcohol abuse, and eating disorders. In an influential paper reviewing the impact of self-monitoring on behavior change, Korotitsch and Nelson-Gray (1999) identify key variables that enhance the reactivity of self-monitoring, including greater motivation for behavior change and greater consistency in monitoring each occurrence of the target event. They further suggest that a distinction may be made between two components of self-monitoring, the awareness or detection of the monitored event and the written or verbal recording of the event's occurrence, and they propose as an empirical question whether increased awareness may be enough to increase or decrease the target response or whether the act of recording these responses is mainly responsible for the reactive effects of self-monitoring.

***Therapeutic effects of writing*.** Written recording of target events and responses with the goal of increasing or decreasing these responses in the direction of therapeutic change is but one way in which the act of writing may have a beneficial impact on mental health. The results of numerous studies have supported the hypothesis that writing might encourage emotional processing and disinhibit maladaptive cognitive and emotional avoidance. Specifically regarding writing about traumatic events, Pennebaker and colleagues (Pennebaker, 1993, 1997; Pennebaker & Beall, 1986) have found that the act of writing about emotionally difficult life events is associated with short-term increases in

physiological arousal and negative affect but also associated with improved physical and psychological health. Their explanation for these findings is that self-disclosure via writing may allow for one's feelings and thoughts to become more concrete, which may serve to disinhibit emotional suppression and allow for increased emotional processing.

In a more recent study examining the mental health benefits of writing about a traumatic event, Sloan, Marx, and Epstein (2005) found support for a more specific hypothesis about the mechanisms underlying the efficacy of written disclosure. These researchers proposed that the benefits of writing about a traumatic event may be accounted for by an exposure hypothesis, such that repeated writing about a traumatic event may serve as an in vivo exposure exercise, similar to repeated exposure to a feared stimulus such as snakes or heights. In order to test this hypothesis, they divided participants into three groups: one that was instructed to write about the same traumatic event for three consecutive writing sessions, one that was instructed to write about three different emotionally upsetting experiences, and a third that was asked to write about three events that did not have a negative emotional impact. They found that the repeated disclosure group showed improvement on measures of psychological and physical health, while the non-repeated disclosure group and the control group did not show such improvement (Sloan, Marx, & Epstein, 2005). The results of this study support the hypothesis that repeatedly confronting the memory of a traumatic event may decrease cognitive avoidance and facilitate cognitive processing of the event.

Summary. In light of the theoretical models of PTSD reviewed earlier and the empirical evidence supporting the mental health benefits of putting trauma memories into writing, a daily writing exercise that involves repeated mental confrontation of trauma

memories is likely to result in decreased symptom severity. From a schema-based perspective, confronting and reflecting on traumatic memories facilitates the assimilation of the trauma-related information stored in those memories into pre-existing schemas about oneself and the world. Associative network models suggest that deliberate exposure to the content of trauma memories in a safe environment may weaken the associative links between the memory of the event and the experience of danger and fear. Brewin and colleagues (1996)'s dual representation model posits that the act of writing about trauma memories may aid the translation of SAM memories into VAM, where they can be consciously accessed and manipulated. Similarly, the SPAARS model might see the benefits of writing about intrusive symptoms in terms of increased assimilation across the various levels of mental representation. The present study did not aim to validate one theoretical perspective over any others, but rather sought to apply the wealth of information on the benefits of "avoiding avoidance" to a simple and cost-effective pre-treatment intervention that could be easily disseminated and applied in real-life treatment settings.

Previous research. Re-experiencing symptom self-monitoring has been used with remarkable success in four previous research programs. The first published report of clinical improvement resulting from self-monitoring of intrusive symptoms was a series of case studies of outpatients with PTSD(Reynolds & Tarrier, 1996) that documented significant symptom improvement in six participants who monitored their intrusions daily for a period of two months. Four of these six individuals no longer met criteria for PTSD at the end of the two-month period. The authors suggested that keeping

an intrusion diary forces individuals to confront their intrusive thoughts, thereby reducing cognitive avoidance and initiating cognitive processing.

Encouraged by these findings, Tarrier and colleagues (Tarrier et al., 1999) included a four-week self-monitoring phase as the first step in a randomized controlled trial comparing cognitive therapy to imaginal exposure in the treatment of PTSD in order to identify individuals who might benefit from a low-intensity intervention such that they would not need to continue with the randomized component of the treatment trial. One hundred six participants with chronic PTSD (duration of symptoms ranging from six months to ten years) resulting from a variety of traumatic events kept a daily written diary for four consecutive weeks, documenting the number of nightmares, unwanted thoughts, flashbacks, and unwanted images they experienced in the previous 24 hours. In addition, participants were asked to record when the experiences occurred, what they were about, and how distressing they were. Of the original 106 participants, 12 (11%) no longer met criteria for PTSD at the end of the four-week monitoring period. These individuals continued to show improvement at three-month follow-up. Again, the authors suggested that self-monitoring of re-experiencing symptoms may act as self-directed exposure, decreasing cognitive avoidance and allowing individuals the opportunity to reappraise the meaning of their experiences.

Four years later, a different research group replicated Tarrier et al's (1999) design and instructed survivors of motor vehicle accidents who met criteria for moderate to severe PTSD (scoring 20 or higher on the Posttraumatic Diagnostic Scale) and who enrolled in their clinical trial to engage in daily intrusion monitoring for three weeks prior to randomization (Ehlers et al., 2003). The findings of this study were similarly striking;

12 of the original 97 participants (12%) no longer met the PTSD severity criteria for the study after the three-week self-monitoring phase. These authors additionally reported on overall symptom change across the self-monitoring phase of the intervention. The effect size associated with the decrease in scores on the Posttraumatic Diagnostic Scale was 0.62 (Cohen's d), and all symptom measures showed significant improvement (all $p < .001$).

Although these findings were provocative and potentially meaningful, re-experiencing symptoms self-monitoring received surprisingly little attention in subsequent PTSD treatment research. Two studies (Hardy & Stallard, 2008; Smith et al., 2007) applied intrusion monitoring to the treatment of children showing symptoms of PTSD, and both found remission rates of close to 25 percent in their samples. In addition, Hardy and Stallard (2008) reported that the majority of the children in their sample rated the intrusion monitoring diary as helpful or very helpful. It is important to note, however, than the participants in this study were recruited from a hospital emergency room and had experienced a motor vehicle accident an average of 12.8 days prior to the start of the monitoring period. Participants in the Smith et al. (2007) study were recruited from a trauma clinic and represented a slightly more chronic population (mean of 4.9 months since trauma), but neither was particularly severe in terms of PTSD symptoms.

While the results of the five studies reviewed above strongly suggest that re-experiencing symptom self-monitoring may be an effective and efficient intervention for PTSD, there are notable limitations to the interpretation of their results. First, none of the trials to date has included a no-monitoring control condition against which to compare

the self-monitoring group's level of symptom change. Without such a comparison condition, it is impossible to conclude whether the self-monitoring intervention itself is contributing to the positive outcome, or if more non-specific mechanisms are at play, such as regular contact with treatment providers, increased motivation resulting from acceptance into a clinical trial, or simply the passage of time. Furthermore, the previous intrusion monitoring studies have failed to examine the potential influence of demographic variables such as age, gender, type of trauma experienced (e.g., combat, rape, natural disaster), and years of education. Finally, the data analyses involved in these five prior studies focused mainly on identifying the percentage of participants who no longer met study criteria, rather than assessing overall changes in symptom levels and daily functioning.

Proposed Intervention

The current study was designed to extend the previous intrusion monitoring studies in three key ways. First, a no-monitoring comparison group was used to control for the effects of non-specific factors and symptom decay over time. Second, the study sample was derived from a population of combat veterans enrolled in a specialized PTSD treatment program at a large Veterans Affairs hospital, thereby testing the validity of prior findings in a chronic and severe combat veteran population. Third, a greater number of variables, including demographic variables and symptoms of PTSD-related psychopathology (e.g., depression) were assessed and analyzed in order to examine factors that could potentially moderate the effects of symptom self-monitoring and to assess the overall impact of this type of intervention on daily functioning and mental health.

Hypotheses. Three hypotheses were specifically tested in the current study:

1. Participants who engage in four weeks of intrusion monitoring will show greater reductions in the frequency of and distress associated with avoidance and intrusive symptoms compared with those in the no-monitoring control group.
2. Participants who engage in four weeks of intrusion monitoring will show greater reductions in overall PTSD symptoms and symptoms of related psychopathology compared with those in the no-monitoring control group.
3. There will be a positive correlation between the number of days on which monitoring was completed and improvement in re-experiencing symptoms, overall PTSD symptoms, and symptoms of related psychopathology.

In addition the effect of potential moderators of treatment response will be evaluated. These analyses will be exploratory; no specific a priori hypotheses will be tested.

Methods

Participants

One hundred thirty-seven United States military veterans were enrolled in the study. All participants were veterans of post-Vietnam War military conflicts who were seeking specialized treatment for PTSD through the Trauma Recovery Program (TRP) at a large Veterans Affairs (VA) Medical Center in the southeast. Prior to beginning the study, all participants were diagnosed with PTSD by a mental health professional by means of a semistructured clinical interview. The two exclusion criteria for the study were unwillingness to commit to trauma-focused therapy and presence of a comorbid diagnosis that was likely to interfere with compliance or with the efficacy of treatment, such as drug or alcohol dependence and/or a psychotic disorder that was not well-managed by medication. Veterans meeting diagnostic criteria for such disorders were asked to address these issues prior to being accepted into the TRP. The study was reviewed and approved by the hospital's Institutional Review Board.

Research Design

The study took place in the context of a comprehensive treatment program at the VA hospital, which included an initial five-week psychoeducation group known as "PTSD 101." PTSD 101 had been developed to help bridge the gap between initial diagnosis and treatment because the waiting period for specialized PTSD treatment (PE or CPT) was typically four to six weeks, during which time veterans received little support and had minimal contact with treatment providers. At the time of this study, veterans presenting for specialized treatment for PTSD were given a comprehensive diagnostic assessment with a TRP clinician. Then, if PTSD was diagnosed as the primary

target for treatment, veterans were given the choice between enrolling in a PTSD 101 class while they waited for individual exposure-based treatment or simply waiting until the individual therapist became available. Interested veterans selected a class based on the match between the class schedule and their availability.

For purposes of this study, PTSD 101 groups were randomly assigned active or control status in order to control for possible confounding variables such as time of year and clinician characteristics. In anticipation of low rates of compliance with the intrusion monitoring, more active than control groups were assigned to insure an adequate number of self-monitoring participants. Active group participants were asked to complete the daily re-experiencing self-monitoring forms described below throughout the duration of their PTSD 101 class. The control participants engaged in "treatment as usual," completing the class without the addition of the intrusion monitoring component. The active and control groups completed identical questionnaire packets at the same time points, received equal clinician contact time, experienced the same general class format, and were exposed to the same content in each class, with the minor exception of the active group also receiving a brief overview of the intrusion monitoring procedure at the end of the first class. Veterans were not aware of the active (versus control) status of a class prior to selecting a start date.

Procedure

Veterans were asked to arrive 15-30 minutes early to the initial session of PTSD 101 in order to complete a pre-treatment assessment packet, which they turned in to the group leader before the end of the session. Week 1 of PTSD 101 is designed as an introduction to PTSD and includes a discussion of the symptoms of PTSD, common

reactions to trauma, and the role of environmental triggers in the expression of PTSD symptoms. Appendix A includes the full set of PTSD 101 handouts common to both conditions. In the active condition, class leaders also read a brief description of the intrusion monitoring procedure at the end of the first class meeting (cf. Appendix B). A packet of seven self-monitoring forms, an instruction sheet, and a sample monitoring form (cf. Appendix B) were then handed out to class members and questions and concerns were addressed. Participants were asked to set aside a time every day to complete one form and to bring the packet of forms back with them to the next class. At the start of subsequent class sessions, completed homework packets were collected and participants were praised for their efforts. New blank packets were handed out before the end of each class session.

Measures

Participants in both the active and control conditions completed a questionnaire packet that included all the measures listed below at the beginning of the first class and at the end of the last class. Participants also completed some of the questionnaires at the beginning of classes two, three, and four.

Demographic questionnaire. All participants completed a demographic questionnaire at the Week 1 assessment point. This measure asks participants to report demographic information, including age, gender, military branch affiliation, marital status, years of education, employment status, number of war zones served in, and traumatic brain injury diagnosis.

PTSD Symptom Scale – Self-report (PSS-SR). The 17 items of the PSS-SR (Foa, Riggs, Dancu, & Rothbaum, 1993) assess the frequency with which each of the DSM-IV

diagnostic indicators of PTSD have been experienced in the previous week. The PSS-SR has three subscales and a total score. The subscales assess different clusters of PTSD symptoms corresponding to categories set forth in the diagnostic criteria for PTSD (cf. Table 1): intrusive/re-experiencing symptoms (PSS Intrusive, 5 items), avoidance/numbing symptoms (PSS Avoidance, 7 items), and hyperarousal symptoms (PSS Hyperarousal, 5 items). Each item is rated on a four-point scale ranging from zero ("not at all") to three ("more than four times"). Total scores range from zero to 51, with higher scores indicating greater symptom severity. Reliability and validity analyses have indicated that the PSS-SR has satisfactory internal consistency, high test-retest reliability, and good concurrent validity (Foa et al., 1993).

PTSD Checklist – Civilian version (PCL-C). The PCL-C (Blanchard, Jones-Alexander, Buckley, & Forneris, 1996) is a 17-item self-report measure assessing subjective distress associated with PTSD symptoms. The 17 items are nearly identical to those on the PSS-SR, but whereas the PSS-SR assesses symptom frequency, the PCL-C asks raters to indicate how much they were bothered by each symptom over the past week. Again, subscales exist for each cluster of symptoms: intrusive (PCL Intrusive, 5 items), avoidance (PCL avoidance, 7 items), and hyperarousal (PCL Hyperarousal, 5 items). Items are rated from one ("not at all") to five ("extremely"), with possible total scores ranging from 17 to 85.

Beck Depression Inventory – II (BDI-II). The BDI-II is a 21-item self-report measure designed to assess symptoms of depression in adults and adolescents (Beck, Steer, & Brown, 1996, as cited in Beck, Steer, Ball, & Ranieri, 1996). The items are rated on a four-point scale ranging from zero to three. The total score can range from

zero to 63, with scores below 13 indicating minimal depression and scores above 29 indicating severe depression. The internal consistency of the BDI-II is high, and the test-retest reliability is also good (Beck et al., 1996). In addition, the BDI has shown good convergent and discriminant validity (e.g., Krefetz, Steer, Gulab, & Beck, 2002; Storch, Roberti, & Roth, 2004).

Anxiety Sensitivity Index-3 (ASI-3). The ASI-3 (Taylor et al., 2007) is an 18-item measure designed to assess fear of arousal-related sensations. The measure includes items such as, "It scares me when my heart beats rapidly." Respondents are asked to indicate the degree to which they agree with the statements, which they indicate on a scale ranging from zero ("very little") to four ("very much"). The ASI-3 is divided into three subscales representing physical, cognitive, and social concerns. Each subscale is comprised of six items. The ASI-3 has demonstrated strong reliability and validity in large, diverse samples (Taylor et al., 2007).

Posttraumatic cognitions inventory (PTCI). The 36-item PTCI (Foa, Ehlers, Clark, Tolin, & Orsillo, 1999) is designed to assess individuals' belief in trauma-related cognitions, such as, "I have to be on guard all the time." Respondents rate the items on a seven-point scale ranging from "totally disagree" to "totally agree." Three subscales have been identified: negative cognitions about self, negative cognitions about the world, and self-blame. The scale and its three subscales have all shown high internal consistency and test-retest reliability (van Emmerik, Schoorl, Emmelkamp, & Kamphuis, 2006). In addition, total scale and subscale scores have been found to significantly correlate with scores on a PTSD diagnostic scale, indicating convergent validity, and

patients with PTSD score significantly higher than those without PTSD, suggesting that discriminant validity is high as well (van Emmerik et al., 2006).

Data Analysis

Data were analyzed using IBM SPSS Statistics Version 19. Baseline differences between the groups were analyzed using independent samples t-tests or chi square analyses, depending on the nature of the data. Repeated Measures Analyses of Variance (ANOVA) was used to analyze the effect the independent variable (e.g., monitoring vs. no-monitoring condition), the within-subjects effect of time, and the interaction between group assignment and time on each outcome variable. Repeated Measures Analysis of Covariance (ANCOVA) was used to assess the impact of demographic moderators.

Results

Participant Characteristics

Demographic and clinical features of the 137 enrolled participants are presented in Tables2 and 3. The typical participant was a male (*n* = 121; 88.3%), African-American (*n* = 103; 75.2%), married (*n* = 73; 53.3%), unemployed (*n* = 48; 35.0%) Army veteran (*n* = 102; 74.5%). There were no significant differences between the active and control groups on any demographic or clinical variables at baseline.

Eighty-eight of the 137 enrolled veterans were considered study completers. The criteria for this designation were attendance and completion of assessments at Week 1 and Week 5 and attendance at at least one other session of the PTSD 101 group. Of the 87 veterans initially randomized to the active condition, 33 (37.9%) did not complete the study. A comparable 32% of the control group (16 of 50) also failed to complete. Demographic and clinical features of the 88 completers are presented in Tables4and 5. Independent samples t-tests using completers' data only revealed no differences between the active and control groups at baseline. Independent samples t-tests also indicated that there were no differences in demographic variables or symptom scores at baseline between completers and dropouts. The remaining analyses were conducted using the completers' data only.

Preliminary Analyses: Active Group vs. Control Group

Change in avoidance symptoms. The hypothesis that individuals who engaged in re-experiencing symptom self-monitoring would show greater reduction in avoidance symptoms compared with those in the no-monitoring control group was not supported. Table 6 presents the results of the statistical analyses. No significant interactions

between active group and time were found on the avoidance subscales assessing frequency of avoidance (PSS Avoidance) or distress associated avoidance(PCL Avoidance). A significant main effect of time was found on the PCL-C avoidance scale ($F(1, 84) = 6.96$, $p = .010$). As Figure 1 displays, both groups decreased in their levels of distress associated with avoidance symptoms over the course of the study.

