2013

Optimal treatment for post traumatic stress disorder

BOSTON UNIVERSITY

SCHOOL OF MEDICINE

Thesis

OPTIMAL TREATMENT FOR POST TRAUMATIC STRESS DISORDER

by

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Submitted in partial fulfillment of the

requirements for the degree of

Master of Arts

2013

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ABSTRACT

Though recognized previously as “shell shock” or “combat neurosis” Post-traumatic Stress Disorder (PTSD) is an anxiety disorder that was first introduced in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-III)* in 1980. Diagnosis of PTSD requires the experience of a traumatic event followed by symptoms including avoidance, hyperarousal, re-experiencing, numbing and intense fear. The current treatment options include psychotherapy and pharmacotherapy. Brain stimulation is also emerging as an effective treatment option. The most widely studied and successful treatment is termed Prolonged Exposure therapy (PE). This involves the therapeutic repetition of the traumatic experience in order for the patient to understand that they are no longer in danger. Despite the effectiveness of PE, many individuals continue to suffer from PTSD. There are several obstacles between research and practice, as well as barriers to care for those suffering from PTSD. Even when evidence based practice is applied to those in need, there is still a high rate of treatment failures. Further research must be done to determine the best course of treatment for the increasing number of individuals suffering from PTSD.

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ABBREVIATIONS

AACAP	American Academy of Child and Adolescent Psychiatry
ACT	Acceptance and Commitment Therapy
APA	American Psychiatric Association
ASD	Acute Stress Disorder
BA	Behavioral Activation
BAI	Beck Anxiety Inventory
BDI	Beck Depression Inventory
CAPS	Clinician-Administered PTSD Scale
CBT	Cognitive Behavioral Therapy
CBCT	Cognitive-Behavioral Conjoint Therapy
CES	Cranial Electrotherapy Stimulation
CGI-I	Clinical Global Impression—Improvement Scale
CIDI	Composite International Diagnostic Interview
COPE	Concurrent treatment of PTSD and substance use disorders using Prolonged Exposure
CPG	Clinical Practice Guidelines
CPT	Cognitive Processing Therapy
DBS	Deep Brain Stimulation
DBT	Dialectical Behavior Therapy
DoD	Department of Defense
DSM	Diagnostic and Statistical Manual of Mental Disorders

ECT	Electroconvulsive Therapy
EMDR	Eye movement desensitization and reprocessing
HDE	Humanitarian Device Exemption
IE	Imaginal Exposure
IOM	Institute of Medicine
IR	Imagery Rescripting
IRT	Imagery Rehearsal Therapy
ISTSS	The International Society for Traumatic Stress Studies
MAOI	Monoamine Oxidase Inhibitor
MDD	Major Depressive Disorder
MST	Magnetic Seizure Therapy
NHMRC	Australian National Health and Medical Research Council
NICE	UK National Institute for Health and Clinical Excellence
NMDA	N-methyl-D-aspartate
OCD	Obsessive Compulsive Disorder
OEF	Operation Enduring Freedom
OIF	Operation Iraqi Freedom
OND	Operation New Dawn
PCL	PTSD Checklist
PCL-M	PTSD Checklist Military Version
PE	Prolonged Exposure therapy
PMA	Premarket Approval

PSS-I	Posttraumatic Stress Disorder Symptoms Scale-Interview Version
PTSD	Post Traumatic Stress Disorder
RCT	Randomized Controlled Trial
SER	Social and Emotional Rehabilitation
SIT	Stress Inoculation Training
SNRI	Selective Norepinephrine Reuptake Inhibitor
SSRI	Selective Serotonin Reuptake Inhibitor
TCA	Tricyclic Antidepressants
tDCS	transcranial Direct Current Stimulation
TFT	Thought Field Therapy
TIR	Trauma Incident Reduction
TMS	Transcranial Magnetic Stimulation
TMT	Trauma Management Therapy
TRD	Treatment Resistant Depression
TX	Treatment
VA	Veterans Affairs
VHA	Veterans Health Administration
VK/DD	Visio Kinesthetic Disassociation
VNS	Vagus Nerve Stimulation
VR	Virtual Reality
VRE(T)	Virtual Reality Exposure Therapy

INTRODUCTION

The term Post-traumatic Stress Disorder (PTSD) was only first introduced in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-III)* in 1980. Despite its relatively recent introduction, the symptoms have long been recognized by other names such as 'shell shock' or 'combat neurosis.' Since the inception of the term PTSD, research has increased rapidly to attempt to treat this complex disorder.

PTSD is an anxiety disorder that affects individuals who have directly experienced a traumatic event. The traumatic event may involve "actual or threatened death or serious injury, or other threat to one's physical integrity; or witnessing an event that involves death, injury, or a threat to the physical integrity of another person; or learning about unexpected or violent death, serious harm, or threat of death or injury experienced by a family member or other close associate" (DSM-IV-TR, 2000). Traumatic events can include violent physical assault, natural disasters, rape, and automobile accidents. One of the most common forms of trauma experienced by individuals with PTSD is combat. This is becoming an increasingly common occurrence, as the United States has been at war in Iraq and Afghanistan for over ten years. These wars have included Operation Enduring Freedom (OEF), Operation Iraqi Freedom (OIF) and Operation New Dawn (OND). During these operations over two million American service members have been deployed, and nearly half of them more than once (Wall, 2012). Due to advancements in protective equipment and battlefield

medicine, the survival rate of injury is currently nine in ten for OEF and OIF (Wall, 2012).

In addition to the experience of a traumatic event, a PTSD diagnosis requires the individual to respond with an intense fear and/or helplessness, with characteristic symptoms including persistent reexperiencing of the traumatic events, avoidance of stimuli associated with the trauma, numbing of general responsiveness, and symptoms of increased arousal. When all of these symptoms are present for at least one month, causing significant distress and functional impairment, the disorder can be diagnosed as PTSD (DSM-IV-TR, 2000).

The three main symptoms of PTSD are broadly classified as re-experiencing, avoidance, and increased arousal. Each of these classes of symptoms may present in varying ways. Reexperiencing of the traumatic event may occur by intrusive thoughts and images, recurrent dreams of the event, acting as if the event were recurring, psychological distress when exposed to external or internal cues that remind the individual of the traumatic event or physiological reactivity to these external or internal cues. Avoidance may present as an effort to avoid thoughts, feelings, or conversations associated with the trauma; efforts to avoid activities, places or people that stimulate recollections of the trauma; inability to recall important aspects of the trauma, diminished participation or interest in activities; feelings of estrangement or detachment from others; restricted range of affect; or sense of a foreshortened future. Increased

arousal may be indicated by difficulty sleeping, irritability, outbursts of anger, difficulty with concentration, hypervigilance, and exaggerated startle response (DSM-IV-TR, 2000).

PTSD is a complex and heterogeneous disorder, affecting many different types of people in many different ways. It also has acute, chronic and delayed onset forms. If symptoms persist for longer than three months, the individual is considered to have chronic PTSD. If symptoms last for less than three months, but at least one month it is considered acute PTSD. Prior to one month, PTSD cannot be diagnosed, but the symptoms are instead considered an acute stress reaction or acute stress disorder. When the onset of symptoms is at least six months following the traumatic event, the individual is considered to have delayed onset PTSD (DSM-IV-TR, 2000).

