Annotated Bibliography for Articles on PTSD Treatments


The authors of this article point out the potential criticism of Biofeedback, EMDR, and Relaxation Training research is the lack of comparison with other treatments. The study was carried out to address this lack in the literature. Participants were Vietnam War veterans receiving inpatient treatment for posttraumatic stress disorder. Participant’s responses to a set of scales, the Problem Report Form (PRF), were collected, during evaluation, at admission more than two months later, and at discharge, 90 days after admission. The PRF has eight Likert-like scales which measured areas that veterans regarded as critical. Comparing the relative effects of the incremental addition of Eye Movement Desensitization and Reprocessing (EMDR), Relaxation Training, and Biofeedback found that EMDR was for most problems (including: nightmares, intrusive thoughts, flashbacks, anxiety, anger, depression, and relationship problems) the most effective extra treatment, greatly increasing the positive impact of the treatment program.


Marshall et al. (2001) set out to determine the efficacy of the SSRI paroxetine (compared to control) in treating chronic PTSD. This was the first study to demonstrate that an SSRI could effectively ameliorate each of the major symptom clusters of PTSD (re-experiencing, avoidance/numbing, and hyperarousal). Paroxetine-treated patients in both 20mg/day and a 40
mg/day groups demonstrated significant improvement on primary outcome measures compared to placebo. This study also demonstrated that paroxetine treatment response did not vary for gender, trauma type, time since trauma, or severity of baseline PTSD or depressive symptoms. Compared to placebo, patient subgroups with and without a baseline diagnosis of major depressive disorder (MDD) also demonstrated PTSD symptom reduction. The authors conclude that the results of this study provide evidence that paroxetine treatment at 20 and 40 mg/day is effective and well tolerated in the treatment of adult men and women with PTSD. A limitation of this study is that it did not explore long-term treatment outcomes.


Kornør et al. (2002) conducted a meta-analysis (7 articles) to evaluate the efficacy of early trauma-focused cognitive-behavioral therapy (TFCBT) in comparison to supportive counseling (SC) on the prevention of PTSD in high-risk populations. Studies included met the following criteria: 1) randomized controlled trial (RCT) published in peer-reviewed scientific journal, 2) study population of adults with symptoms of acute stress disorder (ASD) or symptoms of post-traumatic stress disorder (PTSD), 3) individual TFCBT initiated within three months post trauma, 4) a non-pharmacological comparison intervention, 5) outcomes measured as symptoms and/or diagnosis of PTSD (primary outcome), anxiety and/or depression (secondary outcomes) at follow-up (minimum one month after treatment completion). Results indicated that TFCBT is more effective than SC in preventing chronic PTSD in patients with an initial ASD diagnosis.
There was insufficient evidence to support the effectiveness of TFCBT in preventing PTSD in traumatized populations without an ASD diagnosis. This evidence originated from one research team. Therefore, replications are necessary to assess the generalizability of these findings.


The author’s goal of this article was to “begin a dialogue among those conducting research on the biological aspects of PTSD and those professionals concerned with developing effective psychological treatments from PTSD”. This article presents a basic overview of important biological findings related to PTSD including findings related to: psychophysiology, the neuroendocrine system, the hippocampus, event related electroencephalographic potentials, and sleep research. Each subsection ends with a discussion of clinical implications of the research findings provided. The article concludes by discussing various empirically validated psychological treatments for PTSD including: exposure therapy, anxiety management training, combined treatment approaches, treatment of sleep-related symptoms, and other innovative treatments approaches for PTSD including acceptance and commitment therapy & interpersonal psychotherapy. A brief discussion about early intervention and prevention of revictimization is also provided.


In this review Yehuda (2002) discusses scientific developments related to the neurobiology of PTSD and their treatment implications. The author discusses the findings of low cortisol in individuals with PTSD as well as the role the amygdala plays in the initiation of the stress
response. Yehuda also discusses the role the hypothalamic-pituitary-adrenal axis plays in the eventual activation of the adrenal gland and subsequent release of cortisol. After discussing a number of studies investigating cortisol levels in different populations of PTSD sufferers, Yehuda suggests the possibility that preexisting low levels of cortisol (before trauma) may account for the development of PTSD. The most common complaint of trauma survivors with PTSD is that they feel misunderstood. The author suggests that the therapeutic value of urging patients to “get on with their lives”, a common practice of mental health workers, is limited by the biological stress response that has never been properly terminated in individuals with PTSD. Yehuda recommends how this knowledge can aid in improving the therapeutic process.


