PTSD: Extinction, Reconsolidation and the Visual-Kinesthetic Dissociation Protocol

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Abstract. Every year thousands of returning military, state and local police officers and civilians of every description suffer from the intrusive symptoms of PTSD. Current treatments rooted largely in extinction protocols require extensive commitments of time and money and are often ineffective. This study reviews several theories of PTSD, and two important mechanisms that explain when treatment does and doesn't work: extinction and reconsolidation. It then reviews the research about and suggests an explanatory mechanism for the Visual-Kinesthetic Dissociation Protocol, also known as the Rewind Technique. The technique is notable for its lack of discomfort to the patient, the possibility of being executed as a content-free intervention, its speed of operation and its long term, if largely anecdotal, efficacy. A case study, specific diagnostics for extinction and reconsolidative mechanisms and suggestions for future research are provided.

Keywords: PTSD, PTSD treatment, PTSD models, Visual-Kinesthetic Dissociation, Rewind technique, extinction, reconsolidation

Although recognized in various forms from the time of Homer's Iliad to the present, the symptoms of Post-Traumatic Stress Disorder (PTSD) were first designated as a distinct psychiatric diagnosis with the publication of DSM-III in 1980 (Bodkin, Alexander, Detke, Pope, & Hudson, 2007; Davidson, & Foa, 1991; Foa, & Meadows, 1997; Friedhelm & Sack, 2002; McHugh, & Treisman, 2007; Spitzer, Wakefield, & First, 2007).

PTSD is defined by the DSM-IV in terms of five criteria. The first criterion is the traumatizing event. In order to qualify as a traumatizing event both of the following must appear: 1. the

person experienced, witnessed, or was otherwise confronted with one or more events that actually involved or threatened death, serious injury or some other threat to the physical integrity of that individual or others. 2. The individual's response involved intense feelings of horror, fear or helplessness.

The diagnostic criteria are further divided into three symptom clusters, the re-experiencing cluster, the avoidance / numbing cluster and the arousal cluster. According to Foa & Meadows (1997) the intrusion or re-experiencing symptoms include the hallmark signs of PTSD including nightmares, intrusive thoughts and flashbacks. The avoidance group includes efforts to avoid memories of the traumatic experience and symptoms of emotional numbing. The third symptom cluster includes symptoms of hyper-arousal including sleeplessness, irritability and hyper-vigilance. Other criteria include the impact and duration of the symptoms (American Psychiatric Association (APA), 1994).

Diagnosis is made when symptoms (at least one from the re-experiencing cluster, three from the avoidance cluster and two from the hyperarousal cluster) cause clinically significant distress or discomfort and have persisted for a minimum of one month (APA, 1994).

The prevalence of PTSD among veterans of the Gulf War, Operation Iraqi Freedom and Operation Enduring Freedom has been estimated at nearly twice the rate of the non-combat population (6%). For Gulf War veterans this figure lies at 10 to 11 percent and for veterans of the Iraqi and Afghanistani theaters, 13 to 17 percent (Gradus, 2010; Hogue, et al. 2008). These returning servicemen present a serious problem for treatment professionals on two fronts. First, they represent a significant influx of problem patients and second, because the currently approved treatments provide inconsistent results with varying levels of effectiveness and subsequent relapse. Another problem is that extinction based treatments, which have enjoyed a favorable position in the treatment literature, often require long term commitments in spite of their inconsistent results (Foa, Keane, & Friedman, 2000; Foa & Meadows, 1997a, 1997b; Massad & Hulsey, 2006; McNally, 2007; Rothbaum, Davis, King, Ferris, & Lederhendler, 2003; Rothbaum, Meadows, Resick, & Foy, 2000; Shalev, Bonne, & Eth, 1996; Wessa & Flor, 2007; Schiller, Monfils, et al. 2010; Ursano, et al., 2004). This paper introduces and suggests a

mechanism for a relatively unknown, short-term treatment for PTSD, The Visual-Kinesthetic Dissociation Protocol (V/KD).

The Visual/Kinesthetic Dissociation (V/KD) Protocol (as discussed below) is an intervention originally designed in the early 1980s for use with phobias. The protocol is one of many anecdotally supported interventions that emerges from the field of Neuro-Lingustic Programming (NLP). NLP is a controversial approach to modeling, replicating and transforming behavior that includes a number of specific therapeutic techniques; among these is the V/KD Protocol. NLP finds its roots in models of the therapeutic techniques of Milton Erickson, Virginia Satir, and Fritz Perls. NLP has generated a great deal of anecdotal evidence of effectiveness but has undergone little empirical testing. There have been recent calls from within the NLP Community for more research into its efficacy claims (Bandler & Grinder, 1975, 1979; Bolstad, 2002; Bostic St. Clair & Grinder, 2002; Dilts, Grinder, Bandler & DeLozier, 1980; Wake, 2008).

Theoretical Models of PTSD

Classical Learning Theory

There are multiple theories of the mechanisms of PTSD. The earlier theories were rooted in basic behavioral psychology. Consistent with classical conditioning paradigms, the traumatic memory is associated with implicit and explicit cues which evoke the memory outside of the conscious control of the sufferer. Implicit memory effects related to amygdalar function and conditioned evocation explain anxiety effects and attentional effects. Implicit and explicit conditioned associations to flashbulb memories explain certain aspects of the re-experiencing facets. Secondary effects are attributed to operant conditioning of efforts to avoid continuing trauma. According to Wessa and Flor (2007), aversive learning in PTSD is extremely resistant to extinction because it is supported by a network of second-order conditionings that maintain behavioral strength in spite of the absence of the original unconditioned trauma. There is also evidence of impaired responsivity of the Ventro-medial Prefrontal Cortex (VmPFC), a crucial mediator of extinction-based learning and modulation of amygdalar responses (Layton & Krekorian, 2002; Foa, Keane, & Friedman, 2000, Foa & Meadows, 1997; Rescorla, 1988; Diamond, Campbell, Park, Halonen, & Zoladz, 2007; Gharakhani, Mathew, & Charney, 2006; Liberzon, Sripada, & De Kloet, 2007).

The Bio Informational Theory

A bio-informational theory set forth by Foa and her colleagues (Foa, Keane, & Friedman, 2000; Foa & Kozak, 1986; Foa & Meadows, 1997), sees emotions as information networks that are presumed to be stored in long-term memory as both a set of propositions about the circumstance and a set of motor programs in response to the circumstance. When a person experiences an emotion they evoke three levels of information: information about the stimulus and its meaning in a given context, information about the appropriate response to the stimulus (emotional language, appropriate motor responses and the autonomic responses that support them), and information about the relationships between the stimulus and possible responses. For fearful responses, the networks tend to hold together as consistent patterns of response, they are easily activated — even by partial stimulus representations — and whether or not they result in overt action, there is always a characteristic autonomic response.