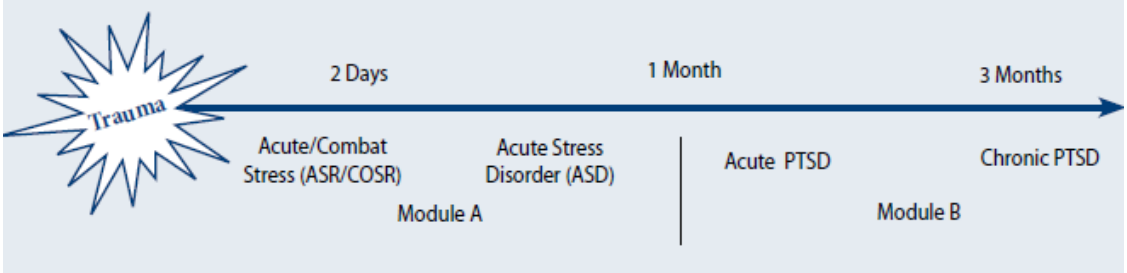

Figure 1. *Stress Reaction Timeline ("VA/DoD Clinical Practice Guidelines," 2009)*

The current treatment options for PTSD have a high rate of failure (Cukor et al., 2009). As many as 41% of patients with PTSD fail to respond to pharmacotherapy (Novakovic et al., 2011) so it is imperative that alternative treatment options be identified.

This review will focus on, but not be completely limited to, PTSD in military populations, including both veterans as well as active duty service members. It has been shown that veterans with PTSD have more severe symptoms than civilians with PTSD. Combat exposure specifically results in worse symptomology than other types of trauma. (Carter, Capone, & Short, 2011). Combat exposures, specifically those involving witness of death, threat of death and witnessing or experiencing serious injury, have been shown to be a significant risk factor for PTSD among male Marines (Phillips et al., 2010). Individuals who experience physical injury as a part of their traumatic experience represent a complication to combat exposure as a risk factor. The patient's physical injury, especially if permanent, serves as a powerful and persistent reminder of the trauma (Jeffreys, Capehart, & Friedman, 2012). Ninety-four percent of veterans returning from Afghanistan and Iraq have reported experiencing such life-threatening, combat-related events (Carter et al., 2011; Hoge et al., 2004) putting them directly at risk for developing PTSD.

While combat exposure is the most stand-out risk factor for development of PTSD in military populations, there are other risk factors as well. In a study of deployed male US Marines, past exposures to violence prior to entering the Marine Corps were also shown to put individuals at greater risk of developing PTSD (Phillips et al., 2010).

Not all individuals exposed to a traumatic event are bound to develop PTSD. A number of genetic and environmental factors prior to the event may

play a role in the development of the disorder. Some individuals may be pre-disposed to developing PTSD. It is hypothesized that vulnerability stems from biological diathesis, early childhood experiences & trauma severity (Novakovic et al., 2011). Additional identified factors include a genetic predisposition, sex, social support structures, peritraumatic dissociation and trauma frequency (Brewin & Andrews, 2000; Brewin & Holmes, 2003; Jeffreys et al., 2012; Keane, Marshall, & Taft, 2006).

In 1995 it was estimated that the lifetime prevalence of PTSD in the general population was 7.8 percent (Kessler, 1995). It is difficult to estimate the actual prevalence of PTSD, especially in military populations. In Vietnam veterans, the lifetime prevalence of PTSD has been estimated to be 18.7 percent. The prevalence of PTSD in Veterans of the Iraq and Afghanistan wars (OEF/OIF/OND) is estimated to be between 15 and 17 percent. Many experts believe these rates underestimate the actual prevalence of PTSD due to a variety of factors (Jeffreys et al., 2012). One major factor is underreporting, which in itself has a variety of causes. Among active duty personnel there is a stigma associated with mental health disorders and fear of negative effect on one's career (Hoge et al., 2004; Iversen et al., 2011; Wall, 2012).

Not only is PTSD difficult to treat in the short term, but many patients with PTSD suffer for a long time. One case study described a Vietnam veteran who suffered from re-experiencing, avoidance, and hyperarousal symptoms for more

than 40 years following several traumatic events during his deployment in Vietnam. (Carter et al., 2011)

Treatment options include preventative measures, immediately following a traumatic event (Roberts et al., 2010). as well as treatments that begin following a diagnosis of PTSD. The two major types of treatment include psychological interventions and pharmaceutical interventions (Hetrick et al., 2010). Brain stimulation modalities are also emerging as effective treatments for PTSD.

Psychological interventions include Cognitive Behavioral Therapy (CBT), eye movement desensitization and reprocessing (EMDR) supportive counseling, interpersonal therapy and psychodynamic treatments (Hetrick et al., 2010).

Pharmaceutical interventions commonly include Selective Serotonin Reuptake Inhibitors (SSRIs) as well as Selective Norepinephrine Reuptake Inhibitors (SNRIs). There are also a number of emerging pharmaceutical agents that hold potential in the treatment of PTSD.

No brain stimulation modalities are currently FDA approved for the treatment of PTSD, but Electroconvulsive Therapy (ECT) and Transcranial Magnetic Stimulation (TMS) are commonly used to treat severe depression and may also be effective for PTSD. Other modalities are still under investigation.

A complicating factor in the treatment of PTSD is that a majority of patients have comorbidities such as Traumatic Brain Injury (TBI) or substance abuse/dependence. These comorbidities are limiting for research and in clinical practice.

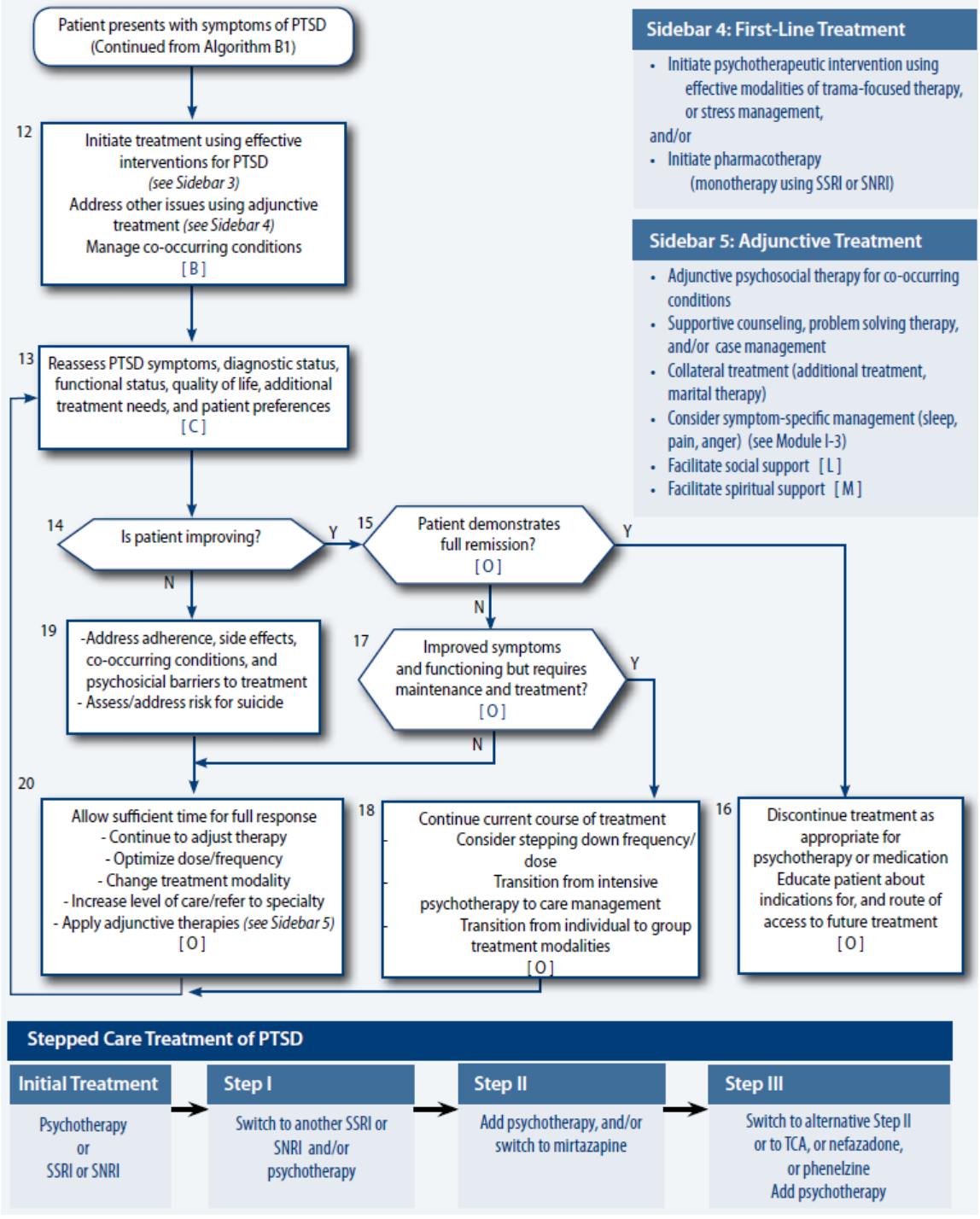

Figure 2.