This article reviews available research data supporting the use of psychotherapy in the treatment of PTSD. Several interventions are discussed including: critical incident stress debriefing, psycho-education, exposure therapy, EMDR, stress inoculation therapy, trauma management therapy, cognitive therapy, psychodynamic therapy, hypnotherapy, image rehearsal, memory structure intervention, interpersonal psychotherapy, and dialectical behaviour therapy (DBT). They conclude that most methodologically robust studies indicate that psychotherapy helps to relieve symptom severity; but caution that there is no consistent information about whether these interventions are helpful in improving other domains of impairment and associated disability, even though these problems are often the greatest concern to patients. Furthermore, the authors conclude that the available evidence does not indicate when, and for whom, various psychotherapeutic therapies should be provided, or whether different modalities of treatment can
and should be combined, or sequentially offered. The authors end by providing some clinical guidelines based on the literature for clinicians treating patients with PTSD.


Tucker et al. (2004) explain that PTSD often co-occurs with a number of inflammatory conditions. This article examines the complex relationship between the immune system which has reciprocal communication with and influences the hypothalamic pituitary adrenal (HPA) axis. Specifically the authors aimed to examine the effects of double blind treatment of PTSD with SSRI’s and placebo on cortisol and on two cytokines (cytokines are chemical modulators of the immune response) affecting different aspects of immune function. They set out to determine whether response to treatment is reflected by changes in the interrelated neuroendocrine and neuroimmune measures. The results of this study demonstrate that in physically healthy, untreated PTSD patients there are robust differences in two cytokines important in diverse aspects of immune functioning and that these cytokines normalize with improvement of PTSD and depressive symptoms (interestingly whether with SSRI or with placebo treatment). Lastly, these results suggest that the interactive influences of the neuroimmune and neuroendocrine systems noted in healthy subjects may be restored with treatment.


The authors of this article present findings from a multi-dimensional meta-analysis of studies published between 1980 to 2003 on psychotherapies from PTSD. Study selection criteria limited
the meta-analysis to studies testing a specific psychotherapeutic treatment for PTSD for efficacy against a control condition, an alternative credible psychotherapeutic treatment, or a combination of two or more of these. Studies included had to use a validated self-report measure of PTSD symptoms or a validated structural interview. Participants were limited to adults. Results indicated that psychotherapy for PTSD leads to a large initial improvement from baseline. More than 50% of patients treated with various forms of CBT or EMDR improve. **Conclusions:** The majority of patients treated with psychotherapy for PTSD in randomized trials recover or improve. Exclusion criteria and polysymptomatic presentations render generalizability to the population of PTSD patients indeterminate. The majority of patients post-treatment continue to have substantial residue symptoms and follow up data beyond brief intervals are absent.


Smoking rates in the general public continue to fall. In contrast, the smoking rate of individuals with psychiatric disorders has continued despite new information and changing social values. This review article examines the epidemiological studies that have revealed a bireational causal relationship between tobacco dependence and post-traumatic stress disorder (PTSD). Rasmusson et al. also discuss possible neurobiological mediators of the relationship between PTSD and tobacco dependence and provide an overview of effective scientific methods for assessing and managing ‘smoking status’ as an experimental variable in clinical research studies of PTSD and other mental health disorders. Neurobiological subjects discussed include the HPA-axis, cortisol, DHEA, amygdala, hippocampus, pre-frontal cortex, and the ventral tegmental area (VTA).