According to this model, treatment of PTSD, or other fear-based pathologies is dependent upon two elements: (1) Elicitation of the fearful experience as fully as possible; and (2) Modification of the memory structure to include information (experiences) that transform the meaning of the memory structure. The PTSD response is generally linked to multiple stimuli and is more easily evoked than other fearful responses. In order to ensure effective treatment, care must be taken to ensure that the fear response is evoked as fully as possible. In transforming the memory structure at the root of PTSD symptoms, new information that is incompatible with some or all of its pathological structure must be incorporated into the memory schema itself (Foa, Keane, & Friedman, 2000; Foa and Kozak, 1986; Foa & Meadows, 1997).

Dual Representational Theory

More recently, behavioral explanations have given way to explanations rooted in cognitive theory that involve levels of conscious control and an hypothesized need to integrate the memory of the trauma into a coherent life narrative. Among these is the Dual Representation theory of Brewin and Dalgleish (Brewin, Dalgleish, & Joseph, 1996).

Brewin and Dalgleish hold that traumatic memories are stored in two separate forms, explicit verbally accessible memories, and implicit situationally accessible memories. Each type of memory is stored in a different manner and in a different brain system. PTSD occurs when

traumatic events produce incomplete or inconsistent records in the two kinds of memory store, and when those memories prove to be incompatible with the existing schemas and beliefs that define the patient's world. Healing proceeds through two stages as the client moves to 1) Integrate the explicit narrative of the traumatic event with their beliefs and expectations (as recommended by social-cognitive theories); and 2) Make the implicit experience explicit by re-experiencing and coming to grips with the non-verbal traumatic experience (as recommended by information processing theories). Accordingly, healing is never complete until both kinds of processing are accomplished.

Many of these theories are unsatisfactory insofar as they tend to rely upon top down processes and ideas of integration rather than a direct assault on the emotional core of the problem. In a 2006 review of progress in the neurobiology of PTSD, van der Kolk (2006) notes that emotions are seldom under conscious control. He concludes that they are much more often in control of us than we are of them.

It is instructive that, in line with root behavioral principles, Foa et al. (1989), Brewin et al. (1996), and others have pointed to the need to direct our attention to the core facet of the problem, the traumatic memory itself.

The Temporal Dynamics Model of Emotional Memory Processing

A model which appears to specify all of the mechanisms involved in the basic phenomenology of PTSD at the level of physiology, is set forth by Diamond, Campbell, Park, et al. (2007) in their exhaustive review, *The Temporal Dynamics Model of Emotional Memory Processing: A Synthesis on the Neurobiological Basis of Stress-Induced Amnesia, Flashbulb and Traumatic Memories, and the Yerkes-Dodson Law*. According to these authors, the phenomenology of PTSD is explained by temporally sequenced patterns of long-term potentiation (LTP) and long-term depression (LTD) in the hippocampus, amygdala and prefrontal cortex (PFC) as part of a generalized response to stress.

Long-term potentiation is the (generally assumed) process whereby short-term memory is converted into long-term memory. It includes protein synthesis and physical changes in the structure of neurons at the synapse. Long-term depression refers to processes that inhibit the creation of memories more generally and subsumes a more diffuse set of phenomena.

At the center of the theory is the observation that intense fear or stress sets in motion a period of enhanced LTP in both the amygdala and the hippocampus that is followed shortly by a refractory period in which these structures only continue processing with some difficulty. Like the dual factor model of Brewin, Dalgleish and Joseph (Brewin et al., 1996) and Layton and Krikorian's appeal to amygdalar and hippocampal mechanisms (Layton & Krikorian, 2002), Diamond et al. appeal to activation of the amygdala to explain the implicit aspects of the fear memory and the sequential activation and partial suppression of hippocampal function to explain the flashbulb aspects of traumatic memory. Crucially, their theory depends upon LTP as the signal that the structure is *currently responding to stressors* and focuses on temporal sequencing as key to the observed effects.

Diamond et al. provide extensive support for the idea that under conditions of extreme stress, which create high levels of amygdalar activation, the hippocampus switches from its higher order function as integrator of time, place and sequence to a simpler focus on the immediate context. Stress initiates a brief period of facilitated LTP which is followed by a longer refractory period in which the structure may be thought of as having limited response potentialities. This refractory period isolates the memory and creates a near indelible trace of the dangerous circumstance that is not subject to interference from subsequent stimuli (in the immediate context) and may be useful for protecting the organism from similar threats in the future.

This activation of the hippocampus is short-lived and does not produce a coherent narrative of the event but only the sharply focused memory of the trauma or its precursors. These are the flashbulb memories that accompany trauma and that are often characterized by what Loftus has called gun focus, and others have described as tunnel vision. It is important to note, however, that in the present formulation, the hippocampus enters a phase of lessened efficiency rather than total inactivation. The continued lowered sensitivity of the hippocampus explains the disorientation, amnesia and the perception that the event is not connected in time and not susceptible of integration into the continuing narrative of the patient's life.

At the same time that the hippocampus is recording what will become the fragmented flashbulb memories (vivid, eidetic representations of the memory formed under periods of great emotional impact) of the incident (driven by amygdalar afferents), the amygdala is also in a phase of enhanced LTP. In this case, the amygdala, the center of emotion, creates long-lasting and extinction-resistant behavioral connections between the emotional response and the cues surrounding the event. These connections become the root of the flashbacks, nightmares and phobic aspects of PTSD. The event is encoded as a verbally inaccessible set of conditioned

responses. Like the hippocampus, the amygdala enters a refractory period after the initial period of enhanced LTP. The length of activation for this implicit conditioning phase appears to last somewhat longer for the amygdala. In this case, the amygdalar deactivation would explain numbness, fugue states, and dissociations in the immediate aftermath of the event.

Unlike the amygdala and the hippocampus, the pre-frontal cortex (PFC) — which normally modulates amygdalar function — is not subject to enhanced LTP under the effects of extreme stress, and almost immediately falls into a state of Long-Term Depression (LTD). Insofar as it is no longer capable of modulating amygdalar function, the amygdala becomes the driving force in the current state of the organism. Moreover, because the PFC is the center of executive function and is responsible for most higher-level functions including evaluations, decision making, and divided attention tasks, when it enters a state of lessened responsivity, the capacity for creating a coherent narrative of the stressful circumstance is lost.