Overview of Treatments for PTSD ("VA/DoD Clinical Practice Guidelines,"2009)

PRESENTATION OF PUBLISHED DATA

Psychological Interventions

Cognitive Behavioral Therapy (CBT) is a broad category of psychotherapy that encompasses many of the techniques used to treat PTSD. : CBT aims to provide patients with skills to control fear and reduce anxiety and promote habituation. In the case of PTSD treatment, CBT will activate fear by (Solomon, 1992).

Under the umbrella of cognitive-behavioral therapies are exposure based therapies. These therapies involve the confrontation of fear by patients. By exposing patients to objectively harmless, but feared stimuli, anxiety related to these stimuli may be reduced. The temporal length of the exposures can vary, as can the arousal level. There are also various mediums of exposure including imaginal and in vivo. Specific types of exposure therapy include systematic desensitization (SD), and prolonged exposure (PE) therapy (Foa & Meadows, 1997).

Prolonged Exposure (PE) therapy is currently considered the first line treatment for PTSD. Among cognitive behavioral therapies for treatment of PTSD, PE has been the subject of the most extensive study. Prolonged exposure therapy has the greatest number of replications conducted around the

world. It also has the greatest number of comparisons to other evidence-based treatments for trauma. The therapy has been successfully applied to clinical practice, is tolerated well by patients, and has shown treatment gains that are maintained (Nacasch et al., 2011).

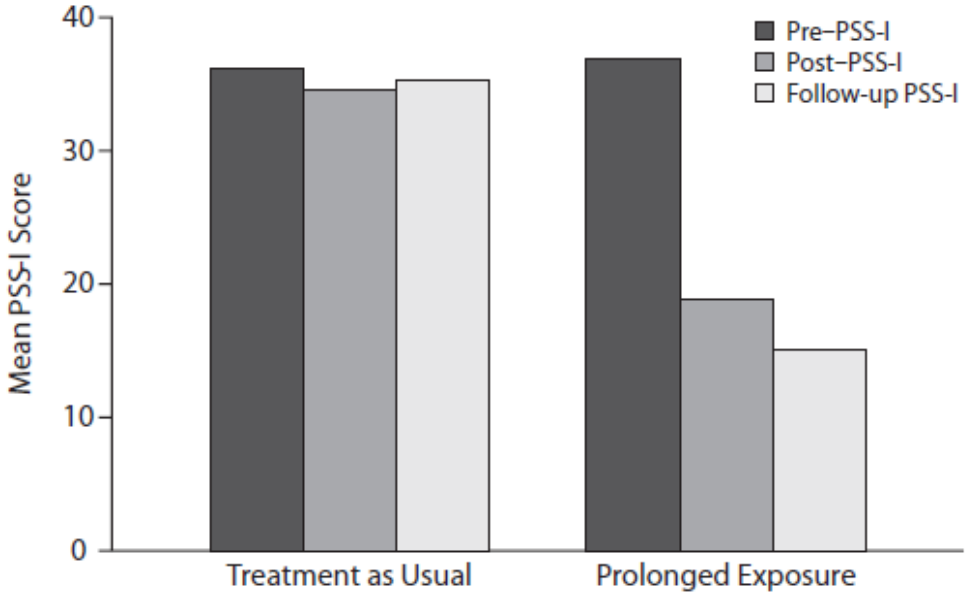

Figure 3. *Plot of Pretreatment and Posttreatment PSS-I Scores in Prolonged Exposure Group Versus Treatment as Usual Group. PSS-I = Posttraumatic Stress Disorder Symptoms Scale-Interview Version (Nacasch 2011)*

Prolonged exposure therapy was developed by Edna Foa in 1994 to include four main components: education-rationale, breathing retraining, behavioral (in vivo) exposures, and imaginal exposures. These four components

are introduced in that specific order over the course of nine treatments sessions. The imaginal exposure sessions are recorded, and clients are instructed to listen to the recording each day. They are also instructed engage in behavioral exposures of increasing difficulty on a daily basis. (Resick et al., 2002)

By systematically exposing patients to memories of traumatic events, they can be desensitized and recognize that they are no longer in danger. The theoretical basis of exposure therapy lies in a model of PTSD as a failure to extinguish the fear response. (Foa & Meadows, 1997b; Osuch et al., 2009). This model of PTSD is built upon theories such as Lang's (1979) bioinformation theory which views fear as a cognitive structure including representations, responses and meaning of specific stimuli. Using this definition of fear combined with Rescorla's (1988) theory of condition as a change in meaning, PE can be viewed as a mechanism by which erroneous associations are corrected. By activating fear and presenting corrective information this deconditioning can be achieved (Foa & Meadows, 1997). Through this deconditioning, patients are able to come to the realizations that "(a) being in objectively safe situations that remind one of the trauma is not dangerous; (b) remembering the trauma is not equivalent to experiencing it again; (c) anxiety does not remain indefinitely in the presence of feared situations or memories, but rather it decreases even without avoidance or escape; and (d) experiencing anxiety/PTSD symptoms does not lead to loss of control" (Foa & Meadows, 1997).

Prolonged Exposure therapy has improved PTSD symptoms in several studies over the past decade. Nacasch (2011) showed that PE is beneficial in the amelioration of combat- and terror-related PTSD symptoms. It was superior to treatment as usual in both the short- and long-term reduction of PTSD and depression symptoms (Nacasch et al., 2011).

Systematic Desensitization (SD) is another type of exposure therapy. SD uses minimally arousing, imaginal, brief exposures. Pioneered by Wolpe in 1958, this technique combines imaginal exposure to feared stimuli with relaxation in a graded hierarchical fashion (Foa & Meadows, 1997a). Though some studies have shown improvement in post-trauma symptoms, they generally lacked the quality and rigor necessary to draw meaningful conclusions (Foa & Meadows, 1997a).

In the past, other types of exposure therapies were called **imaginal flooding** and **implosive therapy**. These therapies did have some success, but have not been studied as extensively as prolonged exposure therapy (Foa & Meadows, 1997).

CBT has been successful, not only in treating PTSD, but in preventing PTSD immediately following an acute traumatic event. Prior to a diagnosis of PTSD, an individual may be diagnosed with Acute Stress Disorder (ASD) following a traumatic event. This disorder has some similar features to PTSD,

but is specific for symptoms that occur within the first month of trauma (DSM-IV-TR, 2000). In a randomized controlled trial of 24 individuals with acute stress disorder, it was found that only 8% of people fulfilled the diagnostic criteria for PTSD following 5 sessions of CBT. This was compared to 83% of patients who fulfilled the diagnostic criteria following 5 sessions of supportive counseling. Additionally, of the patients who received CBT, only 17% fulfilled the diagnostic criteria for PTSD 6 months later. This was compared to 67% of the patients who received the supportive counseling, 6 months later (Bryant, 1998).