Change in re-experiencing symptoms. The hypothesis that four weeks of intrusion monitoring would lead to greater reduction in intrusive symptoms compared with no monitoring was not supported. Repeated measures ANOVAs were conducted to compare the relative change in the active and control groups' frequency of intrusive symptoms (PSS Intrusive) and distress associated with intrusive symptoms (PCL Intrusive). No significant main effects or interactions were found. Table 6 presents these findings.

Change in overall PTSD symptoms. The hypothesis that intrusion monitoring would lead to greater reduction in overall PTSD symptoms was also not supported. Repeated measures ANOVAs indicated no significant interactions between group and time on the PSS-SR and PCL-C total scores. A significant main effect of time was found on the PCL-C total score ($F(1,84)=4.75$, $p= .032$), which is best explained by the significant decrease in distress related to avoidance symptoms (cf. Figure 1). Table 6 displays these results.

Change in related pathology. The hypothesis that intrusion monitoring would lead to greater reduction in symptoms of depression, anxiety sensitivity, and maladaptive cognitions related to trauma compared with the no-monitoring control condition was not

supported. No significant main effects or interactions were found with the Repeated Measures ANOVAs. Table 6 displays these results.

Demographic Moderators

To test the impact of demographic variables on changes in symptoms of PTSD and related psychopathology, Repeated Measures Analyses of Covariance (ANCOVAs) were carried out, with each of the demographic variables entered as covariates. The impact of moderators on total scores was examined first, with follow-up analyses conducted using subscale scores when a significant interaction was found. The results indicated that participants' age and the number of war zones in which they served moderated the relationship between monitoring status and change in frequency of PTSD symptoms (PSS-SR), and age moderated the relationship between monitoring group and change in distress related to PTSD symptoms (PCL-C). Table 7 displays the three-way interaction terms for the total score moderator analyses. Table 8 shows the results of the follow-up analyses; these analyses indicated that the moderating effect of age and number of war zones on changes in frequency of PTSD symptoms was driven by changes in frequency of avoidance symptoms alone. The impact of age on distress was found for both avoidance and intrusive symptoms. Neither variable affected hyperarousal symptoms.

Chi-square tests indicated that there was a significant relationship between age and number of war zones served in, such that service in one war zone was overrepresented in younger patients and underrepresented in older patients (*Pearson chi-square* = 12.41, $p = .002$; cf. Table 9). This relationship is to be expected, given that soldiers in the younger age category tended to be newer to the military and were not yet

of age during the first Iraq war, one of the potential areas of service among the veterans treated by the TRP Acute Team. Since age and war zones were significantly related and age affected both avoidance and intrusive symptoms, only age was used to report the directionality of the interactions. Participants were classified into three age groups to create groups of roughly equal size: 30 and under (n = 27), 31 to 44 (n = 34), and 45 and over (n = 27).

Repeated Measures ANOVAs separated by age group showed that while there was no significant interaction between active status and time among individuals in the middle and older age categories, a significant interaction between active status and time was detected among younger individuals on frequency of avoidance symptoms. The interaction between group and time showed a trend toward significance in the younger age category when examining changes in distress associated with intrusive and avoidance symptoms. Table 10 displays these results. As Figures 2, 3, and 4 portray, younger participants in the active group showed a decrease in these symptoms from baseline to final assessment, whereas younger control group members' scores tended to increase or remain the same.

Monitoring Compliance

Compliance with the self-monitoring intervention was highly variable. Thus, exploratory analyses were completed to explore the possibility that participants who monitored more often might have shown greater benefit from the intervention. The mean number of days on which monitoring was completed was 10.9 (of 28 possible days), and the median was 7.5 days. No significant correlations were found between cumulative monitoring days and pretest measures, nor were correlations found between cumulative

monitoring days and pre-post symptom change (cf. Tables 11 and 12).Figure 5 depicts the frequencies of cumulative monitoring days. The active group was classified by monitoring compliance into low (*n* =21, 0-6 days), moderate (*n* =19, 7-20 days) and high (*n* =14, 21-28 days) compliance groups for the purposes of further data analysis. As Table 13 shows, the group by time interaction was only significant for PTCI Self ($F(3, 73) = 2.83$, $p = .045$). The moderate compliance group (7-20 days of monitoring) reported an increase in negative cognitions about themselves over the course of the study, while the control group, low compliance group, and high compliance group showed little change on this measure (cf. Figure 6).

To assess whether monitoring compliance interacted with age to impact the frequency of avoidance symptoms in response to the self-monitoring intervention, post hoc Repeated Measures ANOVAs were conducted with monitoring compliance level as the independent variable, PSS Avoidance as the dependent variable, and age entered as a covariate. The results showed a significant three-way interaction (age by monitoring group by time; $F(3, 80) = 2.72$, $p = .05$). Follow-up analyses indicated that the interaction occurred within the younger age group (cf. Table 14); Figure 7 displays the pattern of this interaction, which follows that seen in the active vs. control analyses (cf. Figure 2). The younger controls increased the frequency of their avoidance over time, whereas the younger veterans in the active condition showed at least minimal improvement in avoidance, with the moderate compliance group being intermediate in outcome.

Discussion

Main Findings

At the broadest level of analysis, four weeks of daily written recording of the frequency, intensity, and content of re-experiencing symptoms did not significantly impact symptom improvement in a population of American military veterans with chronic and severe combat-related PTSD. No significant differences were found between the active and control groups in terms of changes in avoidance or re-experiencing symptoms specifically, PTSD symptoms generally, or symptoms of related psychopathology over the course of the five-week study. Frequency of PTSD symptoms and levels of associated distress generally remained stable throughout the study period. The only construct showing significant pre-post change was the total score of the PCL-C, a measure of distress related to PTSD symptoms across all three symptom domains. This significant decrease in overall PTSD-related distress was likely due to the normalizing influence of being in a class setting with other veterans sharing the diagnosis of PTSD and receiving psychoeducational material about the development and maintenance of PTSD symptoms.

These results contrast with previous uncontrolled studies involving patients with less chronic and severe PTSD (Ehlers et al., 2003; Hardy & Stallard, 2008; Smith et al., 2007; Tarrier, Sommerfield, Reynolds, & Pilgrim, 1999) in which a substantial percentage of participants no longer met study criteria for PTSD after engaging in intrusion monitoring for three to four weeks. Due to the lack of a control group in these previous studies, it is not possible to rule out the effect of time on the observed decreases in PTSD symptoms. The majority of individuals exposed to a traumatic event recover naturally within the first three months post-trauma (Resick et al., 2008), so the fact that

the majority of the participants in previous intrusion monitoring studies had been exposed to trauma within the year prior to the start of the study, if not more recently, is a complicating factor when interpreting the substantial decreases in symptoms found in these uncontrolled trials.

On a more detailed level of analysis in the current study, statistically significant and meaningful findings emerged. Both age and number of war zones served in were found to be significant moderators of the relationship between group assignment and pre-to-post change in the frequency of avoidance symptoms (PSS Avoidance). Age was also found to moderate changes in distress related to intrusive and avoidance symptoms (PCL Intrusive and PCL Avoidance). Follow-up analyses showed that while older veterans did not show differential response on these measures based on active group status, significant or marginally significant group-by-time interactions were found in the younger age group (30 and under). On all three outcome measures, younger veterans in the active group tended to show a decrease in these symptoms, whereas age-matched controls tended to increase or show no change on these measures from baseline to final assessment.

Compliance with the intrusion monitoring assignments was highly variable and, on average, less than 50% (mean 10.9 of 28 days, median 7.5 of 28 days). No correlations were found between cumulative monitoring days and either baseline symptom scores or changes in symptoms from baseline to final assessment, and no differences in demographic characteristics or baseline symptoms were found among those who monitored at a high, moderate, and low rate, suggesting that neither symptom severity nor demographic variables such as age significantly influenced compliance rates. Only one statistically significant relationship was found between compliance and

symptom change; the moderate compliance group (7-20 days of monitoring) reported a greater increase in negative cognitions about themselves over the course of the study compared to the low and high compliance groups and to the no-monitoring control group.

Acceptability and feasibility. To assess veterans' subjective reactions to the self-monitoring intervention, optional feedback forms were handed out to active group participants during the last class session of PTSD 101. Unfortunately, only 12 of the 54 active group completers turned in the form. One might assume that any participant who had a strong negative reaction to the intervention would have been motivated to report on their experience. In contrast, nearly all(n = 10) indicated that they had found the exercise helpful. Only two veterans stated that it may have been harmful because it was "discomforting" and "shed light on troubling thoughts and memories." A few participants specifically noted that the intervention felt "safe" because they were only asked to document what they were already experiencing. Common reasons listed for not completing the forms were forgetfulness, lack of time, and reluctance to think about the content of distressing memories. Another noteworthy finding from these informal feedback forms was that many of the veterans reported a higher compliance rate than was documented by study clinicians based on the number of monitoring worksheets actually turned in. It is possible, in light of this discrepancy, that compliance may have been somewhat higher than it appeared; some veterans may have completed the monitoring forms but failed to turn them in due to absence from class or forgetting the forms at home. Overall, the data collected from these feedback forms suggest that intrusion monitoring was generally viewed as a safe and acceptable intervention strategy.

Interpretation and Theoretical Implications

The lack of significant differences in outcome between the active and control groups runs counter to the results that were expected based on the current theoretical models of the development and maintenance of PTSD. These theories posit that beneficial cognitive and emotional processing of a traumatic event occurs when individuals are exposed to trauma memories in a safe environment, which allows them to incorporate safety information into the cognitive schemas or information processing networks that dictate how they perceive themselves and the world around them (Brewin et al., 1996; Dalgleish, 2004; Ehlers & Clark, 2000; Foa et al., 2007). Exposure to trauma-related re-experiencing events by engaging in intrusion monitoring was expected to facilitate such processing; however, the lack of significant improvement in the active group suggests that this goal may not have been achieved, or if processing did occur, it did not translate into notable symptom improvement.

One hypothesis to explain why cognitive and emotional processing may have been limited in the present study is that participants may have been able to utilize cognitive avoidance techniques despite completing the forms as prescribed. Participants were asked to write only a brief description of the re-experiencing events that occurred throughout the day and were not specifically instructed or encouraged to reflect for long on the content of the events or to write about them in great detail. Therefore, the descriptions tended to be quite general and vague (e.g., “I felt like I was back in Iraq”), suggesting that the traumatic event underlying the intrusive event may not have been fully brought to conscious awareness. Furthermore, the brevity of the descriptions suggests that the trauma-related images may not have been fully translated into the verbal domain, a process that both Brewin et al. (1996) and Dalgleish (2004) identify as a key

factor in recovery from PTSD. Keane et al. (1985) and Foa and Kozak (1986) also note that exposures that are too brief do not facilitate recovery, and they may in fact exacerbate symptoms in some cases.

The instruction that the monitoring forms be completed at a single time each day also may have curtailed cognitive and emotional processing. The re-experiencing events could have been avoided successfully at the time they occurred and only superficially acknowledged later in the day while filling out the form. Similarly, the veterans could have easily forgotten to mention significant re-experiencing events or could have deliberately chosen not to include them on the monitoring form because of the affective intensity associated with their content. As intrusive experiences are by their nature internal and unobservable by others, it is impossible to compare the reported events to the actual events that had occurred throughout the day.

A key component of some theories of emotional processing (e.g., Keane et al., 1985; Foa & Kozak, 1986) is the notion of "affect matching," whereby processing is facilitated when the affect evoked by the trauma memory matches that experienced at the time of the trauma. While study participants were asked to rate the level of distress they felt at the time of the re-experiencing events they reported, this reported distress is not likely to be a good proxy for affect matching. Again, avoidance strategies may have been used to dampen emotional responses at the time of the re-experiencing event, and this reduction in affect would have interfered with the emotional processing that intrusion monitoring was designed to facilitate.

The second hypothesis that reconciles the results of the present study with the current theoretical models of PTSD is that intrusion monitoring did allow cognitive and

emotional processing to occur, but this processing did not translate into detectable symptom reduction in the active group. It is possible that a substantial amount of processing could not be completed in four weeks' time, particularly in the chronic and severe patient population involved in this study. Changes in symptoms might have been detected if the study had extended for a longer period of time or if participants had continued to be evaluated after the self-monitoring period had ended. A number of researchers and theorists have pointed out that incomplete exposures, including those that are too brief in duration, can exacerbate symptoms (Foa & Kozak, 1986, Foa et al., 2007). Also, an increase in symptom severity is commonly observed at the start of exposure treatments (Foa et al., 2007). Both the relatively short data collection window and the possibility of symptom activation due to the nature of the self-monitoring task make it difficult to know whether active participants may have been on their way to showing improvements had the study continued for a longer period of time, or if symptom activation may have counterbalanced symptom improvement among the individuals in the active group. Four weeks of low-level cognitive exposures may lead to symptom resolution in less severe cases, but among individuals for whom avoidance has become a way of life, a more prolonged period of intrusion monitoring might be necessary to show effects.

Several possible explanations may be considered in understanding why younger veterans may have responded to the intervention to a greater degree than older veterans. First, it is likely that younger veterans had a shorter duration of illness at the time of enrollment in the study compared with older participants. This variable was not explicitly measured in the current study but seems highly likely given age differences.

With a shorter time since the traumatic event, trauma memories may be somewhat more accessible and thus more amenable to processing. Also, patterns of cognitive avoidance may have had less time to develop and become entrenched in patients more recently exposed to a traumatic event.

This hypothesis is partially supported by the literature on intrusion monitoring, in that the participants in previous studies tended to have shorter durations of illness than the typical VA patient. For example, in Tarrier and colleagues' (1999) study, one third of participants had experienced their index trauma less than one year prior to the start of the study, and only 23% experienced the trauma more than three years prior. Ehlers et al. (2003) required that the intervention start within six months of the trauma, and Smith et al. (2007) began their intervention an average of 4.9 months after the trauma occurred. Hardy and Stallard's (2008) participants had the shortest time since the trauma, an average of 12.8 days. Given the age-related findings from the current study and the inclusion of more recently traumatized individuals in previous studies with positive findings, re-experiencing symptom self-monitoring might be better considered as a treatment option for those with Acute Stress Disorder (ASD) or as an early intervention for those exposed to trauma but not yet showing a clear pattern of symptoms, rather than as a treatment for chronic PTSD. Follow-up studies are needed to further examine this application of intrusion monitoring.

Another hypothesis for why younger veterans tended to show a greater response to the intervention is that younger veterans may have been exposed to fewer traumatic events compared with older veterans. It is important to note that the number of war zones in which veterans had served was significantly correlated with age, suggesting that the

older veterans may have been exposed to a higher number of combat-related traumas. Exposure to multiple traumatic events may have limited the impact of the intervention, as the re-experiencing events described on the self-monitoring forms of veterans with multiple traumas may have varied widely, weakening the impact of the emotional processing through repetitive exposure. In contrast, an individual who had experienced only one traumatic event or multiple events within a single contextual field would be expected to report intrusive symptoms related to that context. If the monitoring only focused on a single memory or context, this may have strengthened the effects of the intervention by continually exposing the individual to memories of one event or events that shared a similar context.

One previous study (Tarrier et al., 1999) had found that individuals with less severe PTSD symptoms at baseline tended to show a stronger response to the self-monitoring intervention compared with participants with more severe symptoms. However, this finding was not replicated in the current study, and it is important to note that participants' age was not significantly correlated with symptom severity (all Pearson's r values $< .20$). Therefore, the differential response of the younger veterans in this study cannot be attributed to lower symptom severity at baseline.

It is also notable that not only did the younger participants in the active group show a greater response to the intervention compared with older participants, but also symptoms tended to worsen over the course of the study among younger veterans in the control group, whereas those in the active group showed improvement. This finding suggests that the improvement in intrusive and avoidance symptoms among younger veterans was likely due to engagement in the intrusion monitoring task rather than

attributable to some static, baseline characteristic of younger veterans with PTSD. This finding might suggest that the developmental trajectory of PTSD was following a course of worsening symptoms among the younger veterans; without an intervention specifically designed to break the cycle of cognitive avoidance that perpetuates PTSD, these veterans might have become more reliant on avoidance as a coping strategy during the study period. In contrast, those who engaged in intrusion monitoring may have been able to successfully break that negative cycle and achieve some degree of symptom relief. Clearly, there is room for further investigation on this point.

Compliance with the intrusion monitoring assignment did not generally impact changes in symptoms from baseline to final assessment. This finding is surprising and inconsistent with the theory behind using self-monitoring of re-experiencing symptoms as "mini exposures" to trauma memories, as the theory would suggest that greater repetition of these exposures would lead to greater symptom reduction. Only one significant difference among the monitoring groups emerged: individuals with moderate compliance (7-20 of 28 days) showed greater increases in negative trauma-related cognitions about themselves. This finding suggests that those who approached the monitoring task but were inconsistent in completing it may have experienced an increase in self-critical and self-blaming thoughts about the trauma as a result of "opening their minds" to the reality of the event but not reflecting on it frequently enough for cognitive and emotional processing to occur. Those who engaged in little to no monitoring may not have had such symptoms activated in the first place, whereas those who completed the monitoring assignment more regularly may have experienced an initial increase in self-critical thoughts that then resolved as they continued the monitoring. High self-blame and low

self-compassion are baseline characteristics that could be assessed in future studies, as they might impact compliance with monitoring as well as symptom reduction.

One three-way interaction between age group, monitoring compliance, and time was found on the measure of frequency of avoidance symptoms. Post hoc analyses showed that these results followed a similar pattern to those seen in the earlier analysis; among younger veterans, participants in the control group (no monitoring) tended to show an increase in avoidance symptoms over time, whereas all three monitoring compliance groups tended to improve on this construct, with the moderate compliance group showing the least substantial changes.