Further, because the LTP in the amygdala may occur during the refractory phase of hippocampal function, both the PFC and the hippocampal editing and organizing functions will be impaired, resulting in emotionally driven but often inaccurate memories of the circumstances. These disordered recollections of the event -- as a result of stress related inhibition of the integrative functions of the PFC and the hippocampus — may later give rise to fabricated memories, rationalizations, and various psychological and psychiatric responses that contribute to the inability to make sense of the traumatic event.

With the caveat that the model provided by Diamond et al. is based on animal studies, and some of the mechanisms have not been subjected to extensive confirmation, this theory may serve as a foundation for understanding the root mechanisms of PTSD, and to make useful differentiations between the multiple dimensions of the problem. At the heart of the problem are the intrusion or re-experiencing symptoms including flashbacks, intrusive thoughts and nightmares. These appear to be rooted in the emotional memories as both implicit and explicit elements that are fully explained by the mechanism just described. These may be understood as the core of the disorder and for the most part they are the aversive stimuli which drive the next group, the avoidance cluster.

Within the avoidance cluster are found efforts to avoid memories of the traumatic experience and symptoms of emotional numbing. Note that numbing may not be fully explained by operant processes, it may arise as the result of habituation to the more intense stimulation of the traumatic eruptions.

The third symptom cluster arises as a direct expression of implicit memory effects mediated by the amygdala in the absence of PFC and hippocampal regulation. They include sleeplessness, irritability and hypervigilance (APA, 1994; Davidson, & Foa, 1991; Diamond et al., 2007; Foa & Meadows, 1997; Keane, Weathers & Foa, 2000; Lamprecht, Friedhelm & Sack, 2002; McHugh, & Treisman, 2007; Spitzer, Wakefield, & First, 2007).

A second layer of emotional responses often characterizes PTSD patients. These include sadness, anger, fear for the future, and an inability to make plans. These are characterized by Brewer, Dalgleish, and Joseph (1996) as secondary emotions. Depending upon the level to which they have become integrated into the life schemas and personal definitions of the individual, they may or may not be resolved with the elimination of the core symptoms. On a final level are depression, family dysfunction, and substance abuse issues. These and other co-morbidities will generally require treatment independent of the central issue, the primary symptoms of PTSD.

Extinction and Reconsolidation Effects in the Treatment of PTSD

If PTSD is understood as being, at heart, a group of behaviors associated with a classically conditioned fear response, then neural models of learning and forgetting become crucial components of our understanding. Two specific mechanisms appear to be most important theoretical models for the treatment of PTSD. These are extinction and reconsolidation.

Extinction

When the memory linkage between a Conditioned Stimulus (CS) and a fear evoking event (UCS) is extinguished, a new memory is created that communicates the absence of the feared object and blocks access to the original memory that signaled the onset of the feared event. These new memories tend to be context sensitive and somewhat more fragile than the original memories. Extinction, therefore, in the classical learning paradigm, refers to the learning of new information about the changed learning context as it is now provided by the conditioned stimulus (CS). It does not refer to the elimination, forgetting or modification of the memory. Extinction models in the treatment of PTSD are characterized by four specific effects through

which the behavior may be re-established or through which relapse occurs. As they appear in the post-treatment or relapse behavior of PTSD patients, they may be viewed as diagnostic of the fact that extinction is the specific mechanism underlying the treatment. These effects are spontaneous recovery, contextual renewal, reinstatement, and rapid reacquisition (Bouton, 2004; Bouton, & Moody, 2004; Dillon & Pizzagalli, 2007; Hartley & Phelps, 2009; Massad & Hulsey, 2006; Quirk & Mueller, 2007; Rescorla, 1988; Vervliet, 2008).

Spontaneous recovery refers to the reoccurrence of the extinguished or unreinforced fear response after the passage of time. It was first observed by Pavlov and is one of the first evidences that extinction does not remove the memory. As noted, extinction involves the creation of a new contextual association to the effect that in this context, the CS does not predict the feared stimulus (the UCS) and, therefore, the fearful response is irrelevant. That new memory of the new contingencies, if unreinforced, is subject to a time-based decay. It is forgotten over time and the fear re-emerges (Bouton, 2004; Bouton & Moody, 2004; Dillon & Pizzagalli, 2007; Massad & Hulsey, 2006; Rescorla, 1988; Vervliet, 2008).

Contextual renewal refers to the re-emergence of the conditioned response in a new circumstance where the extinction memory was not created. If the patient is subjected to unreinforced (extinction) trials in one context, so that the CS fails to evoke the feared response in that context, a subsequent test of that same CS in another context may show little or no reduction in expression. Even though the original fear response may generalize to multiple contexts, extinction phenomena are much more context dependent. Contextual renewal is contextually bound; the response is only renewed in the contexts where the UCS has again appeared (Bouton, 2004; Bouton & Moody, 2004; Dillon & Pizzagalli, 2007; Massad & Hulsey, 2006; Rescorla, 1988; Vervliet, 2008).

Reinstatement occurs when the fearful stimulus, the UCS, is presented without the CS. In that context where the original UCS is presented, despite the fact that the fearful response had been fully extinguished, the CS will be restored. It will not, however reappear in other contexts where the UCS has not been presented (Bouton, 2004; Bouton & Moody, 2004; Dillon & Pizzagalli, 2007; Massad & Hulsey, 2006; Rescorla, 1988; Vervliet, 2008).

Rapid reacquisition, as the name suggests, describes the reacquisition of the fear memory after it has been successfully extinguished. In this case there is a net savings in the number of trials needed to re-acquire the memory. If, for example, the original fear association took ten trials to install, during post-extinction training, it may take only three (Bouton, 2004; Bouton & Moody, 2004; Dillon & Pizzagalli, 2007; Massad & Hulsey, 2006; Rescorla, 1988; Vervliet, 2008).

Extinction has traditionally been held to be the tool of choice for the treatment of PTSD. Foa and her colleagues have indicated that in its various forms, from desensitization through imaginal and in vivo exposure, inter alia, it is the most well-researched and most highly regarded of treatments and, in combination with cognitive behavioral interventions or supplements, represents the scientific treatment of choice (Foa, Keane, & Friedman, 2000; Foa & Meadows, 1997a, 1997b; Rothbaum, Davis, King, Ferris, & Lederhendler, 2003; Wessa & Flor, 2007).