Cognitive Processing Therapy (CPT) is a treatment specifically designed for treatment of PTSD resulting from sexual assault. The therapy consists of an exposure component as well as a cognitive therapy component. The exposure component consists of writing and reading about the traumatic event. Case studies and a group therapy format have shown this therapy to be effective in treating PTSD (Resick & Schnicke, 1992; Resick et al., 2002). While Prolonged Exposure therapy is useful to extinguish fear, it is hypothesized that CPT may be more effective for patients coping with guilt. Cognitive therapies may help redirect pathological guilt, whereas the types of therapeutic procedures used in PE may even be harmful for guilt. (Resick et al., 2002). The effectiveness of PE on guilt may depend on the type of guilt that a client is experiencing. Global guilt may be more easily dispelled with exposure therapy, however PTSD patients are often suffering from guilt cognitions relating to

hindsight bias or lack of justification. They often focus on behaviors during the trauma and whether or not the traumatic event may have been preventable. CPT may be effective in treating these specific types of guilt. (Resick et al., 2002).

According to the manual of CPT written by Resick and Schnicke (1993), Cognitive Processing Therapy begins with the writing of an "impact statement" in the first session. The second session teaches clients to identify and relate thoughts and emotions among events. In the third session, clients are assigned to write a detailed account of the trauma for homework and read it back to themselves. In the fourth session, the clients read the account to the therapist. In the fifth session, the account is rewritten with the focus of teaching clients to challenge their beliefs on the meaning of the traumatic event. By challenging beliefs, clients are able to identify problematic patterns of cognition. From sessions 7-12 clients focus on one theme each week to correct any overgeneralized beliefs related to that theme. In session 11 clients rewrite their impact statements to reflect their current beliefs. The final statements are used in the last session to evaluate gains and identify areas that they may wish to continue working on. (Resick et al., 2002). In a study comparing CPT and PE in rape victims, it was found that both therapies were highly successful in treating PTSD, including depressive symptoms. CPT was superior to PE in terms of remediating guilt cognitions, as was previously hypothesized. Both therapies showed equal effectiveness for both recent and chronic PTSD and depression. It seems that the number of years that pass following a traumatic event may not

actually change the final outcome. One potential reason for this is that individuals with PTSD tend to avoid reminders of the trauma. This can cause the traumatic memory to become more or less static in individuals with chronic PTSD (Resick et al., 2002). The findings of this study are strong, as the methods were rigorous and sample size was relatively large (n=121 completed treatment).

Another type of CBT is **couples therapy.** There is a clear relationship associating PTSD with relationship distress and aggression (Monson, 2012; Taft et al., 2011). PTSD can also cause psychological distress in the other partner, and negative interpersonal relationships predict worse treatment outcomes (Caska & Renshaw, 2011; Tarrier, Sommerfield, & Pilgrim, 1999). Previously, uncontrolled trials had shown that couples therapy could improve PTSD symptoms and relationship satisfaction (Monson, 2004; Monson et al., 2011; Monson, 2012; Sautter et al., 2009). Monson et al. conducted the first randomized controlled trial of Cognitive-Behavioral Conjoint Therapy (CBCT) for treatment of PTSD, in which cognitive-behavioral approaches were applied to couples in which one partner had PTSD (Monson CM, 2012). A waiting list for treatment was used as the control condition in this study. Compared to the waitlist condition, those who received the couples therapy had significant improvements in intimate relationship satisfaction, as well as clinically meaningful reductions in PTSD symptom severity. These effects were maintained at three month follow-up. Najavits noted that this study had strengths in including

couples of various sexual orientations, detailed descriptions of treatment, use of validated assessments, blinded evaluation and fidelity monitoring (Najavits, 2012). Najavits criticized Monson's study for leaving out follow-up data on subjects who were on the waiting list as well as those who dropped out of the study. Najavits also criticized the appearance of the sample group as one that was "easy to treat," excluding subjects with severe comorbidities or non-supportive partners. The sample size lacked diversity of race and employment status as well. These flaws pointed out by Najavits make it difficult to apply the results to the general population.(Najavits, 2012)

Group therapy is another subset of CBT. Friendships with people in similar situations can help individuals to feel safer and more open about their symptoms. One case study described a veteran who had confined himself to "his bunker" (basement) for decades, but upon commencing group therapy he formed friendships with other combat veterans, stating that, "They know exactly how I feel." (Carter et al., 2011)

Hypnotherapy is a psychosocial treatment that has been used for many years in the treatment of distress related to trauma. Several case studies have reported hypnosis as being a useful treatment for PTSD, however they lack the methodological rigor necessary to apply these results to practice (Foa & Meadows, 1997a).

Eye Movement Desensitization and Reprocessing (EMDR) is one of the newer cognitive behavioral therapies. It is a form of exposure therapy which is accompanied by saccadic eye movements. While the patient focuses on a disturbing image or memory, the patient tracks the therapist's finger as they wave it across the patient's visual field (Foa & Meadows, 1997a). EDMR has been shown to be equally as effective as PE in terms of reducing anger and guilt (Stapleton, Taylor, & Asmundson, 2006).

Stress Inoculation Training (SIT) is an anxiety management program. The goal of anxiety management programs is to provide ways to manage anxiety when it occurs, as opposed to cognitive therapies which aim to correct the mechanisms that underlie pathological anxiety (Foa & Meadows, 1997a). The treatment protocol for SIT provides patients with coping skills such as education, relaxation, cognitive restructuring and behavioral rehearsal. (Resick et al., 2002) The first controlled trial of SIT was conducted by Resick et al. (1988) and showed improvement in fear and anxiety (Resick et al., 1988). Edna Foa compared SIT to PE and found SIT was a viable treatment for PTSD. However, since both trials came from the same research group and only studied female assault victims, further research is needed to apply the results to the general population (Foa,et al., 1991; Foa & Meadows, 1997b).

Pharmaceutical Interventions

Although evidence-based psychotherapy focusing on the traumatic event (such as PE) is considered the favored treatment for PTSD, pharmacotherapy is also an essential treatment option. Pharmacotherapy may be used in conjunction with psychotherapy, or when psychotherapy fails to be successful in an individual. The theory supporting pharmaceutical treatment of PTSD is that trauma is a psychobiological event, which can cause long-term neurobiological changes in the brain (Solomon, 1992). Several studies aim to determine what these changes are in order to best treat them with medication. Understanding the chemical changes that occur in the brain can help to develop pharmacological treatments. There is a considerable amount of research devoted to the study of these neurobiological changes in the brain that occur with PTSD. It has been shown that individuals with PTSD have disturbances in the neurological integration of the fear circuitry, which is important in the "fight or flight" response (Jeffreys et al., 2012; Shin & Handwerger, 2009). Studies have also shown that the hypothalamic-pituitary-adrenal regulation is disrupted (De Kloet et al., 2006; Jeffreys et al., 2012; Wessa et al., 2006; Yehuda, 2001). There is also evidence that key areas such as the amygdala have reduced serotoninergic modulation of the fear response (Jeffreys et al., 2012; Murrough et al., 2011). In addition to these lasting changes in the brain, there is research that focuses on the changes that occur in the brain in the immediate aftermath of trauma. For example, it has been shown that prolonged adrenergic immediately

following trauma has been linked to increased risk for PTSD (Cukor et al., 2009; Vaiva, 2003).