Although compliance with the self-monitoring assignment did not have a large impact on overall outcome in this study, the low rate of compliance is important to consider when interpreting the findings, as the impact of the intervention may have been obscured or weakened by the inconsistent participation of the active group members. One positive perspective on the low compliance rates observed in this study is that low compliance suggests that exposure to re-experiencing symptoms "hits home" for those struggling with PTSD. Exposure-based interventions such as intrusion monitoring often evoke initial anxiety and distress as the underlying fear structures are activated (Foa & Kozak, 1986), and individuals often need considerable encouragement and support to persevere beyond this initial discomfort in order to fully benefit.

Self-monitoring as an intervention technique. The findings from the present study also have implications for the theories supporting the use of written self-monitoring as an intervention strategy in the treatment of mental health problems. Korotitsch and Nelson-Gray (1999) identified, on the basis of empirical evidence, the following eight

variables that affect the reactivity of self-monitoring: target behavior valence; overt or covert nature of the target behavior; motivation of the individual engaged in monitoring; schedule of recording; number of behaviors monitored concurrently; timing of recording; goal setting, feedback, and reinforcement; and obtrusiveness of the self-recording device. They proposed that to optimize the impact of self-monitoring, individuals should be highly motivated to change the behavior they are monitoring, should monitor each instance of a single target behavior that is overt or motoric in nature, should make recordings using an obtrusive recording device just before the occurrence of this target behavior, and should have a clear goal for change and receive reinforcement contingent on behavior change in the direction of this goal. They also present evidence suggesting that behaviors with a positive valence increase in frequency with repeated monitoring, whereas monitoring causes a decrease in behaviors with a negative valence.

In many ways, intrusion monitoring conforms to Korotitsch and Nelson-Gray's (1999) theory-based recommendations. In monitoring re-experiencing symptoms, the goal may be best formulated as, "reflect on and record every re-experiencing event that occurs." From this perspective, the target behavior includes both a covert, cognitive component and an overt, motoric component. As patients are encouraged by clinical staff to reflect and record in response to every re-experiencing event, the target behavior takes on a positive valence, theoretically leading to an increase in the frequency of reflection and recording over time. On the other hand, not reflecting on and recording intrusive symptoms (i.e., avoidance) takes on a negative valence, which, according to the empirical evidence summarized by Korotitsch and Nelson-Gray (1999), ought to cause a decrease in avoidance with repeated self-monitoring.

Korotitsch and Nelson-Gray (1999) recommend recording just before the target event occurs; as intrusion monitoring is done in response to a re-experiencing event, this recommendation may not apply. However, as people become better able to identify situational and emotional triggers, they may be able to preemptively record the content of re-experiencing events typically triggered by certain trauma-related stimuli, which ought to enhance the impact of the self-monitoring intervention. Ideally, individuals would engage in self-monitoring each time they experience an intrusive symptom in order to keep a singular focus on recording the content of a single event. The intervention might be further enhanced by the use of hand-held electronic devices that would allow patients to record each event as it occurs rather than at the end of the day. It is notable that problem areas such as eating where self-monitoring shows strong and reliable effects, the instruction emphasizes recording at the time of eating rather than at the end of the day.

Motivation to change the target behavior is the final aspect of Korotitsch and Nelson-Gray's (1999) theory supporting the use of self-monitoring as an intervention strategy. This aspect of the theory may help explain why the results of the current study did not clearly favor the application of re-experiencing self-monitoring to the treatment of veterans with PTSD. Individuals with PTSD are rarely motivated to reflect on the traumatic events that haunt them because of the anxiety and distress they experience when memories of these events are triggered. Cognitive and behavioral avoidance strategies are highly negatively reinforcing; that is, they stop or prevent the distress associated with trauma memories. Under ideal circumstances in which an individual is able to easily "avoid avoidance," intrusion monitoring is likely to be a highly effective intervention strategy. However, given the role of avoidance in the development and

maintenance of PTSD, it is highly unlikely that anyone carrying this diagnosis is able to stop using cognitive avoidance strategies with ease. Extra effort may be needed to help individuals work toward the goal of resisting the temptation to avoid and instead engaging in self-motivated reflection and recording of re-experiencing events.

Clinical Implications

The findings of the current study tentatively suggest that re-experiencing symptom self-monitoring may be a useful preliminary treatment strategy among individuals with a short to moderate duration of illness, briefer time since trauma exposure, and/or exposure to a single traumatic event or single trauma context. It must be stated clearly that the meaning associated with the moderating influence of age on outcome can only be surmised, not empirically validated, on the basis of data collected in this study. Nonetheless, if younger age is assumed to be a proxy for illness duration, time since trauma, and/or number of traumatic events, some important implications for treatment emerge.

Re-experiencing symptom self-monitoring may be most useful as an early intervention strategy for those recently exposed to a traumatic event. Exposure-based interventions, including PE, are considered the most efficacious for the treatment of ASD, the diagnosis given to individuals showing PTSD-like symptoms in the first month after experiencing a traumatic event (Bryant, Harvey, Dang, Sackville, & Basten, 1998; Ponniah & Hollon, 2009). Furthermore, Bryant and colleagues (Bryant et al., 2008) found that exposure techniques conferred more benefit on those with ASD than cognitive restructuring, suggesting that a primarily exposure-based strategy like intrusion monitoring may be particularly effective in the treatment of ASD.

Another area of empirical support for the application of intrusion monitoring soon after exposure to a traumatic event is the evidence suggesting that high levels of avoidance in the early stages of illness predict poorer outcome. Creamer et al. (1992) noted that those who responded to re-experiencing events early in the course of illness by engaging in avoidance behaviors had higher symptom scores at later data collection points in the study than those who had lower avoidance scores early on. In addition, O'Donnell, Elliott, Lau, and Creamer (2007) found that higher levels of avoidance at baseline and increases in avoidance symptoms over time both predicted the development of PTSD in a sample of individuals with ASD. These findings suggest that an early intervention targeting cognitive avoidance may be essential and impactful as a means of preventing the future development of PTSD. Re-experiencing symptom self-monitoring has the added benefit of being a low-cost, easily disseminated, and individually-tailored intervention strategy.

The data also offered some indication that engaging in a moderate amount of monitoring (defined in this study as completing monitoring forms on 7-20 of 28 days) may have had a deleterious effect on trauma-related negative cognitions. The relationship between monitoring compliance and negative cognitions about oneself is unclear at this point, but it is possible that engaging in intrusion monitoring "only halfway" may exacerbate negative thoughts about the hostility of the world and one's ability to cope with danger and threat. On the basis of this result, a possible recommendation for future applications of intrusion monitoring might be to assess motivation to comply with daily monitoring prior to assigning the task, and only ask those with high motivation to engage in monitoring. Furthermore, assessing compliance

after one week of monitoring and withdrawing the prescription to monitor from those who seem unable to maintain regular compliance might prevent the observed exacerbation of negative cognitions in those who engage in intrusion monitoring only part of the time.

Strengths

The strengths of the current study were its compelling theoretical foundation, the rigor of the study design, and the involvement of a clinical sample seeking treatment in a naturalistic setting. The study grew out of a well-validated theoretical understanding of the factors that contribute to the development and maintenance of PTSD, in combination with direct observation of the need to develop cost-effective and efficient treatment strategies for this patient population. Had the results supported the beneficial effects of re-experiencing symptom self-monitoring across all patient subgroups, the implications for treatment would have been substantial. Given the complexity of the findings that younger veterans showed more substantial benefit, more work is needed to determine if the benefit for some veterans would make it worthwhile to provide intrusion monitoring as a pre-treatment intervention option. Being asked to engage in intrusion monitoring did not appear to be associated with deterioration in this study. Those who were not willing to complete the monitoring assignments simply did not comply. The study was designed to submit previous research findings to rigorous evaluation by including a no-monitoring treatment-as-usual control condition and using a quasi-random group assignment strategy.

Limitations

The main limitations of the current study were the possible obscuring effects of the various components of PTSD 101 group that were concurrent with the study intervention, the short duration of the study, the exclusive reliance on self-report measures, and the lack of control over clinical changes (e.g., medication changes) during the study period. The first limitation is perhaps the most important; PTSD 101 is a fairly intensive, eight-hour educational experience that introduces veterans to the key concepts related to the development, maintenance, and treatment of PTSD. In addition to receiving information through in-class interactive lectures, PTSD 101 class members are taught and asked to practice daily breathing retraining exercises, encouraged to keep a daily log of positive activities related to sleep hygiene, and instructed to engage in self-directed in vivo exposures at least three to four times a week (cf. Appendix A). The active group in the current study was given the intrusion monitoring exercises as an additional homework assignment starting in the first week of class. These multiple routes of intervention may have added too high a burden for study participants, many of whom arrived to class completely naive to mental health treatment. It is also possible that one or more of the other intervention strategies exacerbated symptoms among active group participants, thereby counteracting the positive influence of the intrusion monitoring. While the use of a treatment-as-usual control condition should still be considered a strength of the study design, it is possible that the intrusion monitoring may have had a greater impact if it were the only intervention strategy being employed.

The second limitation of the study, the relatively brief duration of the intervention, was also a deliberate choice based on the previous studies employing re-experiencing symptom self-monitoring. No study had included a monitoring period

longer than four weeks, and all had seen positive results in that short period of time. However, the population used in the current study was substantially more chronic and severe than those used in the four previous studies. It is possible that in a population such as this, a longer period of monitoring is required before significant changes can be observed. This issue is also complicated by the overall low rate of compliance; had the participants completed the monitoring as prescribed, more significant changes might have been observed within the four-week study period.

All data used in the current study was collected via self-report assessments that relied on retrospective appraisal of thoughts, feelings, and behaviors. This method of data collection poses threats to validity and reliability, as memory is known to be fallible and error-prone (Schacter, 1999). Given this limitation, it is important to note that the variables of interest in this study were primarily related to internal, subjective states (e.g., anxiety, depression, nightmares) that could not be directly observed by a third party, so self-report or interview were the only possible methods of data collection. Furthermore, it can be reasonably assumed that biases in memory and reporting style were common to the active and control groups, such that errors in reporting were likely balanced between the groups and therefore did not have a significant impact on the overall results.

The final limitation addresses characteristics of the study sample that were not directly assessed or controlled during the study. While none of the participants were receiving individual psychotherapy at the time of the study, many were meeting with TRP psychiatrists for medication management. It is therefore possible that veterans in both groups were going on or coming off psychotropic medications at the time of the study, which may have impacted the frequency or intensity of their symptoms. Again, it

can be reasonably assumed that such changes were occurring at comparable rates between the active and control groups, but as it was not directly assessed, there remains a slight chance that these changes in medication or other non-assessed clinically significant changes in physical health, employment status, family life, etc. may have had an impact on the results of the study.

Future Directions

In response to the brevity of the intervention and the data collection window, one potentially fruitful avenue of research may be in determining whether the impact of the intervention may extend past the four-week study period. Specifically, determining whether those in the active group, particularly those who complete the monitoring at a higher rate, showed greater or more rapid improvement in the more structured and intensive interventions that they were offered upon completion of the PTSD 101 class could shed light on the possibility of a "sleeper effect" resulting from the four-week intervention. A comparison of the outcomes of the active versus control groups following the patients' subsequent course of individual psychotherapy could indicate whether exposure to information about the potential benefits of intrusion monitoring and instructions on how to engage in this form of monitoring may impact longer-term symptom improvement. Anecdotal reports from clinicians involved in the subsequent exposure treatment of study participants suggested that engaging in intrusion monitoring during the study period may have increased patients' willingness to engage in imaginal exposure and positively impacted overall treatment gains. A controlled study designed to empirically test these informal observations would be highly informative.

Another area of potential future research is on the impact of illness duration on response to intrusion monitoring. Although time since the occurrence of the index trauma was not explicitly assessed in this study, the finding that younger veterans assigned to the active condition fared better than older veterans in the same condition suggests that intrusion monitoring may be more effective if begun sooner after trauma exposure, such as during the first month of symptom expression. One possible explanation for the impact of illness duration on outcome may be that avoidance behaviors often become more strongly entrenched over the course of illness (Creamer et al., 1992; O'Donnell et al., 2007), making a self-directed intervention more challenging for individuals with longer duration of illness. Follow-up studies are needed to assess duration of symptoms and time since the trauma as potential moderators of treatment outcome.

The impact of multiple trauma exposures on individuals' ability to benefit from intrusion monitoring may be another fruitful avenue of research. The theoretical foundation for this intervention rests on the idea that reporting and reflecting on re-experiencing events exposes the individual to memories of the trauma and facilitates emotional processing of the event. If, however, the individual has been exposed to multiple "Criterion A1" traumas, it is likely that their intrusive symptoms will relate to more than one of these events, decreasing the amount of repetition provided by the exposures and diffusing the impact of the intervention on symptoms. Future research in this area may be directed toward assessing the content of re-experiencing events in order to determine whether intrusion monitoring may be more effective for individuals who

have experienced only one traumatic event or only one trauma context, compared with the typical VA patient, who has been exposed to multiple Criterion A1 traumas.

Conclusions

Completing a daily intrusion monitoring assignment prior to engaging in intensive individual treatment did not, on average, have a significant beneficial effect on symptoms of PTSD and related psychopathology among American military veterans with chronic and severe PTSD. Younger veterans showed greater likelihood of benefit from the intervention than those over age 30. The reasons for this differential response are largely unknown, although it is likely that these veterans may have had a shorter duration of illness and may have been exposed to fewer traumatic events in only one context. Younger veterans may also have more health-promoting resources available to them compared with older participants. On the whole, however, re-experiencing symptom monitoring by itself cannot be recommended as a sole intervention for chronic and severe PTSD on the basis of this study, even though studies with other populations have reported significant rates of patients who no longer met criteria for PTSD after such an intervention. Future research may be designed to test the efficacy of intrusion monitoring as a treatment for ASD or as an even more broadly applied exercise to help those who have recently experienced trauma cope with their reactions to the event. It also remains to be seen if engaging in the four-week intervention potentiates longer-term treatment effects, particularly among individuals who had been more compliant with the daily monitoring.

Despite a strong theoretical basis and a firm grounding in previous research, the self-monitoring intervention assessed in the present study did not prove beneficial for the

majority of veterans who participated. The reasons for this discrepancy between the previous findings, the theory-based hypotheses, and the results of the present study are unclear. This study was the first to include a no-monitoring control condition, and the population sampled had a more chronic and severe presentation than those included in the previous studies. Compliance with the instruction to record re-experiencing events on a daily basis was quite low, which could have significantly attenuated treatment response. Future research in this area should consider the barriers to consistent compliance and should continue to examine the degree to which intrusion monitoring may be helpful as a cost-effective early step in the treatment of ASD and PTSD.

References

American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text revision). Washington, D.C.: Author.

Beck, A. T., Steer, R. A., Ball, R. & Ranieri, W. F. (1996).Comparison of Beck Depression Inventories -IA and –II in psychiatric outpatients.*Journal of Personality Assessment, 67,* 588-597.doi: 10.1207/s15327752jpa6703_13

Blanchard, E. B., Jones-Alexander, J., Buckley, T. C., & Forneris, C. A. (1996). Psychometric properties of the PTSD checklist.*Behavior Research and Therapy, 34*, 669-673. doi: 10.1016/0005-7967(96)00033-2

Brewin, C. R., Dalgleish, T., & Joseph, S. (1996). A dual representation theory of posttraumatic stress disorder.*Psychological Review, 103*, 670-686.doi: 10.1037/0033-295X.103.4.670

Bryant, R. A., Harvey, A. G., Dang, S. T., Sackville, T., & Basten, C. (1998). Treatment of acute stress disorder: A comparison of cognitive-behavioral therapy and supportive counseling. *Journal of Consulting and Clinical Psychology, 66,* 862-866.doi: 10.1037/0022-006X.66.5.862

Bryant, R. A., Mastrodomenico, J., Felmingham, K. L., Hopwood, S., Kenny, L., Kandris, E., … Creamer, M. (2008). Treatment of acute stress disorder: A randomized controlled trial. *Archives of General Psychiatry, 65*, 659-667.doi: 10.1001/archpsyc.65.6.659

Chemtob, C., Roitblat, H. L., Hamada, R. S., Carlson, J. G., & Twentyman, C. T. (1988). A cognitive action theory of post-traumatic stress disorder. *Journal of Anxiety Disorders, 2*, 253-275. doi: 10.1016/0887-6185(88)90006-0

Creamer, M., Burgess, P., & Pattison, P. (1992). Reaction to trauma: A cognitive processing model. *Journal of Abnormal Psychology, 101,* 452-459. doi: 10.1037/0021-843X.101.3.452

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

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy, 38,* 319-345. doi: 10.1016/S0005-7967(99)00123-0

Ehlers, A., Clark, D. M., Hackmann, A., McManus, F., Fennell, M., Herbert, C., et al. (2003). A randomized controlled trial of cognitive therapy, a self-help booklet, and repeated assessments as early interventions for posttraumatic stress disorder. *Archives of General Psychiatry, 60*(10), 1024-1032. doi: 10.1001/archpsyc.60.10.1024

Foa, E. B., Ehlers, A., Clark, D. M., Tolin, D. F., & Orsillo, S. M. (1999). The Posttraumatic Cognitions Inventory (PTCI): Development and validation. *Psychological Assessment, 11*, 303-314. doi: 10.1037/1040-3590.11.3.303

Foa, E. B., Hembree, E. A., & Rothbaum, B. O. (2007). *Prolonged Exposure Therapy for PTSD: Emotional Processing of Traumatic Experiences*. New York: Oxford University Press.

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: exposure to corrective information. *Psychological Bulletin, 99*(1), 20-35. doi: 10.1037/0033-2909.99.1.20

Foa, E. B.., & Riggs, D. S. (1993). Posttraumatic stress disorder and rape. In J. Oldham & M. Riba, (Eds), *Review of Psychiatry,* (273-303). Washington, D.C.: American Psychiatric Press, Inc.