Extinction-based exposure treatment is the most common form of intervention, and one of the only treatments supported and funded by the Federal Government. Nevertheless, one would expect that relapse data from extinction-based studies will provide the following predictable kinds of relapse behavior. Because PTSD and trauma-related memories are resistant to extinction, it is to be expected that extinction effects would be variable at best. Because the extinction memory is subject to decay over time, extinction-based treatments may be expected to be characterized by a certain level of temporal instability. In light of the crucial role played by the VmPFC in the inhibition of amygdalar function in extinction training, the decreased function of those circuits under conditions of extreme stress mitigates against the efficacy of exposure models. For these reasons, without further treatment, extinction measures alone may be only partially effective (Diamond et al., 2007; Gharakhani et al., 2006; Liberzon et al., 2007; Wessa & Flor, 2007).

An examination of the exhaustive work of Foa and her colleagues (Foa & Meadows, 1997; Foa et al., 2000) along with data from other researchers (Massad & Hulsey, 2006; McNally, 2007; Wessa & Flor, 2007) reflects that extinction-based results are inconsistent in their long-term effects.

A review by Shalev, Bonne and Eth (1996) finds mixed results with cognitive behavioral interventions based largely on extinction practices. Rothbaum, Meadows, Resick, & Foy (2000) reviewed 19 studies of extinction-based treatments for PTSD at various levels of sophistication. The studies included exposure treatments, systematic desensitization and combinations of treatments including exposure elements. The results were uneven and ranged from little or no effect on PTSD symptoms to 85% reductions in symptoms. Unfortunately they do not report follow-up studies in a consistent manner that would allow us to assess whether the treatments continued to produce positive effects, lost strength, or possibly increased in effect over time. Measurements were often reported in terms of percent of change in treatment score and when effect sizes were reported, they were most often in comparison to waiting list controls and relaxation conditions. This suggests only that the treatments were better than nothing. Practice

guidelines for the treatment of PTSD, provided by the American Psychiatric Association (Ursano et al., 2004) finds similar uneven results and reports that exposure-based treatments and EMDR have the best clinical and research track records. A meta analysis by Bisson, Ehlers, Mathews and colleagues (Bisson et al., 2007) evaluated comparative treatments but provided little useful information for our purposes.

Pitman et al. (1996) reviewed several cases of Vietnam Veterans who were subjected to multiple sessions of imaginal flooding. An average improvement between pre- and post-measures of intrusion and avoidance of 26% reduced to 14% in post treatment follow-up. These findings were consistent with other comparisons between combat and noncombat treated trauma victims including patients from the Israeli "Koach" program where similar problems were encountered. In their final analysis, the authors determined that the extent of emotional processing was unrelated to the efficacy of treatment measured in terms of the decrease of intrusive symptoms.

Reconsolidation

During the early 21st century, reconsolidation came to the forefront of memory research when it was illustrated that propranolol, a noradrenalin inhibitor, when injected directly into the amygdalar tissue of rats, was capable of modifying or erasing the trace of traumatic memories. Other studies found that the introduction of anisomysin into the basolateral amygdala had similar results. Further chemical studies revealed that the crucial element, further upstream, was the inhibition of glutamate production and release, preventing the reconsolidation of the activated memory (Cao et al., 2008; Jacek, Ecedil, & LeDoux, 2006; Riccio, Millin, & Bogart, 2006; Tronson & Taylor, 2007).

This research revived a long-standing debate into the nature of long term memory. Whereas classical extinction, as noted above, held that new memories blocked access to permanent associations between conditioned stimuli and responses in long-term memory stores, reconsolidation theory held that the process of long-term memory consolidation took place over time and that each access to the memory rendered the association labile, subject to change. The research has also differentiated reconsolidation from extinction; reconsolidation, it was emphasized, is not facilitated extinction (Akirav & Maroun, 2006; Alberini, 2005; Cao et al., 2008; Debiec, Doyre, Nader, & LeDoux, 2006; Duvarci & Nader, 2004; Forcato, 2009; Gharakhani et al., 2006; Lee, Milton, & Everitt, 2006; Milekic & Alberini, 2002; Nader, Schafe, & LeDoux, 2000; Riccio et al., 2006; Tronel, Milekic, & Alberini, 2005).

Insofar as the reactivated memory associations were always reinforced with the same or similar data, in the same or a similar context (including subjective contexts) over time, the memory grew less and less susceptible to change. If, however, when the memory was reactivated, the organism encountered new information, the actual content of the memory might be modified or even erased. Here is a clear mechanism for understanding the variability of long-term memory as examined by Elizabeth Loftus and others. Lee has characterized reconsolidation as a mechanism for maintaining the relevance of learned associations (Alberini, 2005; Hupbach, Hardt, Gomez, & Nadel, 2008; Labar, 2007; Lee, 2009; Loftus & Yuille, 1984; Tronel et al., 2005).

The root material regarding reconsolidation is a body of studies of the blockade or enhancement of cellular processes related to glutamate activity in various areas of the brain. In general, in the activation of a presumably permanent, non hippocampus-dependent memory (Eichenbaum, 2006), infusions of protein inhibitors administered before memory activation, tended to erase or decrease the intensity of the memory, while infusions of chemicals that support protein synthesis, enhanced or stabilized the memory. Crucially, the memory must be activated by a brief exposure to a first-order cue. The effects are time dependent and only occur within a specific window of opportunity after the memory is activated. These changes typically do not appear in short term memory but in measures of long term memory. The effects may thus be seen to increase over time (Akirav & Maroun, 2006; Alberini, 2005; Debiec, LeDoux, & Nader, 2002; Duvarci & Nader, 2004; Forcato et al., 2007; Kaang, Lee, & Kim, 2009; Kindt, Soeter, & Vervliet, 2009; Nader et al., 2000; Riccio et al., 2006).

Just as extinction procedures have distinctive characteristics, so reconsolidation mechanisms also have their signature aspects. These include: diminution or complete elimination of the target memory, resistance to spontaneous recovery, lack of net gain in reacquisition learning, and lack of contextual renewal.

Memories so treated tend to be permanently removed or permanently modified. If they are subject to spontaneous recovery, they reappear in the modified form (Loftus & Yuille, 1984). If they have been erased or replaced they cannot be revived without retraining de novo (Cao et al., 2008; Duvarci & Nader, 2004; Forcato, 2009; Forcato et al., 2007; Kindt et al., 2009; Lee et al., 2006). There is also some evidence to suggest that unlike extinction, reconsolidation may not apply to instrumental conditioning and pure place conditioning. The same evidence may be understood as pointing to differences in reconsolidation windows for different kinds of memories or sensitivity to different levels of pretraining for different kinds of memories (Lee, 2009).

Reacquisition in extinction typically shows enhanced efficiency of relearning. That is, it takes fewer trials to reestablish the subject behavior. In memories that have been modified using reconsolidation-based procedures, there is no net gain in relearning; when relearned, they are

learned as if they were totally new behaviors (Cao et al., 2008; Duvarci & Nader, 2004; Kindt et al., 2009).