Potential pharmacological interventions include tricyclic antidepressants (TCAs), mono-amine oxidase inhibitors (MAOIs), selective serotonin reuptake inhibitors (SSRIs), selective norepinephrine reuptake inhibitors (SNRIs), anxiolytic medications, mood stabilizers, and antipsychotics (Hetrick et al., 2010). The most common pharmacological intervention is SSRIs. Pharmacological interventions may also be combined with psychological interventions, however further research needs to be done to determine the efficacy of each intervention alone, as well as combinations (Hetrick et al., 2010).

According to the Department of Veterans Affairs/Department of Defense Clinical Practice Guidelines, first-line pharmacotherapy agents include SSRI and the SNRI venlafaxine. Second-line agents include nefazodone, mirtazapine, TCAs, and MAOIs. These agents are considered second line because they have less evidence for usefulness in treatment of PTSD, and have greater potential for side effects (Jeffreys et al., 2012).

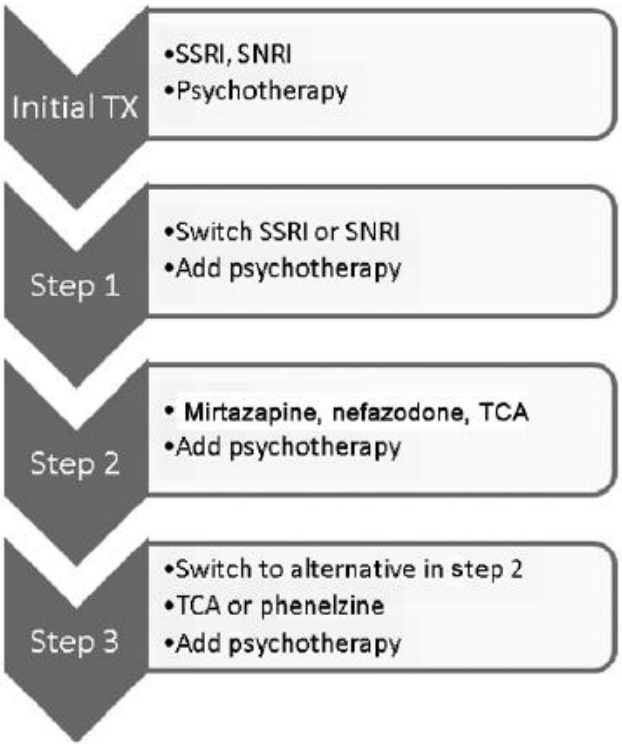

Figure 4. *Suggested lines of treatment in pharmacotherapy for PTSD. Adapted from VA/DoD Clinical Practice Guideline for Management of Post-Traumatic Stress.*(Jeffreys et al., 2012)

The only two medications currently approved by the Food and Drug Administration for treatment of PTSD are the SSRIs sertraline and paroxetine. Both of these medications help to reduce the three core symptom clusters of PTSD: re-experiencing, hyperarousal, and avoidance. These medications are hypothesized to be effective based on their affect on the serotonergic receptors, which as mentioned previously, have been shown to be altered in the amygdala of patients with PTSD. Both drugs have been shown to be effective by

randomized controlled trials (Brady, 2000; Jeffreys et al., 2012; Marshall et al., 2007).

One of the most commonly prescribed SSRIs is **sertraline** (trade names Zolft, Lustral). Bradey et al. (2000) conducted a large (n=187), multi-center, randomized placebo-controlled study to examine the efficacy of sertraline for the treatment of PTSD. They found sertraline to be significantly more effective than placebo for the treatment of PTSD as measured by a number of assessments. Those taking sertraline had a 70% reduction in PTSD symptom severity in the first four weeks on the Clinician Administered PTSD Scale Part 2 (CAPS-2) and Impact of Event Scale (IES). This is compared to the placebo response rate of 32% (Brady, 2000). They also found that 53% of subjects were either much or very much improved by the end of treatment (p=.008 vs placebo) (Brady, 2000).

In a double-blind randomized controlled trial of **paroxetine** for chronic PTSD, subjects who received paroxetine were significantly more responsive than those on placebo, on a number of measures. 66.7% of those receiving paroxetine were classified as responders based on the Clinical Global Impression—Improvement Scale (CGI-I), as compared to 27.3% of those treated with placebo. Subjects receiving paroxetine also showed greater reduction of dissociative symptoms and self-reported interpersonal problems. Those receiving paroxetine

continued to improve during a 12-week maintenance phase, while those receiving placebo did not (Marshall et al., 2007).

Other SSRIs have shown varying effectiveness. **Fluoxetine** (Prozac, Sarafem, Fontex) has been shown to be superior to placebo in terms of treating PTSD symptoms, as well as preventing relapse (Davidson et al., 2005; Jeffreys et al., 2012; Martenyi & Soldatenkova, 2006).

Citalopram (Celexa, Cipramil) and **fluvoxamine** (Luvox) have been less studied for their effectiveness in the treatment of PTSD (Jeffreys et al., 2012).

The SNRI **venlafaxine** (effexor) is considered an off-label use in the treatment of PTSD, as it is not FDA approved for the treatment of PTSD (Jeffreys et al., 2012). A randomized controlled trial has shown strong evidence that venlafaxine is effective in the treatment of PTSD (Davidson, 2006; Jeffreys et al., 2012).

Second-line medications recommended by the VA/DoD CPG include tricyclic antidepressents (**TCAs**) and monoamine oxidase inhibitors (**MAOIs**). These medications are effective, but have considerably more side effects than SSRIs and SNRIs. There is a relatively small difference between the therapeutic and toxic dosages of these medications, so they carry a risk of overdose (Jeffreys et al., 2012).

Also included in the list of second-line agents is **Mitrazapine (**Remeron, Avanza, Zispin), which can be a useful alternative to those who suffer from sexual side effects of SSRIs. It acts on the serotonin system, and at high doses demonstrates noradrenergic stimulation (Chung et al., 2004; Jeffreys et al., 2012).

Nefazodone (Serzone, Nefadar) is also recommended as a second-line medication, however it should be used with extreme caution. Liver failure has been reported in about 1:300,000 cases, so it is important to closely monitor liver functions in conjunction with using this medication. Nefazodone has been shown to ameliorate some of the hyperarousal symptoms of PTSD such as sleep disturbance and anxiety (Davis et al., 2004; Jeffreys et al., 2012).

Benzodiazepines and **antipsychotic** drugs have been used in the past for the treatment of PTSD despite a lack of evidence for their efficacy. The prescription of benzodiazepines for PTSD can actually be quite harmful as they mask the outward symptom of anxiety without addressing the underlying PTSD. This may lead clinicians to believe that the patient has improved, when they in fact still remain untreated for their PTSD. Benzodiazepines also have a higher potential for abuse than other drugs, and are thus contraindicated for patients with addiction problems. Benzodiazepines have also been shown to interfere with fear extinction which is an integral part of prolonged exposure therapy

(Jeffreys et al., 2012). Like benzodiazepines, antipsychotics have not been shown to be effective for PTSD treatment. A large multisite VA cooperative study showed that the antipsychotic **risperidone** (Risperdal) was no more effective than placebo at decreasing PTSD symptoms over 24 weeks (Krystal, 2011, 2011). Antipsychotics also carry a risk of harmful side effects such as obesity and metabolic syndrome (Jeffreys et al., 2012).

In addition to the established first- and second-line pharmaceutical treatments, there are also several emerging pharmaceuticals that are not yet approved for PTSD treatment.

D-cycloserine (also known as DCS, Seromycin) holds the most significant potential as an emerging pharmacological treatment for PTSD (Cukor et al., 2009). D-cycloserine is a broad spectrum antibiotic, which has been used as a cognitive enhancer over last decade. It acts as a partial agonist at the N-methyl-D-aspartate (NMDA) receptor, which plays an essential role in learning and memory. D-cycloserine has also had positive results in the treatment of acrophobia, social phobia, and social anxiety. It is hypothesized that D-cycloserine may facilitate fear extinction, enhancing or accelerating treatment effects of Prolonged Exposure or Virtual Reality (Cukor et al., 2009).