The main purpose of this review by Yehuda is to discuss the inconsistent findings in the neuroendocrinology of PTSD literature and their therapeutic implications. Yehuda summarizes important neuroendocrine observations in cortisol and provides recommendations regarding how to interpret the discrepancies in the literature. This article examines how methodological details contribute to the variability in the cortisol literature. Yehuda also discusses investigations into the use of metyrapone (a drug that prevents adrenal steroidogenesis thus removing negative feedback inhibition of cortisol on the pituitary and hypothalamus).


CBT is one of the most popular treatments for posttraumatic stress disorder, however approximately half of patients do not respond to CBT. Bryant et al. set out to investigate the capacity for neural responses during fear processing to predict treatment response in PTSD. They hypothesized that poor response to CBT would be associated with greater activation in the amygdala and reduced mPFC recruitment during fear processing before treatment. Bryant et al. found that poor improvement after treatment was associated with greater bilateral amygdala and ventral anterior cingulate activation in response to masked fearful faces. They concluded that excessive fear responses in response to fear eliciting stimuli may be a key factor in limiting response to CBT for PTSD. Furthermore Bryant et al. concluded that this excessive amygdala
response to fear may reflect difficulty in managing anxiety reactions elicited during CBT, and this factor may limit optimal response to therapy.


The authors of this article carried out a systematic meta-analytic review of 38 randomized controlled trials. Study selection criteria were stated. The following criteria were used (this list is not exhaustive, please see article for full list): studies were only included if PTSD symptoms were the main target of treatment and used a randomized controlled design. All participants had to be adults and have PTSD symptoms for at least 3 months following a traumatic event. Type of traumatic event was not restricted. **Conclusions:** Trauma focused CBT (TFCBT), EMDR, stress management and group CBT were all shown to improve symptoms more than waiting list or usual care. Efficacy of other therapies examined was inconclusive. There was no evidence of a difference in efficacy between TFCBT and EMDR but there was evidence that both TFCBT and EMDR were superior to stress management and other therapies, and that stress management was superior to other therapies.


Alterations in the hypothalamic-pituitary-adrenal (HPA) axis has been linked to post traumatic stress disorder (PTSD). Specifically, 24-hour urinary cortisol values have been found to be lower in subjects with PTSD compared to help individuals with or without exposure to trauma. Other studies have found no differences in cortisol levels were even higher levels of cortisol in PTSD.
Olff et al. set out to examine the effects of psychotherapy in 21 PTSD patients, with and without coexisting depression, on the levels of six stress related hormones, including cortisol. They found that after a brief eclectic psychotherapy (BEP) significant changes occurred in levels of cortisol and DHEA. Those who responded to therapy showed increased levels of these stress hormones after therapy, while nonresponders showed decreases after therapy. Differences were only found after controlling for depressive symptoms. Olff et al. concluded that effective psychotherapy for PTSD may alter dysregulations in the HPA axis, but that comorbid depressive symptoms should be taken into account.


This article is a good review of the long-term effects of childhood abuse on brain and neurobiology, as well as the functional plasticity of the brain in the aftermath of trauma. The authors point out that stress is associated with the loss of branching of neurons in the hippocampus and an Inhibition of hippocampal neurogenesis. Bremner et al. discuss how stress affects memory and hippocampus, the HPA axis, and the amygdala and the MPFC. The effects of treatments, such as antidepressant medications and changes in environment, on the brain in PTSD are discussed as well as the role glutamate plays in symptoms of dissociation.

Bryant et al. (2008) set out to investigate the extent to which treatment response in posttraumatic stress disorder (PTSD) is predicted by rostral anterior cingulate cortex (rACC) volume. Using structural magnetic resonance imaging, Bryant et al. scanned subjects with PTSD ($n = 13$), traumatized control subjects ($n = 13$), and non-traumatized control subjects ($n = 13$). Subjects with PTSD then underwent eight sessions of cognitive therapy and were reassessed for PTSD. Results indicated that treatment responders had larger rACC volume than non-responders. In addition, symptom reduction was also linked to larger rACC volume. Bryant et al. explain that these findings are consistent with evidence for the neural basis of extinction learning. They concluded that PTSD patients with larger rACC volume may be better able to regulate fear during cognitive behavioral therapy and therefore will achieve greater treatment gains.