Contextual renewal does not occur. This means that even when the unconditioned fear stimulus is presented in a novel context, the learned fear response does not reappear. This is not enhanced extinction (Duvarci & Nader, 2004).

Procedurally, reconsolidation and extinction are initiated using distinct protocols. The crucial difference appears to be that a single, short presentation of the conditioned stimulus reactivates and labilizes the memory, while longer presentations and multiple presentations activate the extinction memory. When protein synthesis inhibitors or other chemical agents are used, they must be administered before the reactivation trial to affect either process. Where experiential effects are used as amnestic agents — essentially modifying, overwriting or eliminating the target memory — their efficacy is in part determined by their temporal proximity to the eliciting stimulus. That is, the sooner, the better (Lee, 2009; Pedreira, Perez-Cuesta, & Maldonado, 2004).

In human studies of the phenomenon, it has been found that reconsolidation can be used to eliminate or modify the emotional component of a traumatic memory while leaving the declarative elements intact. It has also been shown that reconsolidation processes have a direct effect on the first order memory but not on associative chains based on that original experience (Kindt et al., 2009).

Shiller et al. (2010) have illustrated the efficacy of reconsolidation in the modification of conditioned fear memories in humans. In this study, massed extinction trials during a reconsolidation window created reductions in autonomic responses that were not subject to spontaneous recovery or reinstatement. Those effects persisted after one full year.

Reconsolidation and the Visual Kinesthetic Dissociation Protocol

Reconsolidation effects have been suggested as a powerful method for the treatment of PTSD. While historically, the mechanism has been associated with the non-pharmacological creation

of retroactive amnesia, there has been little work that explicitly links this material to treatments for PTSD. Despite other explanations, the authors believe that a little known intervention originally described by Richard Bandler (1985) makes specific use of reconsolidation mechanisms and deserves serious reconsideration (Forcato et al., 2007; Labar, 2007; Riccio, Millin, & Bogart, 2006).

The technique, Visual Kinesthetic Dissociation (V/KD), is supported by anecdotal reports by practitioners that cover nearly a quarter century. Consistent with the pattern of memory reconsolidation, the intervention includes minimal evocation of the problem response along with multiple overlays of new associations that, in essence, rewrite the memory. As in previously reported reconsolidation studies, the traumatic event either becomes inaccessible, significantly modified, or subject to non-traumatic declarative access (Alberini, 2005; Hupbach, Hardt, Gomez, & Nadel, 2008; Labar, 2007; Tronel, Milekic, & Alberini, 2005).

Among the anecdotal reports are those provided by Richard Bandler, Steve and Connierae Andreas, Robert Dilts, and William McDowell who severally relate that each of them has treated thousands of persons suffering from PTSD and phobic conditions with immediate, lasting results from this short term intervention. In many cases they report complete symptom alleviation after long term follow-up (Andreas & Andreas, 1989; Bandler, 1985; Dilts & Delozier, 2000; McDowell & McDowell, nd).

The procedure was originated by Richard Bandler and first appeared in his *Using Your Brain for a Change* (1985). An expanded version of the procedure appeared in the Andreas' description in the *Heart of the Mind* (Andreas & Andreas, 1989). Dilts and Delozier (2000) provide a slightly different version of the protocol in their *Encyclopedia of Systematic NLP*. The technique has since been popularized in the UK as the rewind technique (Guy & Guy, 2003; Muss, 1991, 2002).

In early studies, the roots of the technique were traced to the work of Erica Fromm and her use of hypnosis for the control of pain and anxiety. In light, however, of Bandler and Grinder's extensive studies of the work of Milton Erickson and their participation in his classes and with his students, it would seem more likely that the technique has its roots in Erickson's well documented use of dissociative techniques (Bandler, 1985; Bandler & Grinder, 1975; Bandler & Grinder, 1979; Dietrich, 2000; Dietrich et al., 2000; Erickson, 1964/1980, 1980; Fromm, 1965; Koziey & McLeod, 1987).

Psychological investigations of the technique are limited to three scientific studies, two reviews and several mentions in the literature. Figley reviews the technique and provides a case study. Each of the referenced studies recommends the technique as a valuable tool for treating PTSD and makes suggestions for further research (Carbonell & Figley, 1999; Dietrich et al., 2000; Figley, 2002; Hossack & Bentall, 1996; Koziey & McLeod, 1987; Muss, 1991, 2002). There is also one non-peer reviewed study that is reported on-line (Guy & Guy, 2003).

Koziey and McCleod, writing in 1987, reported their experiences in treating two rape victims with a mixed technique employing Bandler's three-place dissociation in combination with hypnotic trance. An initial pretreatment session was used to review the technique and to complete an assessment package. In a second session, the authors used hypnotic trance to provide a resource state to ensure that the traumatic memories would not become overwhelming. One week later, in a second treatment session, the patients completed another set of evaluations, were hypnotized and then the hypnotized subjects were led through the three part dissociation. Here, they were led through an imagined, dissociated review of the trauma in which they watched themselves watching themselves, viewing a movie of the trauma. The movie began with a still image of the client in a safe time before the traumatic event, projected on an imaginary screen. The experience ended with a safe place after the trauma with each of the clients merging their own dissociated identities with their imagined selves on the screen and sharing the learnings from their experience. Unspecified measures of 28 dependent variables showed significant changes in pre-post comparisons with near total abatement of symptoms in one of the subjects.

Muss (Muss, 1991, 2002) reports having used the technique first with 19 police officers who met DSM III diagnostic criteria for PTSD, and later with all manner of traumatized persons (Muss, 2002). In nearly all of the 19 police cases, he reports remission of symptoms. He provides no control conditions and few details of the study, however, in long term follow ups (3 month to three years), for 15 of the 19 cases, he reports a complete absence of intrusive imagery. Crucially, as noted by Andreas and Andreas, he indicates that the technique is appropriate to clients whose primary symptoms are experienced as intense, suddenly arising experiences of the trauma symptoms usually experienced as flashbacks or panic reactions (Andreas, 2008, Personal communication).

A third study, by Hossack and Bental (1996), included five subjects who were treated with a combination of guided visualizations, Jacobsen's deep muscle relaxation and two sessions of the VK/D protocol. Although one of the five subjects was unable to complete the visualizations associated with the VK/D procedure, the four completers all reported significant reduction of intrusive images and were able to return to normal life activities.

Guy and Guy report that the technique (renamed by Muss (1991, 2002) as the Rewind Technique) was applied to thirty people between 2000 and 2002.