Propanolol is a non-selective beta-adrenergic blocker used for hypertension. The theory behind its use in PTSD is that there is excessive epinephrine released during trauma, and that administration of propanolol immediately after may prevent development of PTSD (Cukor et al., 2009; Vaiva, 2003).

Ketamine is a non-barbituate anesthetic, which acts as an antagonist at the NMDA receptor, and may cause disruption of memory process. It is associated with dissociation and psychosis, which raises the concern that it may worsen PTSD (Cukor et al., 2009). Surprisingly a retrospective chart review of OEF/OIF veterans showed that those who received ketamine had lower rates of PTSD than those who did not (McGhee et al., 2008).

Prazosin is an alpha-1 adrenergic receptor blocker, used for hypertension and benign prostatic hyperplasia (Cukor et al., 2009). It has proved to be effective in treating PTSD-related nightmares (Calohan et al., 2010; Jeffreys et al., 2012; Taylor et al., 2008).

MDMA (Methylenedioxymethamphetamine, Ecstasy) is a drug that results in positive moods, improved trust and emotional alliance. It may contribute to successful treatment by helping to strengthen the therapeutic alliance. However, negative emotions and psychobiological distress in aftermath seem to contraindicate its use in vulnerable populations (Cukor et al., 2009)

Clonidine is an alpha-2 adrenergic receptor agonist used to treat conditions such as hypertension, ADHD and a number of other conditions ("Clonidine: MedlinePlus Drug Information," n.d.). By blocking reconsolidation of persistent traumatic memories it may be effective in the treatment of PTSD. In animal models it has been shown to disrupt fear-related memories in a dose-dependent manner, with lasting effects (Gamache, Pitman, & Nader, 2012).

Table 1. *Brain Stimulation Modalities. Key source: www.fda.gov* (Novakovic et al., 2011).

Electrical		Magnetic	
Convulsive	*Non-convulsive*	*Convulsive*	*Non-convulsive*
External stimulation	*External stimulation*	*External stimulation*	*External stimulation*
ECT FDA: Class III device (Depression), 1979, no PMA	CES FDA: Sanctioned 1991 (sleep and anxiety) use may or may not be supervised by a professional tDCS No FDA approved Indication	MST No FDA approved indication	rTMS FDA: General approval (Depression (TRx1); Migraine prophylaxis) 2008; trials ongoing
Internal stimulation/ implanted	*Internal stimulation/implanted*	*Internal stimulation/ implanted*	*Internal stimulation/implanted*
N/A	DBS FDA: General Approval 1997 (Essential Tremor); General Approval 2002 (Parkinson's); HDE 2003 (Dystonia); HDE 2009 (OCD); Trial completed, PMA decision pending (Epilepsy); Trials ongoing (TRD) DCS No FDA approval; experimental only VNS FDA: General Approval 1997 (Epilepsy); General Approval 2005 (TRD) DCS No FDA approval; experimental only VNS FDA: General Approval 1997 (Epilepsy); General Approval 2005 (TRD)	N/A	N/A

Brain Stimulation Interventions

Individuals suffering from chronic PTSD often are not cured by psychotherapy and/or pharmacotherapy. Brain stimulation offers an option which can be used as either an alternative to, or in conjunction with other treatment modalities. Brain stimulation has been a useful treatment in psychiatry for several decades, but has not been extensively studied for the treatment of PTSD. It is hypothesized that the efficacy of other treatments may be in part due to relief of associated depression and anxiety. Brain stimulation may be able to address PTSD in a more targeted way than other methods. The table below shows brain stimulation interventions and the conditions they are FDA approved to treat, and illustrates the differences between convulsive and non-convulsive; electrical and magnetic; and internal and external modalities (Novakovic et al., 2011).

Vagus nerve stimulation (**VNS**) involves electrical pulses to activate vagal afferents. It is invasive, as it requires surgical implantation of a device, but following the implantation the stimulation may continue wherever patients travel. VNS is FDA approved for the treatment of epilepsy and treatment resistant depression (TRD) (Novakovic et al., 2011). VNS is hypothesized to be a potential treatment for anxiety disorders because the vagus nerve is involved in sending information to regions of the brain that are important for anxiety regulation. When used for epilepsy and TRD patients have reported anxiolytic effects, confirming this hypothesis. An open-label trial of VNS was well-tolerated

in subjects with a number of treatment-resistant anxiety disorders including panic disorder, obsessive compulsive disorder (OCD) and PTSD. There was some efficacy in reducing anxiety, but this was primarily observed in the subjects with OCD. There were only 2 subjects with PTSD, one of whom improved their anxiety ratings 41%, while the other had a worsened score by 3%. It is difficult to draw conclusions about the effects on PTSD from this limited data, and future study may be limited due to the invasiveness of this procedure (George et al., 2008).

Transcranial Magnetic Stimulation (**TMS**) is a way of inducing cortical neurons to fire. Repetitive TMS (rTMS) was approved by the FDA for treatment of major depression as well as migraine prophylaxis in 2008 (Novakovic et al., 2011). Prefrontal rTMS has been shown to increase cerebral oxygen perfusion at high-frequency (20 Hz) and decrease cerebral oxygen perfusion at low-frequency (1 Hz) (Speer et al., 2000). A placebo controlled trial of high-frequency (10 Hz) rTMS showed positive effects for treatment of PTSD (Cohen et al., 2004). Unfortunately these results have not yet been proven repeatable. A more recent trial of rTMS combined with exposure therapy was also largely negative in results, although there was some reduction in hyperarousal. This study was limited by sample size (n=9) and suggested that further exploration of optimal rTMS parameters is warranted. (Osuch et al., 2009).

Electroconvulsive Therapy (ECT) is the oldest form of brain stimulation. It was first introduced in 1938 and is regarded as a highly effective treatment for severe depression, mania and catatonia. Electricity is applied to the scalp under general anesthesia to induce a seizure. While the mechanisms are not completely understood, it is known that seizure induction is necessary for therapeutic effect. It may be administered unilaterally or bilaterally, and other forms are being investigated. While ECT has not been intensively studied for PTSD, it may have some efficacy for reducing comorbid depression (Novakovic et al., 2011). Two case studies of women with co-morbid depression and PTSD have had positive results (Novakovic et al., 2011). Margoob (2010) performed a prospective study of patients with severe, chronic, treatment-resistant PTSD. They received six bilateral ECT treatments and had a response rate of 82%, with a mean CAPS improvement of 40%. These treatment gains were maintained at 4-6 month follow-up (Novakovic et al., 2011).

Cranial electrotherapy Stimulation (**CES**) is unique in that it can be self-administered, unlike all other brain stimulation modalities. It uses a small alternating current to affect brain function. CES has been FDA approved for the treatment of depression, anxiety and insomnia since 1978. It has yet to be studied for the treatment of PTSD, however it has been shown to reduce anxiety, which is promising (Novakovic et al., 2011).

Magnetic Seizure Therapy (**MST**) produces more focal seizures than ECT by applying high-intensity TMS under anesthesia. There is no FDA approved indication for MST at this point, and much more research needs to be done before it can be used as a treatment for PTSD (Novakovic et al., 2011).

Deep Brain Stimulation (**DBS**) is the most invasive and the most focal of the brain stimulation modalities. It requires an electrode be surgically implanted to administer electrical stimulation. DBS is FDA approved for a number of neurological conditions such as Parkinson's and Epilepsy. It is also approved for treatment resistant depression (TRD). It has not yet been studied for PTSD, as specific targets in the must be identified first (Novakovic et al., 2011).