Foa, E. B., Riggs, D. S., Dancu, C. V., & Rothbaum, B. O. (1993). Reliability and validity of a brief instrument for assessing post-traumatic stress disorder. *Journal of Traumatic Stress, 6,* 459-473. doi: 10.1002/jts.2490060405

Foa, E. B., & Rothbaum, B. O. (1998). *Treating the trauma of rape: Cognitive–behavioral therapy for PTSD.* New York: Guilford Press.

Foa, E. B., Steketee, G., & Rothbaum, B. O. (1989). Behavioral/cognitive conceptualizations of post-traumatic stress disorder. *Behavior Therapy, 20*(2), 155-176. doi: 10.1016/S0005-7894(89)80067-X

Hardy, E., & Stallard, P. (2008). A pilot study to explore the effect of symptom monitoring in children and adolescents with posttraumatic stress reactions following road traffic accidents. *Behavioural and Cognitive Psychotherapy, 36,* 431-435. doi: 10.1017/S1352465808004347

Hoge, C. W., Terhakopian, A., Castro, C. A., Messer, S. C., & Engel, C. C. (2007). Association of posttraumatic stress disorder with somatic symptoms, health care visits, and absenteeism among Iraq war veterans. *American Journal of Psychiatry, 164*(1), 150-153.Article accessed on 11 April 2012 from http://journals.psychiatryonline.org/article.aspx?articleid=97733

Horowitz, M. J. (1974). Stress response syndromes. Character style and dynamic psychotherapy. *Archives of general psychiatry, 31*(6), 768-781. Article accessed 26 April 2012 from www.archgenpsychiatry.com

Horowitz, M. J. (1978). *Stress response syndromes*. New York: Aronson.

Hunt, M. (2009). *The Story of Psychology*. New York: Anchor Books.

Ilardi, S. S., & Craighead, W. E. (1994). The role of nonspecific factors in cognitive-behavior therapy for depression. *Clinical Psychology: Science and Practice, 1*(2), 138-155. doi: 10.1111/j.1468-2850.1994.tb00016.x

Janoff-Bulman, R. (1989). Assumptive worlds and the stress of traumatic events: Applications of the schema construct. *Social Cognition, 7*, 113-136.doi: 10.1521/soco.1989.7.2.113

Keane, T.M., Zimering, R. T and Caddell, J.M. (1985). A behavioral formulation of posttraumatic stress disorder in Vietnam veterans. *The Behavior Therapist, 8*, 9-12.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry, 62*(6), 593-602.doi: 10.1001/archpsych.62.6.593

Korotitsch, W. J., & Nelson-Gray, R. O. (1999). An overview of self-monitoring research in assessment and treatment. *Psychological Assessment, 11*(4), 415-425.doi: 10.1037/1040-3590.11.4.415

Krefetz, D. G., Steer, R. A., Gulab, N. A., & Beck, A. T. (2002). Convergent validity of the Beck Depression Inventory-II with the Reynolds Adolescent Depression Scale in psychiatric inpatients. *Journal of Personality Assessment, 78*, 451-460. doi: 10.1207/s15327752jpa7803_05

Latner, J. D., & Wilson, G. T. (2002). Self-monitoring and the assessment of binge eating. *Behavior Therapy, 33*(3), 465-477.doi: 10.1016/S0005-7894(02)80039-9

Mowrer, O. H. (1951). Two-factor learning theory: summary and comment. *Psychological review, 58*(5), 350-354.doi: 10.1037/h0058956

Nemeroff, C. B., Bremner, J. D., Foa, E. B., Mayberg, H. S., North, C. S., & Stein, M. B. (2006). Posttraumatic stress disorder: a state-of-the-science review. *Journal of psychiatric research, 40*(1), 1-21. doi: 10.1016/j.jpsychires.2005.07.005

O'Donnell, M. L., Elliott, P., Lau, W., & Creamer, M. (2007). PTSD symptom trajectories: From early to chronic response. *Behavior Research and Therapy, 45*, 601-606. doi: 10.1016/j.brat.2006.03.015

Pennebaker, J. W. (1993). Putting stress into words: health, linguistic, and therapeutic implications. *Behaviour research and therapy, 31*(6), 539-548. doi: 10.1016/0005-7967(93)90105-4

Pennebaker, J. W. (1997). Writing about emotional experiences as a therapeutic process. *Psychological Science, 8,* 162-166. doi: 10.1111/j.1467-9280.1997.tb00403.x

Pennebaker, J. W., & Beall, S. K. (1986). Confronting a traumatic event: toward an understanding of inhibition and disease. *Journal of Abnormal Psychology, 95*(3), 274-281. doi: 10.1037/0021-843X.95.3.274

Ponniah, K. & Hollon, S. D. (2009). Empirically supported psychological treatments for adult acute stress disorder and posttraumatic stress disorder: A review. *Depression and Anxiety, 26,* 1086-1109. doi: 10.1002/da.20635

Power, M. J., & Dalgleish, T. (1997). *Cognition and emotion: From order to disorder*. Hove, England: Psychology Press.

Power, M. J., & Dalgleish, T. (1999). Two routes to emotion: Some implications of multi-level theories of emotion for therapeutic practice. *Cognitive and Behavioural Psychotherapy, 27*, 129-142.

Resick, P. A., Galovski, T. E., O'Brien Uhlmansiek, M., Scher, C. D., Clum, G. A., & Young-Xu, Y. (2008). A randomized clinical trial to dismantle components of cognitive processing therapy for posttraumatic stress disorder in female victims of interpersonal violence. *Journal of consulting and clinical psychology, 76*(2), 243-258. doi: 10.1037/0022-006X.76.2.243

Resick, P. A., Monson, C. M., & Rizvi, S. L. (2008). Posttraumatic Stress Disorder. In D. H. Barlow (Ed.), *Clinical Handbook of Psychological Disorders* (pp. 65-122). New York: The Guilford Press.

Resick, P. A., Nishith, P., Weaver, T. L., Astin, M. C., & Feuer, C. A. (2002). A comparison of cognitive-processing therapy with prolonged exposure and a waiting condition for the treatment of chronic posttraumatic stress disorder in female rape victims. *Journal of Consulting and Clinical Psychology, 70*(4), 867-879.doi: 10.1037/0022-006X.70.4.867

Resick, P. A., & Schnicke, M. K. (1992). Cognitive processing therapy for sexual assault victims. *Journal of Consulting and Clinical Psychology, 60*(5), 748-756. doi: 10.1037/0022-006X.60.5.748

Resick, P. A., & Schnicke, M. K. (1996). *Cognitive processing therapy for rape victims: a treatment manual*. Newbury Park: SAGE Publications.

Reynolds, M., & Tarrier, N. (1996). Monitoring of intrusions in post-traumatic stress order: a report of single case studies. *The British journal of medical psychology, 69*, 371-379.doi: 10.1111/j.2044-8341.1996.tb01879.x

Schacter, D. L. (1999). The seven sins of memory: Insights from psychology and cognitive neuroscience. *American Psychologist, 54*, 182-203.

Schneiderman, A. I., Braver, E. R., & Kang, H. K. (2008). Understanding sequelae of injury mechanisms and mild traumatic brain injury incurred during the conflicts in Iraq and Afghanistan: Persistent postconcussive symptoms and posttraumatic stress disorder. *American journal of epidemiology, 167*(12), 1446-1452. doi: 10.1093/aje/kwn068

Sloan, D. M., Marx, B. P., & Epstein, E. M. (2005). Further examination of the exposure model underlying the efficacy of written emotional disclosure. *Journal of consulting and clinical psychology, 73*(3), 549-554. doi: 10.1037/0022-006X.73.3.549

Smith, P., Yule, W., Perrin, S., Tranah, T., Dalgleish, T., & Clark, D. M. (2007). Cognitive-behavioral therapy for PTSD in children and adolescents: A preliminary randomized controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry, 46*(8), 1051-1061. doi: 10.1097/CHI.0b013e318067e288

Storch, E. A., Roberti, J. W., & Roth, D. A. (2004). Factor structure, concurrent validity, and internal consistency of the Beck Depression Inventory-Second Edition in a sample of college students. *Depression and Anxiety, 19*, 187-189. doi: 10.1002/da.20002

Tarrier, N., Sommerfield, C., Reynolds, M., & Pilgrim, H. (1999). Symptom self-monitoring in the treatment of posttraumatic stress disorder. *Behavior Therapy, 30*, 597-605. doi: 10.1016/S0005-7894(99)80027-6

Taylor, S., Zvolensky, M. J., Cox, B. J., Deacon, B., Heimberg, R. G., Ledley, D. R.,…Bouvard, M. (2007). Robust dimensions of anxiety sensitivity: Development and initial validation of the Anxiety Sensitivity Index - 3. *Psychological Assessment, 19*, 176-188. doi: 10.1037/1040-3590.19.2.176

Thiele, C., Laireiter, A., & Baumann, U. (2002). Diaries in clinical psychology and psychotherapy: A selective review. *Clinical Psychology and Psychotherapy, 9*, 1-37. doi: 10.1002/cpp.302

van der Kolk, B. A. (2007). The history of trauma in psychiatry. In M. J. Friedman, T. M. Keane & P. A. Resick (Eds.), *Handbook of PTSD: Science and practice* (pp. 19-36). New York, NY: The Guilford Press.

van Emmerik, A. A., Schoorl, M., Emmelkamp, P. M. G., & Kamphuis, J. H. (2006). Psychometric evaluation of the Dutch version of the Posttraumatic Cognitions Inventory (PTCI). *Behavior Research and Therapy, 44*, 1053-1065. doi: 10.1016/j.brat.2005.07.002

Wilson, G. T., & Vitousek, K. M. (1999). Self-monitoring in the assessment of eating disorders. *Psychological Assessment, 11*, 480-489. doi: 10.1037/1040-3590.11.4.480

Tables and Figures

Table 1: DSM-IV criteria for posttraumatic stress disorder

Criterion A (both required)	*Stressor*	1. The person has experienced, witnessed, or been confronted with an e event or events that involve actual or threatened death or serious injury, or a threat to the physical integrity of oneself or others
		2. The person's response involved intense fear, helplessness, or horror
Criterion B (at least 1)	*Intrusive recollection/ re-experiencing*	1. Recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions
		2. Recurrent distressing dreams of the event
		3. Acting or feeling as if the traumatic event were recurring
		4. Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
		5. Physiological reactivity upon exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
Criterion C (at least 3)	*Avoidance/ numbing*	1. Efforts to avoid thoughts, feelings, or conversations about the trauma
		2. Efforts to avoid activities, places, or people that arouse recollections of the traumatic event
		3. Inability to recall an important aspect of the trauma
		4. Markedly diminished interest or participation in significant activities
		5. Feeling of detachment or estrangement from others
		6. Restricted range of affect
		7. Sense of foreshortened future
Criterion D *(at least 2)*	*Hyperarousal*	1. Difficulty falling asleep or staying asleep
		2. Irritability or outbursts of anger
		3. Difficulty concentrating
		4. Hypervigilance
		5. Exaggerated startle response
Criterion E	*Duration*	Duration of the disturbance is more than one month
Criterion F	*Functional significance*	The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning
Specifier 1	*Acute vs. Chronic*	1. Acute – Duration is less than three months 2. Chronic – Duration is three months or more
Specifier 2	*With or Without Delayed Onset*	1. With Delayed Onset – Onset of symptoms occurs at least six months after the stressor
		2. Without Delayed Onset – Onset of symptoms occurs within six months of the stressor

Table 2: Demographic information – Overall sample

Gender	**Active (N)**	**Control (N)**	**Total (N)**	**Total (%)**
Male	76	45	121	88.3%
Female	11	5	16	11.7%
Military branch				
Army	65	37	102	74.5%
USMC	14	4	18	13.1%
Navy	5	5	10	7.3%
Air Force	2	1	3	2.2%
Reserves/Nat'l Guard	1	2	3	2.2%
Relationship status				
Married	46	27	73	53.3%
Never married	21	7	28	20.4%
Divorced	15	9	24	17.5%
Separated	4	4	8	5.8%
Remarried	1	3	4	2.9%
Race/Ethnicity				
African-American	63	40	103	75.2%
White, Non-Hispanic	17	6	23	16.8%
Other	2	1	3	2.2%
Hispanic, White	1	2	3	2.2%
Hispanic, Black	1	1	2	1.5%
Asian	2	0	2	1.5%
American Indian/Alaskan	1	0	1	0.7%
Employment status				
Unemployed	32	16	48	35.0%
Employed full-time for pay	23	13	36	26.3%
Full-time student	8	10	18	13.1%
Employed part-time for pay	10	5	15	10.2%
Officially retired	9	1	10	7.3%
Certified 100% disabled	1	0	1	0.7%
Number of war zones				
One	51	32	83	60.6%
Two	29	15	44	32.1%
Three	7	2	9	6.6%
Four	0	1	1	0.7%
Military sexual trauma				
No	83	50	133	97.1%
Yes	4	0	4	2.9%
TBI diagnosis				
No	65	36	101	73.7%
Yes	22	14	36	26.3%

Table 3: Clinical information –Overall sample

	Poss.	Obs. Range	Mean	St. Deviation
Age				
Overall (N=137)		22-60	36.28	8.83
Active group (N=87)		22-54	35.26	8.41
Control group (N=50)		23-60	38.04	9.32
Years of education				
Overall (N=137)		12-18	13.58	1.77
Active group (N=87)		12-18	13.70	1.86
Control group (N=50)		12-18	13.38	1.60
PSS Intrusive	0-15			
Overall (N=137)		0-15	9.55	3.35
Active group (N=87)		2-15	9.76	3.36
Control group (N=50)		0-15	9.20	3.34
PSS Avoidance	0-21			
Overall (N=137)		5-21	15.34	3.87
Active group (N=87)		5-21	15.40	3.97
Control group (N=50)		6-21	15.22	3.72
PSS Hyperarousal	0-15			
Overall (N=137)		7-15	12.41	2.29
Active group (N=87)		8-15	12.56	2.20
Control group (N=50)		7-15	12.14	2.43
PSS Total	0-51			
Overall (N=137)		20-51	37.30	8.04
Active group (N=87)		21-51	37.72	8.32
Control group (N=50)		20-51	36.56	7.54
PCL Intrusive	5-25			
Overall (N=131)		5-25	17.15	4.78
Active group (N=86)		6-25	17.50	4.94
Control group (N=45)		5-25	16.47	4.45
PCL Avoidance	7-35			
Overall (N=131)		11-35	25.75	5.76
Active group (N=86)		11-34	25.92	5.86
Control group (N=45)		14-35	25.42	5.61
PCL Hyperarousal	5-25			
Overall (N=131)		10-25	20.25	4.02
Active group (N=86)		10-25	20.55	4.03
Control group (N=45)		12-25	19.69	3.98
PCL Total	17-85			
Overall (N=131)		31-85	63.15	12.99
Active group (N=86)		31-84	63.97	13.35
Control group (N=45)		35-85	61.58	12.28
BDI-II	0-63			

Overall (N=136)		11-62	31.32	12.01
Active group (N=86)		11-58	31.63	12.23
Control group (N=50)		14-62	30.80	11.74
ASI Physical	0-24			
Overall (N=129)		0-24	10.98	7.03
Active group		0-24	10.44	7.17
Control group (N=45)		0-24	12.00	6.72
ASI Cognitive	0-24			
Overall (N=129)		0-24	12.73	6.94
Active group (N=84)		0-24	12.36	7.35
Control group (N=45)		0-23	13.42	6.13
ASI Social	0-24			
Overall (N=129)		0-24	12.60	5.87
Active group (N=84)		0-24	12.33	6.20
Control group (N=45)		3-24	13.09	5.22
PTCI Self	1-7			
Overall (N=123)		1.05-7	4.12	1.37
Active group (N=80)		1.05-7	4.07	1.38
Control group (N=43)		1.14-6.48	4.23	1.35
PTCI World	1-7			
Overall (N=123)		2.43-7	5.82	1.01
Active group (N=80)		2.43-7	5.82	1.03
Control group (N=43)		2.71-7	5.82	1.00
PTCI Self-blame	1-7			
Overall (N=123)		1-7	2.99	1.63
Active group (N=80)		1-7	3.08	1.69
Control group (N=43)		1-6.40	2.83	1.53
PTCI Total	36-252			
Overall (N=123)		48-231	142.37	39.67
Active group (N=80)		56-231	141.62	40.38
Control group (N=43)		48-217	143.76	38.75

Table 4: Demographic information - Completers

Gender	**Active (N)**	**Control (N)**	**Total (N)**	**Total (%)**
Male	47	30	77	87.5%
Female	7	4	11	12.5%
Military branch				
Army	40	24	64	72.7%
USMC	8	4	12	13.6%
Navy	4	4	8	9.1%
Air Force	1	0	1	1.1%
Reserves/Nat'l Guard	1	1	2	2.3%
Relationship status				
Married	32	17	49	55.7%
Never married	8	4	12	13.6%
Divorced	10	7	17	19.3%
Separated	3	3	6	6.8%
Remarried	1	3	4	4.5%
Race/Ethnicity				
African-American	43	28	71	80.7%
White, Non-Hispanic	7	3	10	11.4%
Other	1	0	1	1.1%
Hispanic, White	1	2	3	3.4%
Hispanic, Black	1	1	2	2.3%
Asian	1	0	1	1.1%
American Indian/Alaskan	0	0	0	0.0%
Employment status				
Unemployed	18	8	26	29.5%
Employed full-time for pay	15	10	25	28.4%
Full-time student	4	9	13	14.8%
Employed part-time for pay	8	3	11	12.5%
Officially retired	6	4	10	11.4%
Certified 100% disabled	0	0	0	0.0%
Number of war zones				
One	32	23	55	62.5%
Two	17	8	25	28.4%
Three	5	2	7	8.0%
Four	0	1	1	1.1%
Military sexual trauma				
No	53	34	87	98.9%
Yes	1	0	1	1.1%
TBI diagnosis				
No	41	24	65	73.9%
Yes	13	10	23	26.1%