All were diagnosed with PTSD or partial PTSD. Participants were interviewed ten days post treatment. Forty percent adjudged their improvement as extremely successful, fifty-three percent as successful and seven percent as acceptable. None rated the treatment either as poor or as a failure (Guy & Guy, 2003).

The following description of the basic protocol depends upon extensive personal communications with Andeas, Dilts and Hallbom and continued reference to their descriptions of the protocol in several written sources (Andreas & Andreas, 1989; Bandler, 1985; Dilts & Delozier, 2000). A detailed protocol is available from the author.

The Visual Kinesthetic Dissociation

It is important to understand that Andreas emphasizes that this technique is only effective when the client's difficulties are essentially a phobic, instantaneous conditioned response to stimuli related to a traumatic event. In general, the symptoms will focus on flashbacks and other immediate panic responses to stimuli associated with the traumatic event. In terms of classical PTSD symptomatology, this is associated with simple PTSD. While it may be effective in complex cases, its efficacy will be limited to the intrusive and avoidant symptoms.

The technique begins by establishing rapport and framing the intervention as a short visualization process. The process is ordinarily comfortable, but sometimes has a very short period of moderate discomfort. This stage may include practice behaviors, including the running of an innocuous, unrelated experience backwards in subjective experience.

Insofar as the technique may be pursued content free, the clinician should decide whether to proceed with or without content. After framing, the problem state is *briefly* accessed by questioning and probing until the client responds physiologically. Access to the problem state is typically marked by physiological and paralinguistic elements that reflect heightened arousal

and fast onset of the physiological and paralinguistic symptoms of fear or trauma. These may include changes in breathing, heart rate, skin tone and color, vocal pitch and speech rate.

As soon as the state is identified, the client's attention should be refocused from the problem state and reoriented to the present context.

The actual three part dissociation begins with asking the client to imagine that he or she is seated in a movie theatre. On the screen is a still image of the client performing some neutral activity in a safe context, at a time before the trauma occurred. The picture on the screen represents one level of dissociation, while watching the picture constitutes a second level of dissociation. Having established these initial dissociative experiences, the client is asked to dissociate from the image of themselves sitting in the theatre by imagining themselves floating away from their body in the theatre to a projection booth behind a plexiglas barrier. From this vantage point, they are instructed to watch themselves in the theatre, watching themselves on the screen.

At this point, an anchor or conditioned association is made between a specific touch stimulus and the current dissociated experience. This is intended to reinforce the experience of dissociation from the image on the screen. This may also be used to evoke or enhance dissociation during later parts of the protocol.

After taking care to create a dissociated context and a conditioned stimulus that can evoke and reinforce that context, the client is instructed to observe a black and white picture of herself on the screen of the movie theatre, at a time before anything ever happened. As the client focuses on the imagined picture, she is directed to watch herself in the theatre as she watches a black and white movie of the triggering event or the root trauma. She is to continue to watch the observer in the theater, seeing herself going through it, all the way to a point past the end of it, where she can see that she survived and is safe once again. She is further instructed that upon reaching the end of the movie, she should stop the movie as a still, black and white image of a safe time after the trauma has passed. After signaling to the clinician that all is well, the protocol either proceeds to the next step or is repeated until the black and white, dissociated movie can be completed comfortably.

Beginning with the safe, black and white representation of herself that is on the screen at the end of the dissociated rehearsal, the client is now instructed to imagine stepping into the movie and experiencing the entire sequence, fully associated, in color, in reverse, at very high speed (in two seconds or less). This step may take several repetitions unless it has been practiced during the framing. If the client reports that the reversed rehearsal has gone well, she may be debriefed and a determination made as to whether further iterations are necessary.

In order to determine whether the procedure has had the desired effect, every effort is now made to evoke the problem state using the same questions and probes that evoked the problem state at the beginning of the procedure. Special attention should be given to those questions that were associated with a clear physiological reaction.

Consistent with practices suggested by Foa and Kozak (1986), each sensory system should be probed for possible triggers for the problem behaviors. If there is no reaction, the intervention is presumed to have worked. When the practitioner is satisfied that she cannot evoke the PTSD response, the basic intervention is complete.

Some practitioners suggest the addition of a third level of novel experience that includes a reliving of the traumatic event in which they are either protected from harm, only acting as if they were a stunt-double in a movie of the event, or they missed the crucial trauma altogether. This level is especially recommended in the case where physical injury was part of the traumatic event.

Mechanism

Each of the authors noted above attempts to explain the mechanism of the intervention by various means including, change in the perceptual structure of the memory (McDowell & McDowell, nd), changes in the integration of the memory mediated by the modulation of arousal (Dietrich, 2000), and dissociation from the traumatic sequellae (Dietrich et al., 2000).

Having reviewed the literature on PTSD, extinction and reconsolidation, the authors believe that the mechanism of the VK/D intervention is most parsimoniously explained in terms of memory restructuring through the mechanism of reconsolidation.

In accordance with previously outlined studies of the phenomenon, the technique begins with a short term activation of the traumatic experience. Insofar as the experience is marked by significant changes in physiology, it meets Foa and Kozak's suggestion that an appropriate intervention be rooted in a full activation of the event. Here, full activation does not imply flooding, only sufficient activation of the core memory to render it labile. Nevertheless, in order to prevent the shutdown of the cognitive capacities detailed by Diamond, et al., the client is distracted from the trauma and his consciousness returned to the present context, as quickly as possible. This not only prevents re-traumatization but it limits the activation of the memory to a temporal window that is appropriate to reconsolidation, but too brief to support extinction (Akirav & Maroun, 2006; Alberini, 2005; Foa & Kozak, 1986; Hupbach et al., 2008; LeBar & Phelps, 1998; Riccio et al., 2006).

This foreshortening of exposure may be crucial to the initiation of memory labilization that allows for reconsolidative memory modification. According to Pedreira, Perez-Cuesta, & Maldonado (2004), neither reconsolidation nor extinction is possible without the termination of the conditioned stimulus—the fear-evoking stimulus context. According to their research, a stimulus offset is required to effect significant change. Both processes require the termination of the fear context before new learning can occur.

Having awakened the traumatic memory, it now becomes subject to reinforcement or modification depending upon the immediate stimulus context. Remembering that the memory is still active in the background, through its intentional revivification and its presence in the semantic context, the VK/D model now provides several experiences of dissociation: the dissociative anchor, the dissociated safe representation on the imagined movie screen and the client's floating out of the body to view the viewer who is sitting in the theater watching the screen. Insofar as the memory is dissociated, and is not actively reinforcing the fear response, its novel stimulus properties may preferentially support memory updating through reconsolidation rather than extinction (Pedreira, Perez-Cuesta, & Maldonado, 2004).