Transcranial Direct Current Stimulation (**tDCS**) involves the polarization of brain tissue application of weak direct electrical currents to the scalp via sponge electrodes. This results in changes of the excitability of the cortex that continue after the period of stimulation, in a similar manner to rTMS. There is currently no FDA approved indication for the use of tDCS (Novakovic et al., 2011).

Comorbidities

PTSD has an extremely high rate of occurrence with other comorbid conditions. Studies have shown rates varying between 62% and 92% of subjects with PTSD having comorbidities (Kessler, 1995). Whether PTSD is the cause or the effect of this high rate of comorbidities remains to be seen. The national comorbidity study showed that PTSD was primary more often than not with comorbid affective disorders and substance use disorders. However, PTSD was less likely to be the primary disorder with respect to anxiety disorders. (Kessler, 1995) In studies of PTSD, comorbidities may frequently be cause to exclude patients from the trial in order to narrow the focus of the study to PTSD alone. This exclusion, however changes the sample to be one that is not at all representative of the general population. When subjects are not excluded for comorbidities, the percentage of comorbidities is very high. For example, in Osuch's 2009 study of rTMS for PTSD, all subjects met criteria for current major depression and all subjects had a history of prior substance abuse (Osuch et al., 2009).

Traumatic Brain Injury (TBI) is defined by the Department of Defense and Department of Veterans Affairs (2009) as "a traumatically induced structural injury and/or physiological disruption of brain function as a result of an external force that is indicated by new onset or worsening of at least one of the following clinical signs, immediately following the event: any period of loss of or a

decreased level of consciousness (LOC), any loss of memory for events immediately before or after the injury (post-traumatic amnesia [PTA]), any alteration in mental state at the time of the injury (confusion, disorientation, slowed thinking, etc.) (alteration of consciousness/mental state [AOC]), neurological deficits (weakness, loss of balance, change in vision, praxis, paresis/plegia, sensory loss, aphasia, etc.) that may or may not be transient, and intracranial lesion. (p. 7) ("VA/DoD Clinical Practice Guidelines," 2009).

TBI may be categorized as mild, moderate or severe. When symptoms persist for an extended period of time, they are termed Postconcussive symptoms or syndrome (PPCS). These symptoms can be divided into three categories: somatic, psychological and cognitive (Wall, 2012).

It can be difficult to differentiate symptoms of PTSD from those of a mild TBI. Sleep disturbances, increased arousal, irritability and decreased concentration are common features of both conditions (Jeffreys et al., 2012).

Substance abuse is one of the most common comorbidities seen in individuals suffering from PTSD. According to a national epidemiological survey, more than 40% of women in the U.S. diagnosed with PTSD also met criteria for an alcohol use disorder (Pietrzak et al., 2011). Additionally, in a survey of OEF/OIF/OND infantry teams, 25% reported alcohol misuse post-deployment. The greatest predictor of alcohol-related problems upon returning home was the experience of direct threat of death and injury while deployed (Wilk et al., 2010).

Furthermore, it was shown that service members who experienced multiple deployments had increased alcohol-related issues than those who had only deployed once (Carter et al., 2011).

There are multiple theories as to why there is such a high rate of comorbid PTSD and substance abuse. One pervasive theory is that substances are used as self-medication. Many individuals are believed to be using alcohol to alleviate symptoms & reduce negative emotions (Carter et al., 2011; Waldrop et al., 2007).

A second theory for the high rate of comorbid PTSD and substance abuse is that there is a common factor underlying both problems. Individuals may possess a lack of awareness and understanding of their emotions, as well as an inability to control behaviors when in emotional distress (Bornovalova et al., 2009; Brady, 2000; Carter et al., 2011).

Surveys have shown that professionals find comorbid PTSD and substance use disorder more difficult to treat than PTSD alone (Najavits, 2012). In addition, it has been shown that a diagnosis of co-occurring alcohol use disorder with PTSD is more detrimental than diagnosis of either alcohol use disorder or PTSD alone (Carter et al., 2011).

The traditional treatment for comorbid PTSD and alcohol use disorders is to treat each condition separately in a sequential manner. This approach has proven problematic because of high dropout and relapse rates (Carter et al., 2011) Another approach, termed parallel treatment, is to treat the two disorders simultaneously but separately. This is preferable to sequential treatment,

however can be uneven and confusing for patients as treatment may not be consistent across separate providers and clinics (Carter et al., 2011; Karlin et al., 2010). Ideally, treatment of PTSD and alcohol use disorder would be integrated.

While trauma focused therapies have been shown to be the most successful for treating PTSD alone, clinicians are reluctant to implement these therapies in those with comorbid substance abuse for fear that it may be unsafe. They fear that triggering a negative affect may cause patients to engage in riskier behaviors, as supported by the self-medication hypothesis.

"Until recently… many experts and clinicians considered the use of prolonged exposure therapy among individuals with substance dependence to be inappropriate unless a lengthy period of abstinence had been achieved. Based on early case reports, it was widely believed that intense emotions elicited during prolonged exposure could place individuals at increased risk for relapse. There is however, an absence of evidence to support or refute this recommendation, because most trials of PTSD treatment have excluded individuals with substance dependence." (Bradley et al., 2005; Mills, 2012).

In order to combine treatment, coping skills could be taught alongside standard PTSD interventions, such as prolonged exposure. One such study has shown reductions in both PTSD and alcohol use disorder symptoms. (Coffey et al., 2006). Unfortunately these integrated treatments show high attrition rates, so therapies that are not exposure-based should also be explored (Coffey et al., 2010). Najavits (2002) explored a coping-skills based group therapy for co-

occurring PTSD and substance abuse, entitled “Seeking Safety”. Though this trial demonstrated some efficacy, others have had mixed results, and a pilot of Seeking Safety with veterans had significant engagement and retention difficulties (Carter et al., 2011; Norman et al., 2010).

One treatment being studied for the treatment of comorbid PTSD and substance is termed “Concurrent Treatment of PTSD and Substance Use Disorders Using Prolonged Exposure (COPE)” (Back et al., 2012; Mills, 2012).

Back’s 2012 case study shows an ideal example of COPE. It explains how an individual progressed through therapy and had significant improvements (Back et al., 2012).

Mills’ 2012 RCT of COPE showed that subjects improved PTSD symptoms, but there was no significant difference with or without substance abuse (Table 2). This is the first RCT to assess efficacy of PE for PTSD in subjects with co-occurring substance dependence. Najavits (2012) praises this trial for the “inclusion of well-trained clinicians, monitoring of treatments quality, measurement of amount of therapies provided as usual treatment, validated measures of patient outcomes, and appropriate statistical analyses.” They also lauded the broad range of subjects included, which are usually excluded from other studies. The subjects in the Mills experiment were not paid, which strengthens the data further, as payment may artificially inflate results. (Najavits, 2012)

Table 2. *Categorical Measures of Outcome in COPE treatment (Mills KL, 2012)*

Outcome Measure	No. (%)			
	Baseline	6 wk	3 mo	9 mo
Abstinent, %[a]				
COPE + usual treatment (n = 55)	0	12 (21.8)	10 (18.2)	10 (18.2)
Usual treatment only (n = 48)	0	15 (31.3)	12 (25.0)	13 (27.1)
Between-group difference at each interview, OR (95% CI)[b]	NA	0.59 (0.24 to 1.46)	0.70 (0.24 to 1.99)	0.59 (0.21 to 1.65)
Diagnosis of substance dependence, %[a]				
COPE + usual treatment (n = 55)	55 (100)	26 (47.3)	26 (47.3)	25 (45.4)
Usual treatment only (n = 48)	48 (100)	28 (58.3)	28 (58.3)	27 (56.2)
Between-group difference at each interview, OR (95% CI)[b]	NA	0.64 (0.25 to 1.63)	0.62 (0.25 to 1.54)	0.64 (0.28 to 1.48)
Diagnosis of PTSD, %[a]				
COPE + usual treatment (n = 55)	55 (100)	48 (87.3)	47 (85.4)	31 (56.4)
Usual treatment only (n = 48)	48 (100)	45 (93.8)	43 (89.6)	38 (79.2)
Between-group difference at each interview, OR (95% CI)[b]	NA	0.41 (0.06 to 2.63)	0.68 (0.19 to 2.44)	0.32 (0.13 to 0.81)[c]

Abbreviations: COPE, Concurrent Treatment of PTSD and Substance Use Disorders Using Prolonged Exposure; NA, not applicable; OR, odds ratio; PTSD, posttraumatic stress disorder.