The aim of this article was to conduct a systematic review of the efficacy of CBT in comparison with studies that used other psychotherapies such as support, relaxation, counseling, and psychoeducation. Studies published between 1980 and 2005 were included. Main outcome variables were remission, clinical improvement, dropout rates, and changes in symptoms. 23 clinical trials were included in the review. **Results:** CBT had better remission rates than EMDR or supportive therapies. CBT was comparable to exposure therapy and cognitive therapy in terms of efficacy and compliance. **Main conclusions:** Specific therapies, such as CBT, exposure therapy, and cognitive therapy are equally effective, and more effective than supportive techniques in the treatment of PTSD. The authors also concluded that possible interactions between modality of treatment and specific populations are a matter for further high quality
methodological trials. In addition, future studies should be conducted with better methodology, with standardized treatment programs, reliable and valid outcome measures, blind independent evaluators, and intention-to-treat analyses.


The authors of this article set out to conduct a review of articles published up until the end of 2008 that used randomized controlled trials to investigate the empirically supported efficacy of psychological treatments for posttraumatic stress disorder (PTSD) and Acute Stress Disorder (ASD). The authors used an inclusion criteria set out by Chambless and Hollon in order to draw conclusions about efficacy, irrespective of trauma type and with regard to particular populations. The authors conclude that trauma-focused cognitive behavioural therapy (CBT) and eye movement desensitization and reprocessing (EMDR) are the psychological treatments of choice for PTSD but that further research of these and other therapies with different populations is needed.


The purpose of this study was to determine whether differences in cortisol or its metabolites predict or correlate with response to therapy for PTSD symptoms. Cortisol and its metabolites were measured from urine samples at pre-treatment, at the conclusion of psychotherapy, and at three month follow-up. Participants in the study were 28 survivors of the World Trade Center
attacks on September 11, 2001. Repeated measures analysis across the three time points cortisol metabolites significantly differed between responders and non-responders. Also of interest, a significant group x time interaction indicated that urinary cortisol levels decline over time in the non-responder group, such that by follow-up, lowered cortisol significantly distinguished non-responders from responders. Yehuda et al. conclude in this study that lower cortisol metabolite activity is associated with avoidance severity and predicts non-responsiveness to psychological treatment for PTSD symptomatology. Diminished cortisol metabolite activity may demonstrate a primary vulnerability and may result in the suppression of the HPA-axis responsiveness.

Ehlers, A., Bisson, J., Clark, D. M., Creamer, M., Pilling, S., Richards, D. et al. (2010). Do all psychological treatments really work the same in posttraumatic stress disorder?

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Ehlers at al. (2010) examine a recently published meta-analysis by Benish, Imel, and Wampold that concluded that all *bona fide* treatments are equally effective in posttraumatic stress disorder (PTSD). The authors contrast these findings with other studies that have concluded that trauma-focused psychological treatments such as trauma focused cognitive behavioural therapy (TFCBT) and eye movement desensitization and reprocessing (EMDR) are more effective than those treatments that do not focus on patient’s trauma memories or meanings. The authors make the following recommendations: 1) Selection procedures that introduce bias should not be used in future meta-analyses, 2) Studies included in meta-analyses should be controlled to demonstrate that the treatments for PTSD are more effective than natural recovery alone, 3) future research should examine active mechanisms of therapeutic change, 4) the reporting of exclusions in meta-analyses should be transparent, and 5) *bona fide* treatments should be defined on empirical and theoretical grounds rather than by judgments of the investigators intent.

Cross sectional epidemiological studies have consistently found high prevalence rates of PTSD among substance using subjects. Longitudinal studies support the “self medication hypothesis” and that individuals who develop PTSD are at risk of developing a substance use disorder. This study examined the temporal course of improvement in PTSD symptoms and substance use disorder among women in outpatient substance abuse treatment. Results of this study also demonstrated that PTSD changes influenced substance use outcomes with minimal evidence that substance use reduction improved PTSD symptoms. The results of this study offer support for the self-medication model and empirical support for PTSD-focused and integrated interventions efficacy in improving substance use outcomes in patients with severe symptoms.