At this point, the first of several layers of active intervention in the structure of the traumatic memory begins. The dissociated black and white movie provides a multi-leveled opportunity for reshaping the memory context. First, it is triply dissociated (Dietrich, 2000; Hossack & Bentall, 1996; Koziey & McLeod, 1987; Muss, 2002). Second, insofar as it is a voluntary re-experience of the trauma, the context is restructured as voluntary rather than involuntary. This is what various authors have described as prescribing the symptom. It is also a direct remedy for the loss of control described by Foa as a significant contributor to PTSD symptomatology (Bandler & Grinder, 1979; Erickson & Rossi, 1980; Foa & Meadows, 1997; Haley, 1973). In this context, the

novelty of symptom prescription may enhance the reconsolidation response as noted by Pedreira et al. and also by Lee. These authors indicate that unexpected stimulus properties support reconsolidative updating of memory content (Lee, 2009; Pedreira et al., 2004). Third, because the movie is viewed in black and white, its emotional impact is further vitiated (Bandler, 1985; Bandler & MacDonald, 1987; Kringelbach, 2005). In theory, all of these elements are incorporated into the structure of the original memory.

Once the client has successfully completed the dissociated review, another layer of new meanings is added to the memory through the reverse, associated rewind of the memory. At this point, attempts have been made to recall the affect associated with the traumatic event and by now, it should already be difficult. This rewind phase constitutes a significant restructuring of the memory. The associated reversal of the experience leaves the client with a subjective memory of the problem, 'undoing itself.' This is performed quickly. The speed takes advantage of the narrow window of memory lability, and the heightened salience accorded to fast moving, multisensory stimuli. Bandler suggested that the reversal of memory sequence was a valuable tool for undoing decisions, preconceived notions and other artifacts of temporal experience (Bandler, 1985; Simons, Detenber, Reiss, & Shults, 2000).

As with many of the elements of this intervention, novelty may be a significant element in its efficacy. It has already been noted how the unexpected features of the intervention may support the reconsolidation mechanism. The complexity of this part of the intervention may also support further modification of the experience through simple cognitive overload. Given the limited capacity of short term memory, the simple fact of learning and executing the reverse rehearsal may not leave sufficient capacity in short-term memory to access the fear response (Miller, 1956; Pedreira et al., 2004).

After completing the multisensory, high speed, reversed, imaginal exposure, the client is again debriefed and every effort is made to reaccess the trauma response. If the intervention has been successful, the client may retain declarative access to the event, but without the strong negative affect that characterizes the symptoms of PTSD (Andreas & Andreas, 1989; Bandler, 1993; Dilts & Delozier, 2000; Kindt, Soeter, & Vervliet, 2009; Muss, 1991).

In those cases where an imagined restructuring of the original event is performed, the mechanism may be as simple as layering-in another set of experiences that are incompatible with the trauma. Moreover, the addition of modified memories reflects one of the standard reappraisal methods of cognitive regulation strategies. Such interventions are known to

increase activity in the ventro-medial prefrontal cortex which exercises a modulatory influence on the amygdala. Insofar as the emotional impact of the traumatizing memory has already been significantly modified, the new version of the traumatic event may serve to provide a coherent narrative for the now, non-traumatizing memory (Diamond, Campbell, Park, Halonen, & Zoladz, 2007; Hartley & Phelps, 2009; Williams et al., 2006).

Case Study

A recent case study provided by William A. McDowell, Ph. D., Professor Emeritus and Chair of Counseling, Marshall University, Huntington, WV (William McDowall, personal communication, July 9, 2010) involved a 30 year-old veteran of the Iraq war. The Veterans Administration had diagnosed him with PTSD and he reported to the NLP therapist after 1.5 years of standard treatment from the VA including individual and group psychotherapy with no abatement of symptoms. At the time that he came to the NLP-trained therapist, he was reporting the following symptoms: flashbacks, nightmares, high anxiety while driving (unable to let others drive him, panicked by large trucks near him often forcing him to pull off the road). He also reported a fear of crowds and enclosed spaces (restaurants, classrooms) where he had to be near an exit with a clear view of the entire space. He also reported some anger problems at home and an inability to have the doors unlocked. The patient minimized family problems but his wife reported that the family was experiencing severe difficulty.

The client was seen for three, one hour, videotaped sessions with a three-day break between the sessions. Pre- and post-tests using the PTSD Checklist (PCL) (Weathers, Litz, Herman, Huska, & Keane, 1993, 1994) were completed before treatment, at the beginning of the second treatment session, after the third session and thirty days post-treatment. Pretreatment scores on the PCL were in the 90% range. After the first treatment session, they were reduced to 30%. All symptoms disappeared after the third treatment. Thirty days post-treatment the symptom scores were still zero and no symptoms were reported by the client.

After the second session, the client reported no more nightmares and abatement of all symptoms by the end of treatment. In order to behaviorally evaluate the client's post-treatment improvement, after the last session *he was driven* to a crowded, noisy McDonalds restaurant and ate with his back to the door. He reported having no anxiety with someone else driving him and was able to eat without being concerned for the exits. A video record of the excursion validates his visible physiological responses and demeanor.

He and his wife were interviewed together at the last session and in a one-month follow-up. At the one-month follow-up, he reported being able to allow his wife to drive and experienced no anxiety while driving near large trucks. He also reported being anxiety-free while in crowded places.

For independent verification, the client's family was asked to substantiate his recovery. His wife reported in a post three-month clinical interview that her husband no longer checked the locks in the house more than once, was able to let her drive, and no longer demonstrated the high anxiety and anger at home that he had before treatment. In her words, "I feel like I have my husband like he was before he left for Iraq."

Directions for Future Research

The preceding article has set forth a theoretical mechanism for the V/K D protocol for the treatment of PTSD and has made specific predictions with regard to its efficacy and how the proposed mechanism, reconsolidation, may be differentiated from protocols that invoke extinction. It was predicted that subjects treated with the V/K D protocol would not be subject to spontaneous recovery, contextual renewal, reinstatement and rapid reacquisition; the hallmarks of extinction (Bouton, 2004; Bouton & Moody, 2004; Dillon & Pizzagalli, 2007; Massad & Hulsey, 2006; Rescorla, 1988; Vervliet, 2008). To the contrary, in cases treated with the V/K D protocol the previously traumatizing and intrusive memories will be transformed, often inaccessible and where accessible to declarative and episodic recall, they will have been rendered non-traumatizing. They will not be subject to spontaneous recovery, contextual renewal, reinstatement and rapid reacquisition (Cao et al., 2008; Duvarci & Nader, 2004; Forcato, 2009; Forcato et al., 2007; Kindt et al., 2009; Lee et al., 2006).