[a] Group × time interaction effect not significant at $P < .05$.

[b] Reference category is the control group.

[c] $P = .02$.

Major depressive disorder (MDD) has a number of overlapping symptoms with PTSD, such as irritability, social withdrawal and sleep disturbance. For this reason it is not uncommon for patients to meet the diagnostic criteria for both PTSD and MDD (Jeffreys et al., 2012).

SUGGESTIONS FOR FUTURE RESEARCH

While research surrounding PTSD treatments is on the rise, and has become a point of focus in the news as of late, there still exists many questions to be answered, research to be done and problems to address.

One major issue, which is difficult to address, is that experimental data contains gaps due to exclusion and inclusion criteria. Some of the most typical PTSD patients have comorbidities or other conditions which exclude them from PTSD research. Najavits (2012) observes that many groups have been consistently excluded in clinical trials of PTSD treatments to date. Clinicians routinely encounter complex cases of PTSD, and a typical individual suffering from PTSD may be excluded from a clinical trial for reasons including comorbid conditions such as substance use disorder; suicidal ideation; histories of self-harm, homelessness, and intimate partner violence (Najavits, 2012).

Even when individuals suffering from PTSD are not involuntarily excluded from research, the disorder itself may prevent them from participation. Estrangement from others and decreased participation in activities are common symptoms, and when coupled with a sense of hopelessness, a patient may be less than willing to participate in any medical care, much less research (Jeffreys et al., 2012).

When subjects with PTSD overcome the above barriers and are successfully enrolled in clinical trials, it is not uncommon for subjects to drop out of research studies, which may skew results. Variables that might affect

treatment completion is an important topic for future research (Resick et al., 2002).

CPT has been shown efficacious as a treatment for PTSD, but studies so far have only been done with rape victims. It is necessary to test with other populations in order to ensure generalizability. Effectiveness studies are also needed in for generalization to non-research settings (Resick et al., 2002).

While there is a significant amount of research on comorbid conditions such as TBI and substance abuse, there is very little for Axis II disorders and bipolar disorder. These disorders are difficult to treat alone, and become more complicated with combined with PTSD. There have been some studies on the use of mood stabilizers and antipsychotics for comorbid bipolar disorder with PTSD, but these have had little success (Jeffreys et al., 2012). Further research needs to be done on the best course of treatment in cases such as these.

In addition to the areas described above where research could be improved, another important issue to address is the gap between research and practice. Even when a treatment is deemed successful by researchers, and the evidence and recommendations are disseminated to providers, the treatments may, and often are not, implemented (Karlin et al., 2010).

Even when clinicians do have the desire and resources to apply research to practice it can be confusing to figure out what the best course of treatment is. Several clinical practice guidelines exist; to the extent that Forbes et al. (2010) wrote a “Guide to Guidelines” in order to assist clinicians in sorting out these

recommendations and making decisions about their use. This review explained that, “clinical practice guidelines are systematically developed statements to assist practitioner and patient decisions about appropriate health care for specific clinical circumstances.” They performed a systematic review of seven of these guidelines for the treatment of PTSD-- VA/DoD, APA, NICE, NHMRC, ISTSS, AACAP and IOM. (Forbes et al., 2010) The guidelines review showed many areas of concurrence in their recommendations for treatment of PTSD. The use of trauma-focused psychological treatment was strongly supported by all guidelines. All guidelines also acknowledged some benefit of pharmacotherapy. SSRIs were the most commonly recommended pharmacotherapy, though the degree to which they were recommended varied. Some guidelines recommended that SSRIs only be used as a second line intervention. EMDR was mentioned in all guidelines, however there was disagreement as to whether it was acceptable as a first line treatment.

When evidence-based treatments are successfully implemented in practice, obstacles and barriers to care still remain. There are unique problems to both veterans and active duty when it comes to having access to the treatment they need for PTSD. One case study describes veteran did not seek treatment for many years because a bad experience with the VA in the 70’s caused him to distrust the “system” (Back et al., 2012). One major factor is underreporting, which in itself has a variety of causes. Among active duty personnel there is a

stigma associated with mental health disorders and fear of negative effect on one's career. (Hoge et al., 2004; Iversen et al., 2011; Wall, 2012).

Additional problems include lack of anonymity and lack of time off for active duty personnel (Iversen et al., 2011). These barriers to care are not exclusive to PTSD, but apply to other mental health problems as well. Despite a high prevalence of alcohol misuse in military populations, the rates of those seeking help for alcohol problems are particularly low (Iversen et al., 2011). In addition, personnel with anxiety or depression are twice as likely as those who are well to report stigmatizing beliefs about mental health problems (Hoge et al., 2004; Iversen et al., 2011).

In an anonymous screen of personnel returning to the U.S. from Iraq and Afghanistan, 15.7% met broad criteria for PTSD. This rate is statistically significant from the 7.8% who met broad criteria in a retrospective review of the mandated non-anonymous screen. This result may indicate underreporting of PTSD, however it should not be overlooked that the anonymous screen was voluntary, whereas the on-the-record screenings are mandated for service members returning from deployment (McLay, 2008).

As different types of therapy are explored, clients should be matched with specific therapies based on their individual symptomology. As certain treatments have shown to be more effective at ameliorating specific symptoms, there may be variation in which treatment is effective for an individual patient.

Translation from research to practice should be another area of focus for mental health professionals. Despite the strong evidence and practice guidelines that support the use of CBT for PTSD, many mental health clinics are still using psychodynamic-oriented, supportive therapy as a common treatment for PTSD (Nacasch et al., 2011).

Literature on this topic is very recent, as the term PTSD was only coined in the 1970's, when the disorder started to become more widely recognized following the war in Vietnam. Of course, the disorder existed previously, but research has only taken off recently. The disorder was added to the third edition of the Diagnostic and Statistical Manual of Mental Disorders in 1980.

One important point to consider is that different treatments may work differently on specific trauma populations. Veterans have a very different experience than other trauma survivors. While many sufferers from PTSD often have reactions of guilt and shame, these feelings often have rational foundations in veterans. A survivor of rape or assault may blame themselves for allowing the incident to have happened, which most would view as an irrational thought. A veteran who killed innocent civilians has more of a rational basis for his guilt, and thus may resist the attempts to challenge these feelings. Instead of trying to decondition this thought process, it may be more constructive to explore was of bearing witness and making reparations through vehicles such as volunteer work (E. B. Foa & Meadows, 1997a).

Each group of PTSD patients has a unique set of factors to consider. Victims of sexual assault, for example, must face the fear that their trauma may reoccur without warning. This is not something that veterans have to face, as they have been removed from the setting of their trauma. Victims of childhood abuse also have unique circumstances, as their trauma may have altered their development. Oftentimes, victims of childhood abuse must reconstruct what they view as normal, which is a unique challenge in treatment (Foa & Meadows, 1997a).

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