Table 5: Clinical information – Completers

	Poss. Range	Obs. Range	Mean	St. Deviation
Age				
Overall (N=88)		23-60	37.64	8.69
Active group (N=54)		24-52	37.04	8.27
Control group (N=34)		23-60	38.59	9.38
Years of education				
Overall (N=88)		12-18	13.67	1.76
Active group (N=54)		12-18	14.00	1.85
Control group (N=34)		12-18	13.15	1.48
PSS Intrusive	0-15			
Overall (N=88)		2-15	9.39	3.37
Active group (N=54)		2-15	9.44	3.53
Control group (N=34)		3-14	9.29	3.15
PSS Avoidance	0-21			
Overall (N=88)		5-21	15.34	3.78
Active group (N=54)		5-21	15.33	3.90
Control group (N=34)		9-21	15.35	3.63
PSS Hyperarousal	0-15			
Overall (N=88)		7-15	12.40	2.29
Active group (N=54)		8-15	12.30	2.24
Control group (N=34)		7-15	12.56	2.40
PSS Total	0-51			
Overall (N=88)		20-51	37.12	8.10
Active group (N=54)		21-51	37.07	8.59
Control group (N=34)		20-50	37.21	7.39
PCL Intrusive	5-25			
Overall (N=86)		6-25	17.09	5.00
Active group (N=54)		6-25	17.19	5.28
Control group (N=32)		7-25	16.94	4.56
PCL Avoidance	7-35			
Overall (N=86)		11-35	25.92	5.57
Active group (N=54)		11-34	25.63	5.83
Control group (N=32)		15-35	26.41	5.15
PCL Hyperarousal	5-25			
Overall (N=86)		10-25	20.31	3.83
Active group (N=54)		10-25	20.20	3.89
Control group (N=32)		12-25	20.50	3.79
PCL Total	17-85			
Overall (N=86)		31-85	63.33	12.99
Active group (N=54)		31-84	63.02	13.80
Control group (N=32)		41-85	63.84	11.68
BDI-II	0-63			
Overall (N=88)		11-62	31.27	11.93
Active group (N=54)		11-55	31.28	12.34

Control group (N=34)		17-62	31.26	11.43
ASI Physical	0-24			
Overall (N=85)		0-24	11.72	6.79
Active group (N=54)		0-24	11.26	6.85
Control group (N=32)		0-24	12.47	6.72
ASI Cognitive	0-24			
Overall (N=85)		0-24	13.20	6.48
Active group (N=53)		1-24	12.55	6.93
Control group (N=32)		0-23	14.28	5.60
ASI Social	0-24			
Overall (N=85)		0-24	12.86	5.52
Active group (N=53)		0-22	12.51	5.77
Control group (N=32)		3-24	13.44	5.12
PTCI Self	1-7			
Overall (N=81)		1.05-6.48	4.14	1.39
Active group (N=50)		1.05-6.19	3.40	1.43
Control group (N=31)		1.90-6.48	4.36	1.30
PTCI World	1-7			
Overall (N=81)		3.43-7	5.88	0.97
Active group (N=50)		3.43-7	5.81	1.05
Control group (N=31)		4-7	5.98	0.83
PTCI Self-blame	1-7			
Overall (N=81)		1-6.40	3.04	1.62
Active group (N=50)		1-6	3.17	1.67
Control group (N=31)		1-6.40	2.84	1.53
PTCI Total	36-252			
Overall (N=81)		56-217	143.25	40.17
Active group (N=50)		56-209	140.49	42.42
Control group (N=31)		89-217	147.70	36.48

Table 6: Results of repeated measures ANOVAs, active status as independent variable

	F	p	Eta Squared	Observed Power
PSS Avoidance				
Main effect (time)	1.89	.172	.022	.275
Interaction (group*time)	.002	.969	.000	.050
PCL Avoidance				
Main effect (time)	*6.96**	*.010**	*.076*	*.741*
Interaction (group*time)	.002	.962	.000	.050
PSS Intrusive				
Main effect (time)	.071	.790	.001	.058
Interaction (group*time)	.514	.475	.006	.109
PCL Intrusive				
Main effect (time)	1.07	.305	.013	.175
Interaction (group*time)	.000	.990	.000	.050
PSS Total				
Main effect (time)	1.81	.182	.021	.265
Interaction (group*time)	.118	.732	.001	.063
PCL Total				
Main effect (time)	*4.75**	*.032**	*.054*	*.577*
Interaction (group*time)	.083	.774	.001	.059
BDI Total				
Main effect (time)	.269	.605	.003	.081
Interaction (group*time)	.530	.469	.006	.111
ASI Physical				
Main effect (time)	.281	.598	.003	.082
Interaction (group*time)	.001	.979	.000	.050
ASI Cognitive				
Main effect (time)	.048	.827	.001	.055
Interaction (group*time)	.005	.947	.000	.051
ASI Social				
Main effect (time)	.870	.175	.023	.272
Interaction (group*time)	.035	.853	.000	.054
PTCI Self				
Main effect (time)	.981	.088	.038	.399
Interaction (group*time)	.088	.300	.014	.178
PTCI World				
Main effect (time)	.646	.424	.009	.125
Interaction (group*time)	.081	.776	.001	.059
PTCI Self-blame				
Main effect (time)	.998	.162	.026	.287
Interaction (group*time)	.017	.898	.000	.052
PTCI Total				
Main effect (time)	.959	.090	.038	.397
Interaction (group*time)	.430	.514	.006	.099

Figure 1: Change in distress related to avoidance symptoms

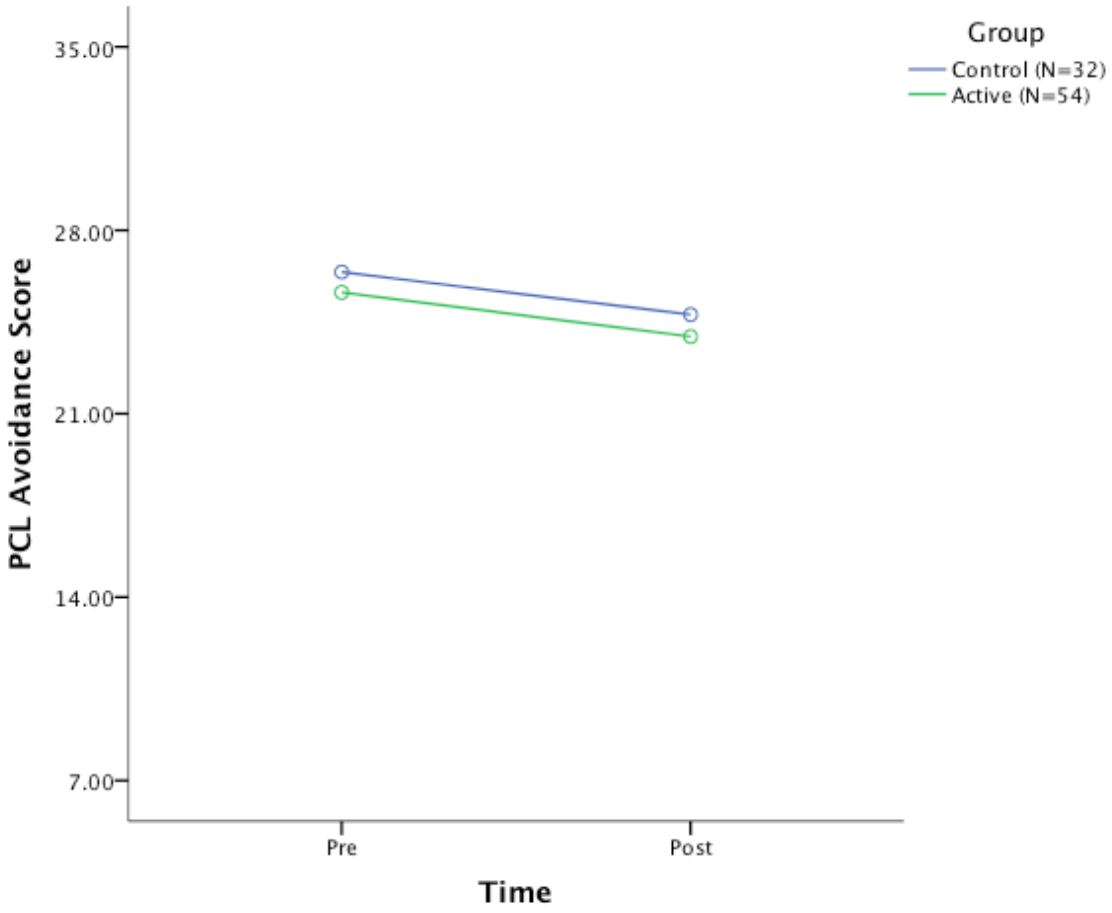


Table 7: Demographic moderator analyses (time*active group*moderator interaction)

	F (Interaction)	p	Eta Squared	Observed Power
PSS Total				
Age	*7.92**	*.006**	*.086*	*.794*
Gender	.147	.702	.002	.067
Marital status	.055	.815	.001	.056
Years of education	.171	.680	.002	.069
Race/Ethnicity	2.70	.104	.031	.369
Employment	.352	.554	.004	.090
Number of war zones	*5.03**	*.028**	*.056*	*.601*
PCL Total				
Age	*4.93**	*.029**	*.057*	*.593*
Gender	.066	.799	.001	.057
Marital status	.350	.556	.004	.090
Years of education	.014	.906	.000	.052
Race/Ethnicity	.003	.955	.000	.050
Employment	.015	.904	.000	.052
Number of war zones	1.11	.296	.013	.180
BDI Total				
Age	.066	.798	.001	.057
Gender	.125	.725	.001	.064
Marital status	.245	.622	.003	.078
Years of education	.679	.412	.008	.129
Race/Ethnicity	1.61	.208	.019	.241
Employment	.980	.325	.012	.165
Number of war zones	2.50	.118	.029	.346
ASI Total				
Age	2.98	.088	.036	.399
Gender	.538	.466	.007	.112
Marital status	.540	.465	.007	.112
Years of education	.086	.770	.001	.060
Race/Ethnicity	2.68	.106	.033	.365
Employment	.036	.850	.000	.054
Number of war zones	.522	.472	.007	.110
PTCI Total				
Age	2.86	.095	.038	.385
Gender	.090	.765	.001	.060
Marital status	1.36	.247	.018	.211
Years of education	.322	.572	.004	.087
Race/Ethnicity	1.66	.202	.022	.246
Employment	1.08	.303	.015	.176
Number of war zones	2.08	.154	.028	.296

Table 8: Follow-up demographic moderator analyses (time*active group*moderator interaction)

	F (Interaction)	p	Eta Squared	Observed Power
PSS Intrusive				
Age	3.38	.069	.039	.444
Number of war zones	3.87	.053	.044	.494
PSS Avoidance				
Age	*9.62**	*.003**	*.103*	*.865*
Number of war zones	*4.14**	*.045**	*.047*	*.521*
PSS Hyperarousal				
Age	3.60	.061	.041	.466
Number of war zones	2.66	.107	.031	.364
PCL Intrusive				
Age	*5.42**	*.022**	*.062*	*.633*
PCL Avoidance				
Age	*6.24**	*.014**	*.071*	*.695*
PCL Hyperarousal				
Age	1.10	.297	.013	.179

Table 9: Chi-square test crosstabulation: age group and number of war zones

		Number of war zones	
Age Group		**1**	**2 or more**
30 and under	Observed	24	3
	Expected	16.9	10.1
31 – 44	Observed	19	15
	Expected	21.3	12.8
45 and over	Observed	12	15
	Expected	16.9	10.1

Table 10: Results of repeated measures ANOVAs: Active group*time interaction terms

	F	p	Eta Squared	Observed Power
PSS Avoidance				
30 and under	*8.79**	*.007**	*.260*	*.813*
31 - 44	.481	.493	.015	.103
45 and over	2.10	.160	.077	.286
PCL Intrusive				
30 and under	4.09**	.054**	.146	.493
31 - 44	.375	.544	.012	.091
45 and over	3.20	.086	.118	.405
PCL Avoidance				
30 and under	3.81**	.063**	.137	.465
31 - 44	.068	.796	.002	.057
45 and over	2.80	.107	.104	.362

Figure 2: Interaction between group and time among younger veterans: frequency of avoidance symptoms

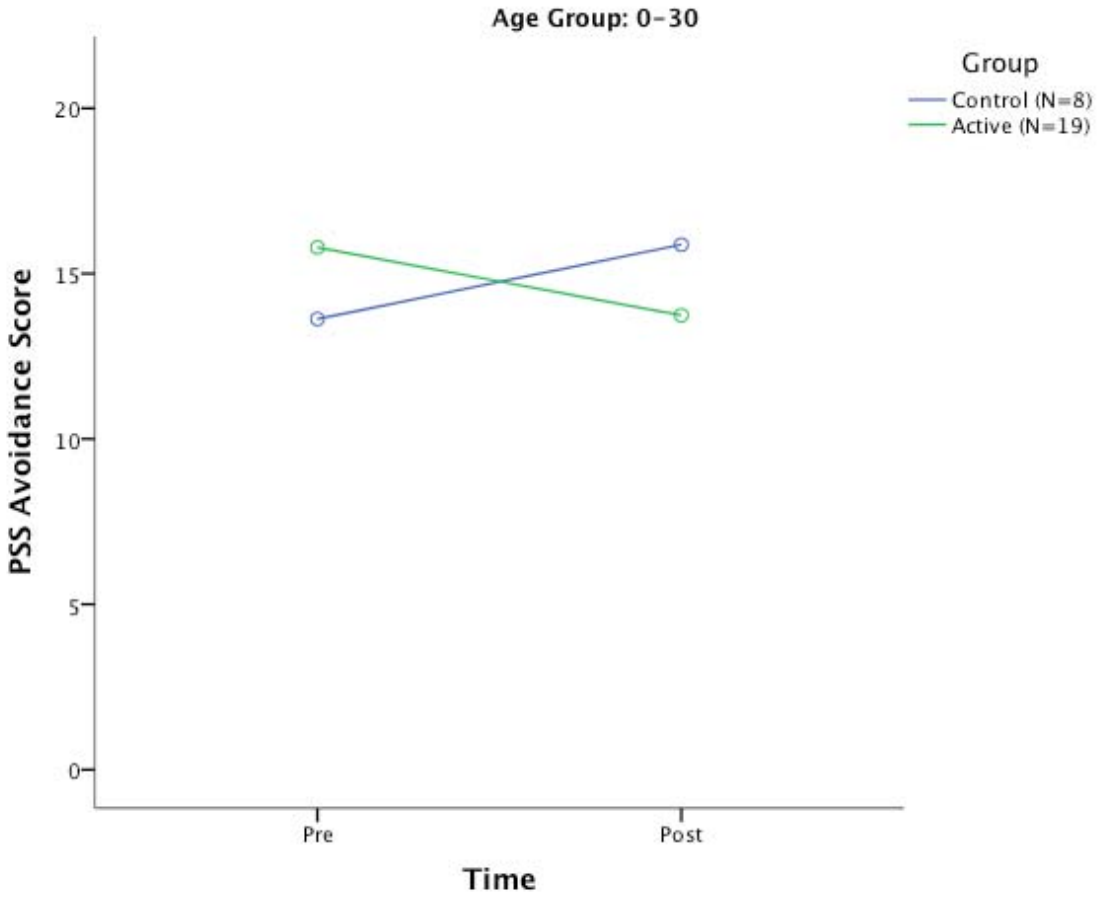


Figure 3: Interaction between group and time among younger veterans: distress associated with intrusive symptoms

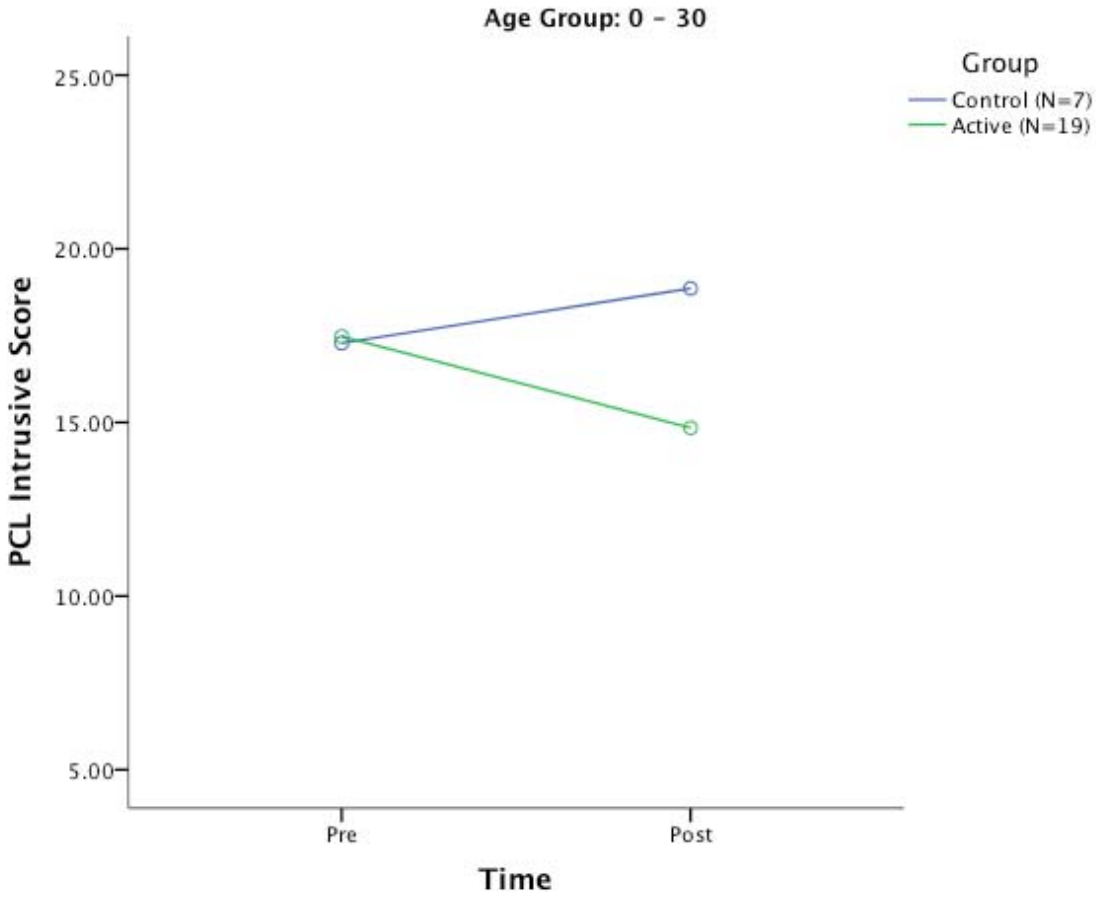


Figure 4: Interaction between group and time among younger veterans: distress associated with avoidance symptoms