These results should be measurable using repeated applications of a standard PTSD symptom inventory such as the PCL-M. The authors predict consistently reduced or continuously reducing scores for the intrusion and avoidance measures of the scales for patients treated with the V/K D protocol and variable or increasing scores for the extinction-based treatments (Weathers, Litz, Herman, Huska, & Keane, 1993, 1994). Increasing scores would indicate the presence of the various kinds of mechanisms that are associated with extinction; spontaneous recovery, contextual renewal, reinstatement and rapid reacquisition. Continued reduction of symptoms and/or symptom intensity would indicate the action of reconsolidation.

In order to test this prediction a pilot study should be conducted consisting of a large sample of recent veterans of the Iraqi or Afghanistani wars who have been diagnosed with PTSD that is primarily characterized by intrusive and avoidant symptoms. A standard diagnostic instrument with well established validity would be used on all patients to exclude patients with significant co-morbidities and for whom the intrusive and avoidant symptoms are not primary. Exclusion criteria would eliminate veterans who suffer from severe cases of co-morbidity (severe long standing alcohol and drug addictions, pre-existing personality disorders, psychoses, or any of these secondary to PTSD symptoms). It should be remembered that the V/K D protocol is specifically targeted at the reliving and phobic elements of PTSD and is not indicated for what Brewin, Dalgleish and Joseph (1996) have characterized as secondary emotions and comorbidities such as alcoholism, drug addictions and family dysfunctions.

After prescreening, patients would be randomly assigned to treatment groups using either the VK/D or an extinction based protocol. In general, the evaluation should follow the Gold Standards for evaluation research as reported by Foa and Meadows (1997).

Discussion

PTSD is a problem that is currently affecting as many as 13 to 17 percent of returning Iraqi veterans and 10 to 11 percent of those returning from Afghanistan. Reports indicate that up to 40% of sufferers remain untreated. Aside from cultural issues that discourage mental health treatment, the long term commitments required by standard treatment modalities and inconsistent results do little to change motivation to obtain treatment (Gradus, 2010; Hoge et al., 2004).

The VK/D model is supported by 25 years or more of anecdotal reports covering thousands of patients. The intervention does not re-traumatize the patient and can be completed in as little as 45 minutes. Although only subject to three peer reviewed evaluations in the last twenty-five years, each of the evaluations deemed it worthy of further investigation. One author, Muss, has continued using the technique and through his efforts it is now a recognized treatment for PTSD in the UK (Carbonell & Figley, 1999; Dietrich, 2000; Koziey & McLeod, 1987; McDowell & McDowell, nd; Muss, 1991, 2002).

Until recently, the possible mechanism of action for this highly innovative treatment was difficult to specify and was often described in terms of brain lateralization, changes in perspective and other non-operationalizable constructs. Here, an argument for a mechanism based upon the emerging evidence for memory reconsolidation has been presented that is congruent with the structural elements of the intervention and predicts similar results (Dietrich, 2000; Hossack & Bentall, 1996; Koziey & McLeod, 1987; McDowell & McDowell, nd).

As noted, reconsolidation protocols depend upon a brief reactivation of the traumatic memory followed, after stimulus cessation, by an intervening amnestic or confounding event. In the VK/D protocol, the memory is briefly activated and several layers of dissociative experience and confounding imaginal memories are introduced during the (presumed) labile period. For experiences subjected to amnestic reconsolidation, insofar as a sufficiently intense memory is introduced during the labile phase of reconsolidation, the memory may be disrupted, erased or modified. After the VK/D process, the original memory becomes either inaccessible, innocuous or is transformed into a similar but nonthreatening memory. Human studies of the reconsolidation phenomenon have found that although the affective dimensions of previously negative memories are gone, the events remain accessible on a declarative level. Similarly, clients who have undergone treatment with the VK/D protocol retain declarative and episodic access to the stimulus event but without the traumatic affect (Andreas & Andreas, 1989; Bandler, 1985; Dilts & Delozier, 2000; Kindt et al., 2009; Lee, 2009; Lee, Milton, & Everitt, 2006; Riccio et al., 2006).

This analysis leads to several falsifiable predictions and diagnostic indicators of the underlying mechanism in PTSD treatments. Because the mechanism outlined here depends upon the known process of reconsolidation, interventions for PTSD may be behaviorally evaluated in terms of their results to determine whether extinction or reconsolidation is operative. Where extinction mechanisms have been invoked, spontaneous recovery, contextual renewal, reinstatement and rapid reacquisition will characterize the post treatment period and further treatment will be necessary in order to deal with the intrusive elements of the disorder (Bouton, 2004; Bouton & Moody, 2004; Dillon & Pizzagalli, 2007; Massad & Hulsey, 2006; Rescorla, 1988; Vervliet, 2008). Where reconsolidative mechanisms have been appropriately marshaled, the memories will be transformed, inaccessible and even if accessible to declarative and episodic recall, they will have been rendered non-traumatizing. They will not be subject to spontaneous recovery, contextual renewal, reinstatement and rapid reacquisition (Cao et al., 2008; Duvarci & Nader, 2004; Forcato, 2009; Forcato et al., 2007; Kindt et al., 2009; Lee et al., 2006). These results may also be used as diagnostics for the evaluation of mechanism where technique and results are variable and may further lead to the refinement of results based upon the length and intensity of the CS presentation and the timing of the introduction of the confounding or amnestic stimulus (Lee, 2009; Pedreira, Perez-Cuesta, & Maldonado, 2004).

Finally, these observations may lead to new interventions based on reconsolidative mechanisms.

Future research into this technique should look towards large scale trials of the protocol in the treatment of PTSD. The international troops returning from service in various theaters of war could provide a significant test population for this already established and relatively unknown treatment. There remain hundreds of thousands of war victims, refugees from earthquakes and tsunamis who would provide a grateful pool of subjects.

Follow up studies and surveys to take advantage of the now anecdotal evidence compiled by NLP practitioners would also be instructive. Such follow-up studies could provide crucial long-term reports of the incidence of post treatment relapse that would be capable of falsifying the proposal that the technique is rooted in reconsolidation rather than extinction.

Further research might also investigate the construction of even shorter interventions that depend upon reconsolidation. For instance, just as the current technique relies in part on a conditioned resource to amplify dissociation, it may be possible to create an even more efficient restructuring using a powerful, positive resource state as a conditioned response whose introduction during the labile period would create a memory transformation or erasure. The current explanation invites further exploration of the mechanism of reconsolidation to other interventions that have been to now, poorly understood.

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