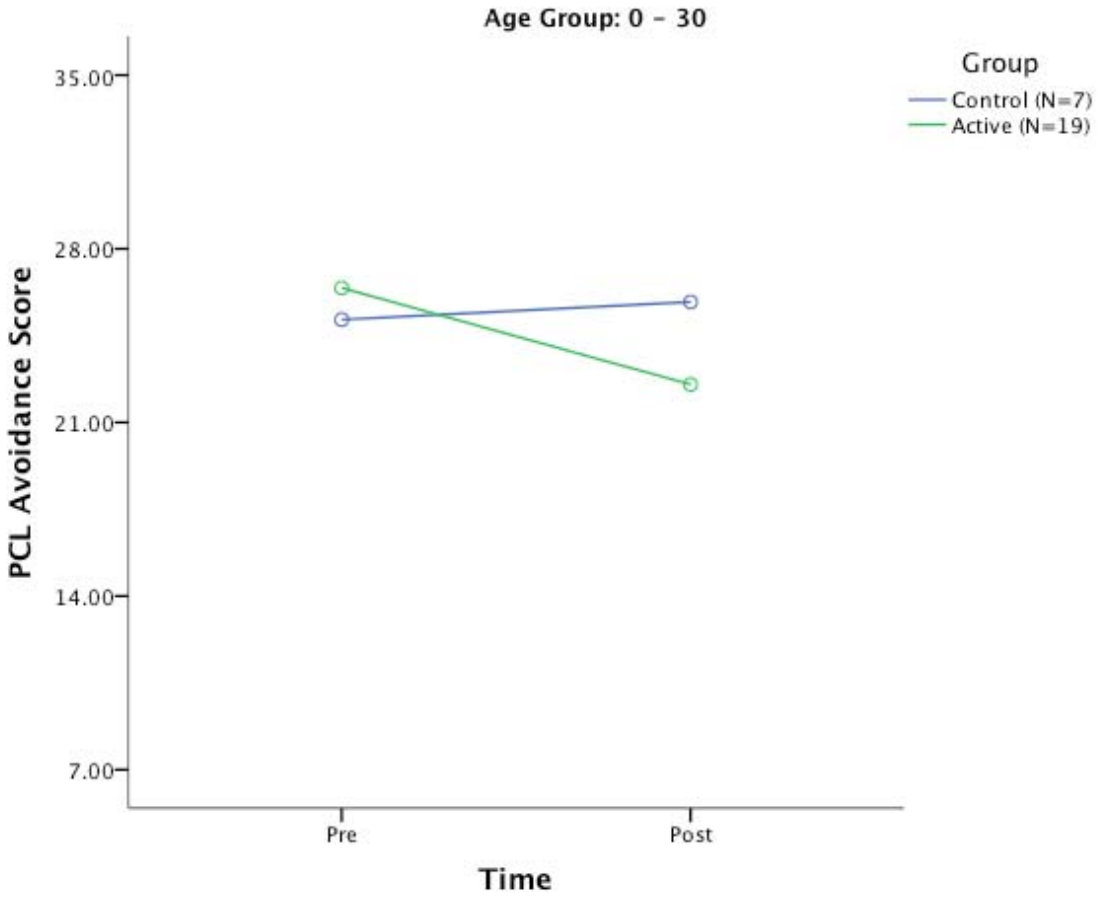


Table 11: Correlations between cumulative monitoring days and baseline data

	Pearson's r	**p**
Age	.142	.305
Years of Education	-.039	.782
PSS Intrusive	-.204	.139
PSS Avoidance	-.173	.212
PSS Hyperarousal	-.171	.216
PSS Total	-.207	.133
PCL Intrusive	-.128	.355
PCL Avoidance	-.166	.232
PCL Hyperarousal	-.222	.106
PCL Total	-.182	.189
BDI-II	-.256	.061
ASI Physical	.047	.737
ASI Cognitive	-.260	.060
ASI Social	-.092	.513
PTCI Self	-.180	.210
PTCI World	-.164	.255
PTCI Self blame	-.158	.272
PTCI Total	-.187	.194

Table 12: Correlations between cumulative monitoring days and pre-post change on symptom measures

	Pearson's r	**p**
PSS Intrusive	-.221	.199
PSS Avoidance	.057	.685
PSS Hyperarousal	-.100	.477
PSS Total	-.076	.598
PCL Intrusive	-.071	.623
PCL Avoidance	-.045	.750
PCL Hyperarousal	-.218	.117
PCL Total	-.159	.275
BDI-II	-.200	.155
ASI Physical	.014	.923
ASI Cognitive	-.105	.465
ASI Social	-.032	.824
PTCI Self	-.161	.295
PTCI World	-.139	.364
PTCI Self blame	-.021	.891
PTCI Total	-.177	.249

Figure 5: Frequencies of cumulative monitoring days, all active group participants

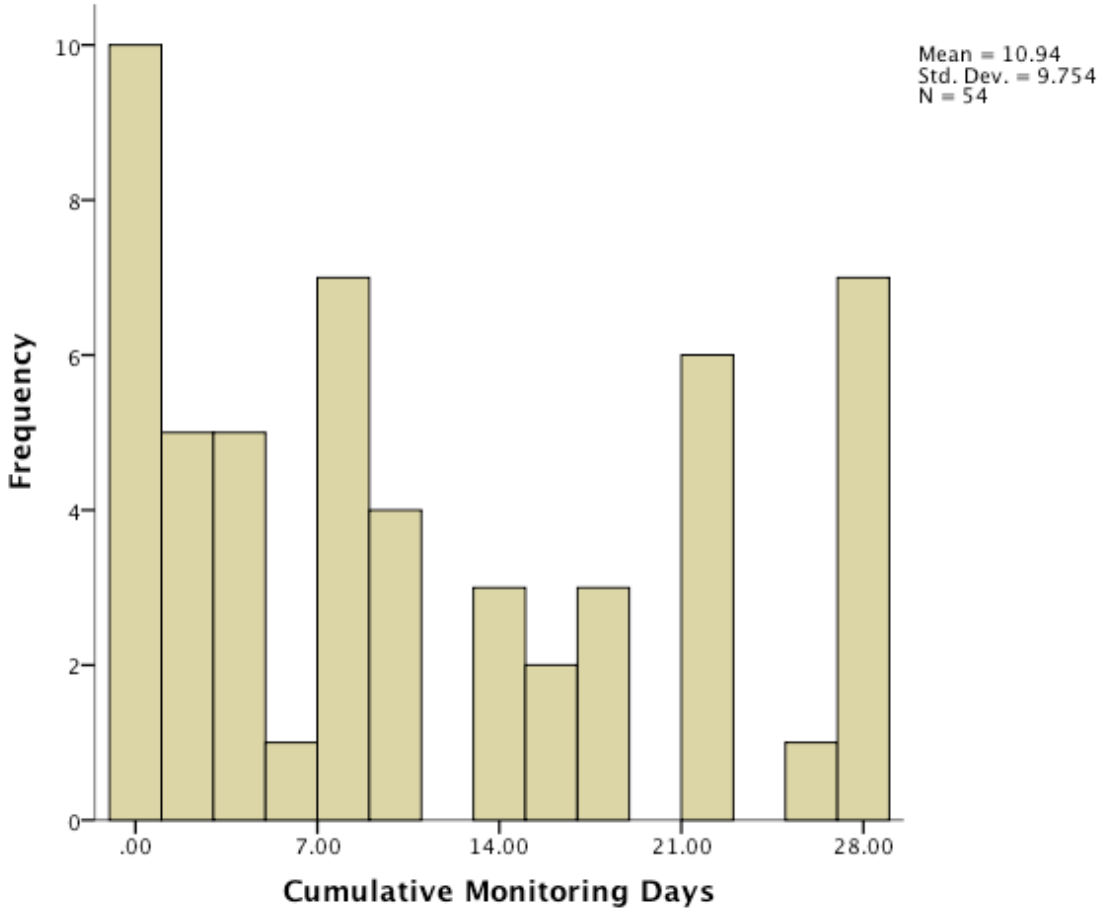


Table 13: Results of Repeated Measures ANOVAs, monitoring compliance group as independent variable

	F	p	Eta Squared	Observed Power
PSS Avoidance				
Main effect (time)	2.00	.161	.023	.287
Interaction (group*time)	.811	.491	.028	.218
PCL Avoidance				
Main effect (time)	6.53*	.012*	.074	.714
Interaction (group*time)	.075	.973	.003	.063
PSS Intrusive				
Main effect (time)	.031	.862	.000	.053
Interaction (group*time)	.611	.610	.021	.172
PCL Intrusive				
Main effect (time)	.741	.392	.009	.136
Interaction (group*time)	.380	.768	.014	.122
PSS Total				
Main effect (time)	1.34	.250	.016	.208
Interaction (group*time)	1.24	.300	.042	.321
PCL Total				
Main effect (time)	4.65*	.034*	.054	.568
Interaction (group*time)	.511	.676	.018	.150
BDI Total				
Main effect (time)	.002	.961	.000	.050
Interaction (group*time)	.843	.474	.029	.226
ASI Physical				
Main effect (time)	.227	.635	.003	.076
Interaction (group*time)	.600	.617	.022	.170
ASI Cognitive				
Main effect (time)	.127	.723	.002	.064
Interaction (group*time)	.306	.821	.011	.107
ASI Social				
Main effect (time)	2.34	.130	.029	.327
Interaction (group*time)	.587	.625	.022	.167
PTCI Self				
Main effect (time)	5.46*	.022*	.070	.635
*Interaction (group*time)*	*2.83**	*.045**	*.104*	*.656*
PTCI World				
Main effect (time)	.633	.429	.009	.123
Interaction (group*time)	2.39	.076	.089	.575
PTCI Self-blame				
Main effect (time)	1.73	.193	.023	.255
Interaction (group*time)	1.01	.392	.040	.265

Figure 6: Differences in outcome among monitoring groups: negative cognitions about self

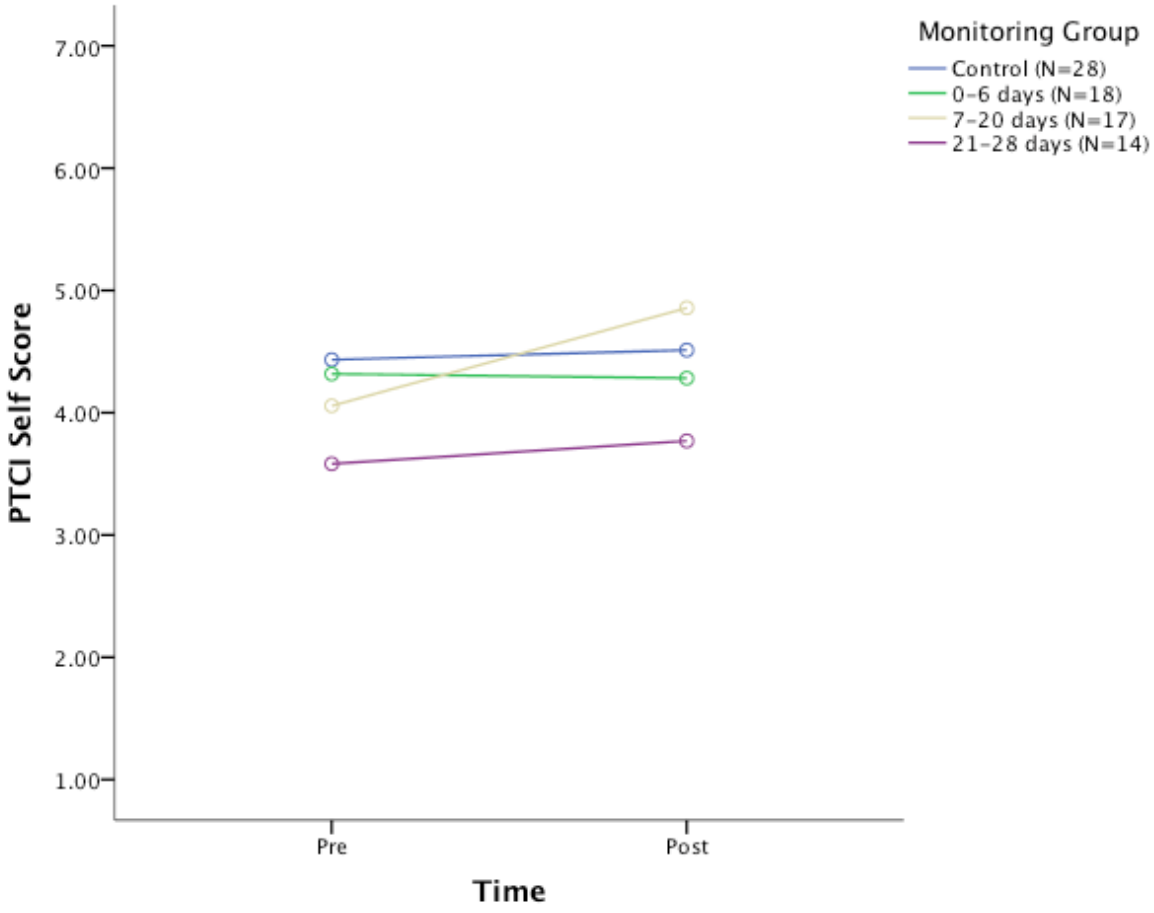
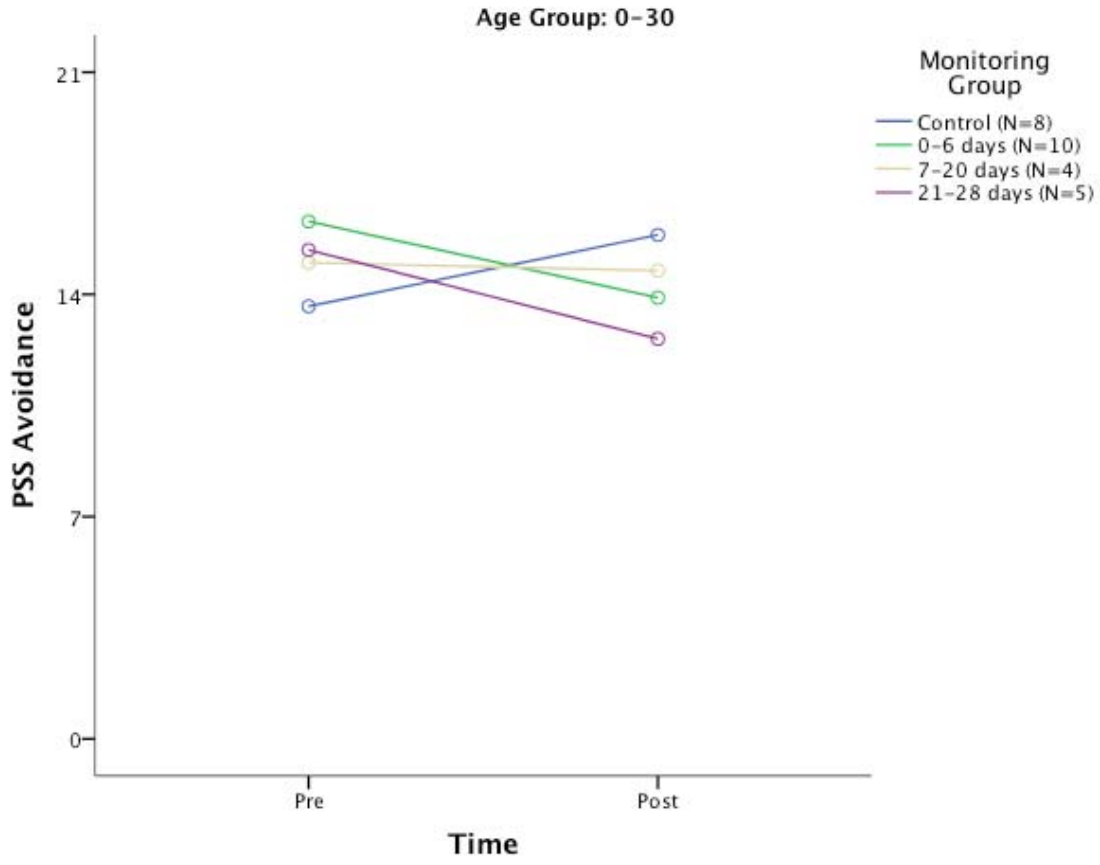


Table 14: Results of repeated measures ANOVAs conducted separately by age group, PSS Avoidance as dependent variable, monitoring compliance level as independent variable: Monitoring group*time interaction terms

	F	**p**	**Eta Squared**	**Observed Power**
PSS Avoidance				
30 and under	*3.33**	*.037**	*.303**	*.679**
31 - 44	.241	.867	.024	.090
45 and over	.941	.437	.109	.224

Figure 7: Interaction between monitoring compliance group and time among younger veterans: frequency of avoidance symptoms



Appendix A: PTSD 101 handouts

PTSD 101

Trauma Recovery Program Acute Team

Course Materials for Sessions 1-5

Group Leaders: _____
_____

Contact Numbers:
Main VA Number: _____

Leaders' Extensions:_____
_____

PTSD 101
Session 1 Agenda

- Welcome and introductions
- Review of group schedule and rules
- What is PTSD 101 about and how does it fit in with the rest of your treatment with TRP?
- What is trauma?
- Activity: common reactions to trauma
- What is PTSD?
- What causes PTSD?
- What are triggers and how do they relate to PTSD?
- Activity: breathing retraining
- Questions and homework

Goals of PTSD 101

1) To help veterans understand what PTSD is, what makes it better, and what makes it worse.

2) To help veterans understand the treatment approaches for PTSD and prepare for the next stage of treatment.

3) To help veterans begin NOW to implement behavior changes to improve PTSD symptoms.

What is Trauma?

- A trauma is an event involving actual or threatened **death** or **serious injury**, or **threat to the physical integrity of you or someone else**.

- A trauma is an event that makes you feel intensely **terrified**, **horrified**, or **helpless**.

- Common examples of traumatic events experienced by OIF/OEF combat veterans include:
  - Being shot at
  - Mortar and rocket attacks
  - Being hit by IEDs
  - Killing others in combat
  - Witnessing others badly wounded or killed in combat
  - Seeing dead bodies and body parts
  - Sexual assault

Common Reactions to Trauma

RE-EXPERIENCING SYMPTOMS

- Intrusive memories, images of trauma; flashbacks
- Nightmares
- Intense distress when reminded of trauma

INCREASED PHYSICAL AROUSAL

- Hyper-vigilance
- Exaggerated startle, jumpiness
- Irritability, anger, or rage
- Sleep problems
- Poor concentration and attention

AVOIDANCE OF TRAUMA REMINDERS

- Efforts to suppress thoughts and feelings about the trauma
- Avoidance of conversations about the trauma or related topics
- Avoidance of activities, places, or people that bring up trauma memories

NUMBNESS

- Loss of interest and/or decreased participation in important activities
- Feeling detached from others, isolated
- Emotional numbness, restricted range of feelings (e.g., can't have loving feelings)
- Loss of sex drive
- Hopelessness or diminished sense of a future life

OTHER PROBLEMS

- Overestimation of danger in the environment
- Loss of trust
- Loss of intimacy/relationship problems
- Impatience
- Over-use of alcohol or drugs
- Depression
- Feelings of guilt or shame
- Feelings of incompetence or inadequacy

Common Reactions to Trauma

A traumatic experience produces emotional shock and may cause many emotional problems. This handout describes some of the common reactions people have after a trauma. Because everyone responds differently to traumatic events, you may have some of these reactions more than others, and some you may not have at all.

Remember, many changes after a trauma are normal. In fact, most people who directly experience a major trauma have severe problems in the immediate aftermath. Many people then feel much better within three months after the event, but others recover more slowly, and some do not recover enough without help. Becoming more aware of the changes you've undergone since your trauma is the first step toward recovery.

Some of the most common problems after a trauma are described below.

1. **Fear and anxiety**. Anxiety is a common and natural response to a dangerous situation. For many people it lasts long after the trauma has ended. This happens when one's views of the world and sense of safety have changed and become more negative. You may become anxious when you remember the trauma. But sometimes anxiety may come from out of the blue. **Triggers or cues** that can cause anxiety may include places, times of day, certain smells or noises, or any situation that reminds you of the trauma. As you begin to pay more attention to the times you feel afraid, you can discover the triggers for your anxiety. In this way, you may learn that some of the out-of-the blue anxiety is really triggered by things that remind you of your trauma.

2. **Re-experiencing the trauma.** People who have been traumatized often re-experience the traumatic event. For example, you may have **unwanted thoughts** of the trauma and find yourself unable to get rid of them. Some people have **flashbacks**, or very vivid images, as if the trauma is occurring again. **Nightmares** are also common. These symptoms occur because a traumatic experience is so shocking and so different from everyday experiences that you can't fit it into what you know about the world. So in order to understand what happened, your mind keeps bringing the memory back, as if to better digest it and fit it in.

3. **Increased arousal** is also a common response to trauma. This includes feeling jumpy, jittery, and shaky; being easily startled; and having trouble concentrating or sleeping. Continuous arousal can lead to **impatience** and **irritability**, especially if you're not getting

enough sleep. The arousal reactions are due to the fight or flight response in your body. The fight or flight response is how we protect ourselves against danger, and it also occurs also in animals. When we protect ourselves from danger by fighting or running away, we need a lot more energy than usual, so our bodies pump out extra adrenaline to help us get the extra energy we need to survive. People who have been traumatized often see the world as filled with danger, so their bodies are on constant alert, always ready to respond immediately to any attack. The problem is that increased arousal is useful in truly dangerous situations, such as if we find ourselves facing a tiger. But alertness becomes very uncomfortable when it continues for a long time even in safe situations. Another reaction to danger is to **freeze**, like the deer in the headlights, and this reaction can also occur during a trauma.

4. **Avoidance** is a common way of managing trauma-related pain. The most common is avoiding situations that remind you of the trauma, such as the place where it happened. Often situations that are less directly related to the trauma are also avoided, such as going out in the evening if the trauma occurred at night. Another way to reduce discomfort is to try to push away painful thoughts and feelings. This can lead to feelings of **numbness**, where you find it difficult to have both fearful and pleasant or loving feelings. Sometimes the painful thoughts or feelings may be so intense that your mind just blocks them out altogether, and you may not remember parts of the trauma.

5. Many people who have been traumatized feel **angry** and **irritable**. If you are not used to feeling angry, this may seem scary as well. It may be especially confusing to feel angry at those who are closest to you. Sometimes people feel angry because of feeling irritable so often. Anger can also arise from a feeling that the world is not fair.

6. Trauma often leads to feelings of **guilt** and **shame**. Many people blame themselves for things they did or didn't do to survive. For example, some assault survivors believe that they should have fought off an assailant, and they blame themselves for the attack. Others feel that if they had not fought back they wouldn't have gotten hurt. You may feel ashamed because during the trauma you acted in ways that you would not otherwise have done. Sometimes, other people may blame you for the trauma. Feeling guilty about the trauma means that you are taking responsibility for what occurred. While this may make you feel somewhat more in control, it can also lead to feelings of helplessness and depression.

7. **Grief and depression** are also common reactions to trauma. This can include feeling down, sad, hopeless, or despairing. You may cry more

often. You may lose interest in people and activities you used to enjoy. You may also feel that plans you had for the future don't seem to matter anymore, or that life isn't worth living. These feelings can lead to thoughts of wishing you were dead, or doing something to hurt or try to kill yourself. Because the trauma has changed so much of how you see the world and yourself, it makes sense to feel sad and to grieve for what you lost because of the trauma.

8. **Self-image** and **views of the world** often become more negative after a trauma. You may tell yourself, "If I hadn't been so weak or stupid this wouldn't have happened to me." Many people see themselves as more negative overall after the trauma ("I am a bad person and deserved this"). It is also very common to see others more negatively and to feel that you can't **trust** anyone. If you used to think about the world as a safe place, the trauma may suddenly make you think that the world is very dangerous. If you had previous bad experiences, the trauma may convince you that the world is dangerous and others aren't to be trusted. These negative thoughts often make people feel that they have been changed completely by the trauma. Relationships with others can become tense, and it may be difficult to become intimate with people as your trust decreases.

9. **Sexual relationships** may also suffer after a traumatic experience. Many people find it difficult to feel sexual or have sexual relationships. This is especially true for those who have been sexually assaulted, since in addition to the lack of trust, sex itself is a reminder of the assault.

10. Some people increase their **use of alcohol or drugs** after a trauma. There is nothing wrong with responsible drinking, but if your use of alcohol or drugs has increased as a result of your traumatic experience, it can slow down your recovery and cause problems of its own. Many of the reactions to trauma are connected to one another. For example, a flashback may make you feel out of control and will therefore produce fear and arousal. Many people think that their common reactions to the trauma mean that they are "going crazy" or "losing it." These thoughts can make them even more fearful. Again, as you become aware of the changes you have gone through since the trauma and as you process these experiences during treatment, the symptoms should become less distressing.

What is Posttraumatic Stress Disorder?

- Posttraumatic Stress Disorder (PTSD) is an anxiety disorder that can result from exposure to trauma.
- PTSD involves four main types of symptoms:

1. **Re-experiencing** (repeatedly reliving) the trauma. This can be in the form of nightmares, intrusive memories or images, flashbacks, or intense emotional or physical reactions to reminders of the trauma.

2. Physical **hyperarousal**. This includes sleep problems, anger/irritability, concentrationproblems, always feeling on edge or on guard, jumpiness, and being easily startled.

3. **Avoidance** of trauma reminders. This may include trying not to think or talk about the trauma, or trying not to have feelings about it. It may also include staying away from activities, people, places, and situations that bring up trauma memories.

4. Emotional **numbing**. This includes losing interest in activities that used to be important to you, feeling detached or estranged from important people in your life, feeling unable to have normal emotions, and losing a sense of a future for yourself.

Some Facts about PTSD

- PTSD is diagnosed when the symptoms described on the previous page last longer than a month and cause significant distress or impairment in functioning.

- The symptoms of PTSD are often accompanied by other problems, such as depression/hopelessness, drug/alcohol abuse, relationship problems, and physical symptoms (e.g., headaches, stomach upset).

- Symptoms of PTSD may not emerge immediately after the traumatic event. Sometimes it is weeks, months, or even years before the symptoms develop.

- Not all trauma survivors develop PTSD. We do not know all the reasons why some survivors develop PTSD and others do not, but some of the factors involved include genetic vulnerability to anxiety, previous experience with trauma, presence of other life stressors, coping skills, and social support.

- In the United States, about 8% of the population will have PTSD symptoms at some point in their lives. Rates are significantly higher among combat veterans.

- PTSD is treatable. There are a number of highly effective interventions available that have been scientifically proven to markedly reduce or even eliminate the symptoms of PTSD. This is not a condition you need to live with forever. Although we cannot change history, we can change the way your history affects your life now. You can recover from your traumatic experience(s).

For more information about PTSD, talk to your mental health care provider or visit the National Center for PTSD website: http://www.ncptsd.va.gov

What Causes PTSD?

- Traumatic events affect the body's **alarm system**.

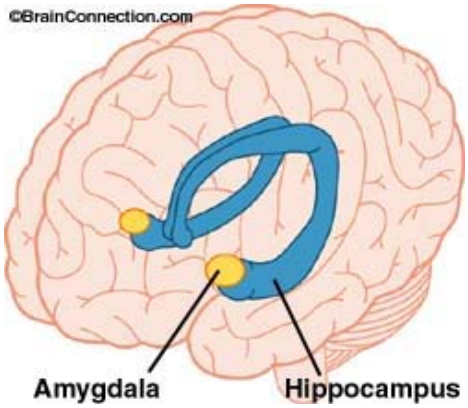
- We are equipped with an alarm system, which when activated triggers the **fight or flight response**. Heart rate and breathing rate increase, muscles tense, adrenaline rushes, etc. The body prepares you to fight off an attack or to flee (escape).

- The fight or flight response is meant to be a **short-term solution** to danger. When the danger is over, the alarm system is supposed to shut down, allowing the body to relax and return to normal.

- Traumatic events, however, can disrupt the functioning of the alarm system so that it **cannot tell when the danger is over** and does not shut down properly. You continue to feel as if the danger is present all the time, and the fight or flight response becomes somewhat **chronic**.

- How does this happen? (See the next page)

PTSD and the Brain



- Two brain structures that play an important role in PTSD are the **amygdala** and the **hippocampus**.
- The hippocampus is responsible for processing information about your life and experiences and storing it away in long term memory for later use.
- The amygdala activates the body's alarm system (the fight or flight response). When the brain perceives a threat, the amygdala becomes active and sends messages to the rest of the body to prepare for danger. The amygdala also processes **emotional memories**.
- Under normal circumstances, these regions communicate with one another and with the rest of the brain in a smooth fashion.
- However, **traumatic stress disrupts the communication between these different areas**. The logical, rational parts of your brain cannot get the message through to the amygdala that the danger is over and it's okay to relax. The hippocampus cannot take the emotional information processed by the amygdala and store it away as a long term memory. So your memories of trauma stay with you all the time and you continue to feel as if you are in constant danger.

Triggers

- A **trigger** is something that elicits feelings of anxiety, fear, anger, upset, or other types of distress.

- For most people with PTSD, triggers are things that are not inherently dangerous, but that remind them of aspects of their traumatic experiences.**The amygdala recognizes the similarity and – not realizing that the danger is over – activates the fight or flightresponse**.

- Certain sights, sounds, smells, physical sensations, places, activities, and situations can be triggers for people with PTSD, and can **produce a surge of anxiety and a strong urge to escape or avoid**.

- Common examples of triggers for OIF/OEF veterans with PTSD include:
    - loud noises
    - crowded public spaces
    - people of Middle Eastern descent
    - trash/objects in the road
    - smell of diesel fuel

- Learning to recognize your own triggers is an important aspect of treatment for PTSD. A worksheet at the back of this packet will help you start monitoring your triggers this week.

Breathing Retraining

- Learning to control your breathing is a simple way to reduce stress and tension.

- The secret is not deep breathing, but **slow** breathing.

- Try the following technique:

  1. Take a normal breath in through your nose, keeping your mouth closed.

  2. Exhale **slowly** through the nose.

  3. While you exhale, silently say to yourself the word "**calm**" or "**relax**" very slowly.
     For example: c-a-a-a-a-a-a-l-m

  4. Pause and count to 4 before you inhale again.

  5. Repeat for 10 minutes.

PTSD 101
Session 2 Agenda

- Review from last week: What is PTSD? What causes it?
- Homework review:
    - What triggers did you notice?
    - Breathing retraining homework
- New topic: avoidance and safety behaviors. What are they and how do they affect PTSD?
- Discuss in vivo ("real world") exposure and the concept of SUDS
- Choose an avoidance or safety behavior to work on this week
- Questions and homework

Review of Session 1

___1. An event that makes you feel intensely terrified, horrified, or helpless.
___2. The repeated reliving of a trauma in the form of nightmares, intrusive memories or images, flashbacks, or intense emotional or physical reactions to reminders of the trauma.
___3. This can be the loss of interest in activities that used to be important to you, feeling detached or estranged from important people in your life, feeling unable to have normal emotions, and losing a sense of a future for yourself.
___4. This includes sleep problems, anger/irritability, concentration problems, always feeling on edge or on guard, jumpiness, and being easily startled.
___5. Trying not to think or talk about the trauma, or trying not to have feelings about it. It may also include staying away from activities, people, places, and situations that bring up trauma memories.
___6. Traumatic events affect the body's _____.
___7.This response includes increased heart rate and breathing, muscle tension, and an adrenaline rush, as the body prepares you to fight off an attack or to flee (escape).
___ ;___8. Two brain structures, ___ and __, play an important role in PTSD.
___9. Something that elicits feelings of anxiety, fear, anger, upset, or other types of distress.
___10. ___is a treatable disorder.

A. Triggers
B. Alarm system
C. Hippocampus
D. Emotional numbing
E. PTSD
F. Physical hyperarousal
G. Fight or Flight response
H. Re-experiencing
J. Amygdala
I. Avoidance
K. Trauma

Review: Common Reactions to Trauma

RE-EXPERIENCING SYMPTOMS

- Intrusive memories, images of trauma; flashbacks
- Nightmares
- Intense distress when reminded of trauma

INCREASED PHYSICAL AROUSAL

- Hyper-vigilance
- Exaggerated startle, jumpiness
- Irritability, anger, or rage
- Sleep problems
- Poor concentration and attention

AVOIDANCE OF TRAUMA REMINDERS

- Efforts to suppress thoughts and feelings about the trauma
- Avoidance of conversations about the trauma or related topics
- Avoidance of activities, places, or people that bring up trauma memories

NUMBNESS

- Loss of interest and/or decreased participation in important activities
- Feeling detached from others, isolated
- Emotional numbness, restricted range of feelings (e.g., can't have loving feelings)
- Loss of sex drive
- Hopelessness or diminished sense of a future life

OTHER PROBLEMS

- Overestimation of danger in the environment
- Loss of trust
- Loss of intimacy/relationship problems
- Impatience
- Over-use of alcohol or drugs
- Depression
- Feelings of guilt or shame
- Feelings of incompetence or inadequacy

Avoidance and Safety Behaviors

- When an activity, place, thing, or situation makes you uncomfortable, a natural impulse is to avoid it altogether or escape from it as quickly as possible.
- When you have PTSD, **behavior often changes as you try to avoid the triggers in your environment**.
- Common examples of avoidance behaviors for OIF/OEF veterans with PTSD include:
    - Staying away from malls, movie theaters, sports arenas, and other crowded public spaces.
    - Refusing to ride in the passenger seat.
    - Shopping late at night to avoid people.
    - Not answering the phone.

- It is also common to develop **safety behaviors**: rituals and habits intended to reduce distress.

- Common examples of safety behaviors for OIF/OEF veterans with PTSD include:
    - Always sitting with a wall at your back.
    - Constant visual scanning for threat.
    - Carrying a weapon.
    - Checking locks on doors and windows repeatedly.
    - Patrolling the perimeter of your home.

- Learning to recognize your avoidance and safety behaviors is an important aspect of treatment for PTSD. A worksheet at the back of this packet will help you start monitoring these behaviors this week.

Why Are Triggers, Avoidance, and Safety Behaviors Important?

- Trying to avoid things that make you anxious or uncomfortable or protect yourself with safety behaviors is only natural. Unfortunately, when it comes to your triggers **avoidance doesn't work**. It may reduce your distress for a little while, but in the long run **avoidance makes PTSD worse**.

- Why? Because most of the time triggers are things that are not inherently dangerous. They feel dangerous, but they aren't dangerous. But when you avoid those triggers, you **never get to learn that they are actually safe**. The amygdala continues to label them as associated with trauma and they continue to have the power to produce fear. In fact, the fear can grow over time.

- Avoidance of all triggers also leads to **isolation**, which can contribute to **depression** and **relationship problems**.

- Learning to **overcome the urge to escape/avoid** and to **face your triggers directly** without relying on safety behaviors is what makes PTSD better.

Overcoming Avoidance through In Vivo ("Real World") Exposure

- When you have PTSD, your brain's alarm system still operates as if you are in constant danger, the way you were in the combat zone. It sends out many **false alarms** giving you feelings of intense danger even in safe situations.

- Since avoidance of all situations that trigger distress makes PTSD worse over time (and leads to isolation), the solution is to **face the feared situations** instead.

- In **in vivo ("real world") exposure**, you intentionally place yourself in situations that are outside of your comfort zone and **stay** there until your brain's alarm system begins to learn that nothing bad will happen and you begin to feel more comfortable.

- Unlearning fear takes a lot more time and practice than learning it. So in real world exposure you **repeatedly** visit the feared situations for a **prolonged period of time**.

- You also stop using **safety behaviors** in feared situations. Safety behaviors prevent the alarm system from learning that the new situations aren't dangerous. If you rely on safety behaviors, your alarm system will always think they are the only thing keeping you from harm. You will never feel really comfortable because the situation will never feel as safe as it actually is.

Example of In Vivo Exposure

- Many combat veterans with PTSD experience high anxiety in crowded public spaces like shopping malls. Their alarm systems feel that such places are likely to be "targets" for deadly attacks. In such spaces, they may utilize the safety behavior of constant visual scanning for threat.

- In vivo exposure can help reduce the distress experienced in a crowded mall. By confronting the feared situation **repeatedly** and for a **prolonged period of time**, the alarm system begins to recalibrate as nothing terrible happens.

- For example, one might start by sitting on a bench in an out-of-the-way hall in the mall and refrain from scanning by focusing on a magazine. After that starts to feel more comfortable, one could work up to sitting in busier areas like a main hallway, or the food court.

- If in vivo exposure is practiced repeatedly and for enough time, **anxiety always comes down**. You habituate to the new situation and it doesn't feel threatening any more.

SUDS

(Subjective Units of Distress)

SUDS ratings are a way of communicating the level of distress you feel. The term "distress" is intentionally very broad, so it can refer to feeling anxious, angry, scared, upset, jumpy, or any other negative emotional state.

We use a scale from 0 to 100.

0 represents no distress at all (i.e., completely calm, relaxed).

100 represents very extreme distress, fear, or anxiety -- the most upset you have ever been in your life. Usually when people say they have a SUDS of 100 they are experiencing physical reactions (e.g., sweating, heart pounding, trouble breathing, dizziness) as well as intense emotional distress.

Using SUDS ratings is a good way for you to notice when your distress is going up and when it is going down. Distress related to fear and anxiety always comes down eventually.

0----------25----------50----------75----------100

No Distress	Moderate Distress	Maximum Distress

SUDS ratings are subjective. A situation that makes one person feel 100 SUDS may make another person feel 0 SUDS. It will be helpful to identify some situations that correspond with different SUDS ratings for you.

SUDs Anchor Points:

0 – _____

50 – _____

100 – _____

In order to work, in vivo exposure must:

- Be repeated (practice daily)
- Be prolonged (stay for 90 minutes or until your SUDS drops by 50%)
- Involve self-exposure to mildly and moderately anxiety-provoking situations
- Start low – set yourself up for success!

PTSD 101
Session 3 Agenda

- Interactive activity: what have we learned so far?

- Homework Review:
  - What additional triggers did you notice this week?
  - What about avoidance and safety behaviors?
  - In Vivo Exposure
    - How did it go?
    - Plan goals for next week.

- Sleep hygiene: How to improve your sleep

- Questions and homework

Sleep Hygiene

Many people with PTSD have trouble sleeping. This can be due to hyperarousal and being too "revved up." Some people with PTSD feel they must be on guard all the time, even at night. It takes time to re-learn that sleep here is safe. Lack of sleep makes a person more irritable, anxious, and depressed. Restoring sleep makes a person feel better and function better in life. Below are some common sense strategies for improving the quality of sleep. You can also talk to your doctor about medications to help you sleep.

BIG IDEA: Associate your bedroom with sleep

- Do not go to bed until you are sleepy.
  If you are not sleepy at bedtime, do something else. Read a book, listen to music, browse through a magazine, practice your breathing exercises. Find something relaxing, not stimulating, to take your mind off worries about sleep.
- If you are not asleep after 20 minutes, get out of bed.
  Find something else to do that will make you feel relaxed. If you can, do this in another room. Your bedroom should be where you go to sleep, not a place to go when you are bored. Once you feel sleepy again, go back to bed.
- Use your bed for sleeping and sex only.
  Don't read, write, eat, watch TV, talk on the phone, or play cards in bed

BIG IDEA: Get yourself on a schedule

- Get up at the same time every morning.
  Do this even on weekends and holidays.
- Avoid taking naps.
  Some people nap during the day because they feel safer. This may interfere with your ability to get a good night's sleep.
- Keep a regular routine.

Regular times for meals, medications, chores, and other activities help keep the body's inner clock running smoothly.

BIG IDEA: Eliminate things that will interfere with sleep

- Do not use alcohol as a sleep aid.
  Alcohol disrupts your sleep cycle, making it less restorative.
- Avoid caffeine completely.
- Do not smoke or use any tobacco product in the evening.
  Like caffeine, nicotine is a stimulant and can interfere with sleep.
- Don't go to bed hungry, but don't eat a big meal near bedtime either.
- Avoid exercise within 6 hours of your bedtime.
  You should exercise on a regular basis, but do it earlier in the day. Talk to your doctor before you begin an exercise program.
- Try to get rid of or deal with things that make you worry.
  If you are unable to do this, then find a time during the day to get all of your worries out of your system. Write them down on a "worry list," or give yourself a designated "worry hour" to confront them. Your bed is a place to rest, not a place to worry.

BIG IDEA: Foster sleep-promoting behaviors

- Begin rituals to help you relax each night BEFORE bed.
  This can include such things as a warm bath, light snack, or a few minutes of reading.
- Keep your bedroom quiet, dark, and a little cool.
  Remember, it is your sanctuary!

Consider Your Attitude toward Sleep

- Thoughts can have powerful effects on your emotions and on your body.
- Many combat veterans have negative attitudes about sleep. Many fear that if they sleep, it means that they are not protecting their families, that they are weak or vulnerable, or that they will have nightmares about traumatic events. This can make them fear or avoid sleeping, even though their bodies need sleep badly!
- Even if they don't fear or deliberately avoid sleep, many people with insomnia worry a lot about their sleep difficulties.
- Negative attitudes and worry about sleep, and about other situations and issues in your life, only increase stress and make sleeping more difficult and less restorative.
- In contrast, people who have stress-reducing attitudes and beliefs tend to sleep better. If you can change your thinking, it may help reduce your sleep problems.

Ask yourself:

What negative things do I tell myself about sleep?

What more positive thoughts could I tell myself instead?

PTSD 101
Session 4 Agenda

- Review: What we know so far.

- Homework Review:
  - What triggers, avoidance and safety behaviors did you notice this week?
  - In vivo exposure: How did it go?
  - Sleep hygiene: What sleep behaviors did you change? How did it go?

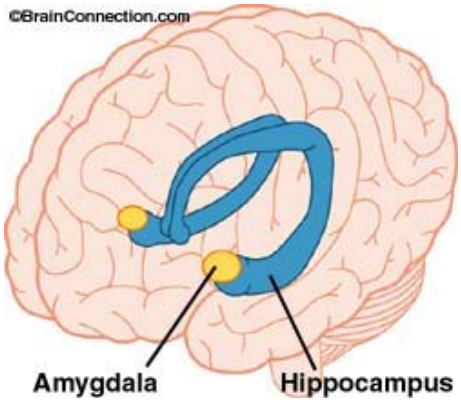
- Treatment for PTSD:
  - Exposure therapy: what is it and why does it work?
  - What do you actually do in therapy?

- Video explaining treatment
  - Note: this video includes some brief footage from OIF. No combat is depicted.

- Discuss video

What We Know So Far

- The brain's alarm system has taken over. It overrides reason and logic and functions as if you are still in great danger.



- Since your brain's alarm system is convinced you are still in danger, it will sound the alarm and send you waves of fear, anxiety, anger, or general distress whenever it encounters something even remotely similar to aspects of the traumatic events you have experienced in the past.

- These false alarms create the urge to avoid or escape the places, activities, people, and things that seem to trigger them.

- BUT, avoidance and the use of safety behaviors rob the brain of the chance to learn that you are in a different place, a different time, and less danger now.

What Works and What Doesn't

- Avoidance, escape, and the use of safety behaviors may make you feel better in the moment, but in the long run they prevent PTSD from getting better. That is because they prevent your brain's alarm system from learning what is really dangerous and what isn't.

- In fact, over time the use of avoidance, escape, and safety behaviors can increase fear, irritability, and distress.

- On the other hand, repeated exposure to the memories and real-life situations that the brain fears (while resisting the use of safety behaviors) makes PTSD better, because it allows the alarm system to learn.

- Exposure is best done by "starting low and going slow."

- You must repeat the same exposure exercise many times for it to work. The alarm system is stubborn!

Treatment of PTSD

- The treatment that has been consistently found to work for PTSD is called **exposure therapy**.

- Exposure therapy is a type of psychotherapy that teaches you how to **stop using avoidance** as a coping strategy for dealing with trauma.

- Exposure therapy works by:
  - Helping you get back out in the world without anxiety (i.e., "exposing" you to the places and activities of everyday life).
  - Helping you face and process the memories of your traumatic experiences (i.e., "exposing" you to the memories).

Revisiting Traumatic Memories: Imaginal Exposure

- It is natural to want to avoid thinking about traumatic memories, but many people find it is almost impossible to get over them without facing them.

- It is important to revisit traumatic memories, so that you can really process them in a way that you couldn't at the time that the trauma occurred.

- Imaginal exposure is a therapeutic method in which you repeatedly revisit a traumatic memory in detail.

- By allowing yourself to remember, you give yourself a chance to:
  - Allow the emotions associated with your traumatic memory to run their course.
  - Organize your memory and get a new perspective on it.
  - Realize the difference between remembering the event and reliving it.
  - Realize that the distress tied to the memory will fade with repeated exposure.
- Build your own sense of competence and confidence by learning you can handle it.

Active Treatment Options

Individual Trauma-Focused Therapy	Emory Virtual Reality Treatment
Approx. 12-15 individual sessions	6 Sessions + Medications (Placebo vs. D-cycloserine vs. Alprazolam)
60-90 min sessions	Emory Research study
Individual attention	Brief Format [6 sessions]
Homework weekly	No homework
Modified to fit individual needs	Flexible scheduling available
Appointments available any day [within normal business hours]	Recreates combat environment using VR technology
Must commit to consistent day/time for appointments	Additional treatment may be needed
	Compensation is provided

PTSD 101
Session 5 Agenda

- Review:
  - What have we learned about PTSD?
  - What is exposure therapy and how does it work?

- Homework review:
  - What triggers, avoidance, and safety behaviors did you notice this week?
  - In vivo exposure: How did it go?
  - What sleep hygiene techniques did you put into practice?
  - What questions do you have about exposure therapy?

- The role of medications in treating PTSD

- Stages of treatment: What's next for you?

- Feedback about the class

- Completion of questionnaires

The Role of Medication in Treating PTSD

- There is no one medication that can treat all the symptoms of PTSD. However, there are many medicines that can help relieve some of the symptoms.

- For example, antidepressants, mood stabilizers, antipsychotics, and sleep medications can all play a role in the treatment of PTSD.

- Medicine can help by improving sleep, reducing nightmares, reducing anxiety and irritability, and improving mood, among other ways.

- The importance of taking medications depends on a number of individual factors, including the presence of other mental health problems (e.g., depression) in addition to PTSD.

- The medicines prescribed by the TRP psychiatrists are not addictive and will not interfere with the effectiveness of therapy. In fact, they can help you get the most out of treatment.

- Only your psychiatrist can give you guidance on whether you need medication, and on what kind and how much you should take.

- Never start a new medicine, change your dose, or stop taking a medication without consulting your psychiatrist first. Do not try medicines prescribed for your friends or family members – they may not be right for you!

What Happens Next?

Trauma Recovery Program
Stages of Recovery

STAGE 1: LEARN, COPE

- **PTSD 101**
- Medication management (for some)

STAGE 2: HEAL

- Trauma-focused therapy
  - Individual trauma-focused therapy
  - Virtual Reality study at Emory
- Continued medication management (if applicable)

STAGE 3: HEALTHY LIVING

- Continue applying lessons from treatment independently

Review: Goals of PTSD 101

1) Help veterans to understand what PTSD is, what makes it better, and what makes it worse.

2) Help veterans to understand the treatment approaches for PTSD and to prepare for the next stage of treatment.

3) Help veterans begin NOW to implement behavior changes to improve PTSD symptoms.

Appendix B: Intrusion monitoring instructions and forms

PTSD 101 Self-monitoring Instructions - Clinician

The final part of your homework is something that you'll be asked to do each week for the duration of this class. We talked earlier about re-experiencing symptoms like nightmares, flashbacks, and intrusive memories. Over the next four weeks, you're going to be keeping track of those symptoms by filling out this form (hold up form) each night. The different things you're going to monitor are:

Nightmares, which are distressing dreams about the traumatic event or events you experienced. Nightmares may be directly or indirectly related to the event itself. For example, you might dream about a specific firefight, or you might dream about war more generally.

Flashbacks, which are memories so vivid that they seem to be really happening again. You may act or feel as though you are back in the traumatic situation.

Unwanted thoughts/memories/images, which are things that come into your mind unexpectedly or intrusively. These memories, thoughts, or images may be triggered by things like people, places, smells, or internal feelings, or they might seem to come to you completely out of the blue.

Physical and emotional reactions to trauma reminders, which are things that happen in your mind or in your body when you are confronted with a reminder of the traumatic event or events you experienced. You might have physical symptoms, like feeling your heart pounding or racing, feeling lightheaded or dizzy, getting sweaty palms, or having shortness of breath. You might also (or instead) have strong feelings like anger, sadness, fear, or horror when you are confronted with a reminder of the trauma.

The front page of your packet of forms has all of these definitions listed for you. You should complete one form every day, right before going to sleep. It shouldn't take more than 5 minutes to complete. Try to make it a part of your nightly routine so that you don't forget any days – it's really important that you fill one out every day and bring the packet back with you each time we meet.

We've also included an example sheet in your packet to give you a sense of how to fill out the forms. There are 4 different types of symptoms that you'll be tracking – for each one, you'll write down how many different times you had that experience during the previous 24 hours. If you don't remember an exact number, just give your best estimate. You'll then rate how distressed you were by those experiences, on average, with 0 being not distressed at all and 100 being the most distressed you could imagine being. Then you'll write in what triggers were associated with the experience. Finally, you'll write a brief description of the content of the experiences. Try to be as specific as possible – the more detail the better. You can always use the back of the form to write more if you run out of space.

Any questions?

HOMEWORK: Intrusion monitoring

How to use these forms:

-There are 7 recording sheets in this packet. You will use **one each day.**

-You'll be writing about 4 different types of experiences:

1. **Nightmares** – Distressing dreams related to the trauma you experienced
2. **Flashbacks** – Memories associated with the trauma that are so vivid they seem to be really happening again
3. **Unwanted thoughts, memories, and images** – Things related to the trauma you experienced that come into your mind NOT at a time when you are trying to think about those things
4. **Physical and emotional reactions to trauma reminders** – Feelings in your body or emotions that occur when you are confronted by something that reminds you of the trauma

-Please complete the forms at the **same time each day**. Think back over the previous day and fill in the spaces on the form relating to each type of symptom.

-Under the word "Triggers," write down things you saw, heard, or thought about that might have brought on the experience you had.

-Under the word "Content," write down a brief summary of what you experienced. Try to give as much detail as possible. If you need more space, feel free to write on the back of the packet.

-Use the example form on the back of this sheet as a guide.

-Feel free to ask your class instructor if you have any questions!

Name: _____ Last 4: _____

Date: _____ Clinician's name: _____

Take some time to think back over the previous day. Try to remember each time you were reminded of the traumatic event(s) you experienced and rate the following:

Nightmares/Bad dreams

How many? 2

Distress (0-100): 85

Triggers:	Content:
Raining outside	1. The roof flew off the house, and I was left stranded and alone. 2. Water was rushing into my bedroom and covering me in my bed, and I couldn't breathe. I woke up in a cold sweat

Flashbacks

How many? 1

Distress (0-100): 90

Triggers:	Content:
Thunder and lightning	As soon as I heard the thunder, I ducked for cover under the bed. It felt exactly like the night of the hurricane.

Unwanted thoughts/memories/images

How many? 5

Distress (0-100): 90

Triggers:	Content:
Sound of rain	1. "I'm going to drown"
Thunder and lightning	2. "I'm not safe in the house"
Tree limb falling out back	3. "I'm not safe anywhere"
	4 & 5. "It's happening again"

Physical and emotional reactions to triggers

How many? 9

Distress (0-100): 95

Triggers:	Content:
Watching the Weather Channel	Panic, anxiety, anger
Sounds of the storm	Heart racing, sweaty palms, shortness of breath, lightheaded, dizzy, feels unreal

Name: _____ Last 4: _____

Date: _____ Clinician's name: _____

Take some time to think back over the previous day. Try to remember each time you were reminded of the traumatic event(s) you experienced and rate the following:

Nightmares/Bad dreams

How many? _____

Distress (0-100): _____

Triggers: Content:

Flashbacks

How many? _____

Distress (0-100): _____

Triggers: Content:

Unwanted thoughts/memories/images

How many? _____

Distress (0-100): _____

Triggers: Content:

Sound of rain

Physical and emotional reactions to triggers

How many? _____

Distress (0-100): _____

Triggers: Content: