Title

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Journal

Current Psychiatry Reports, 17(4)

ISSN

1523-3812

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Publication Date

2015-04-01

DOI

10.1007/s11920-015-0560-6

Peer reviewed

HHS Public Access

Author manuscript

Curr Psychiatry Rep. Author manuscript; available in PMC 2016 April 01.

Published in final edited form as:

Curr Psychiatry Rep. 2015 April ; 17(4): 560. doi:10.1007/s11920-015-0560-6.

Psychological Mechanisms of Effective Cognitive–Behavioral Treatments for PTSD

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Abstract

Several psychotherapies have been established as effective treatments for posttraumatic stress disorder (PTSD) including prolonged exposure, cognitive processing therapy, and cognitive therapy for PTSD. Understanding the key mechanisms of these treatments, i.e., how these treatments lead to therapeutic benefits, will enable us to maximize the efficacy, effectiveness, and efficiency of these therapies. This article provides an overview of the theorized mechanisms for each of these treatments, reviews the recent empirical evidence on psychological mechanisms of these treatments, discusses the ongoing debates in the field, and provides recommendations for future research. Few studies to date have examined whether changes in purported treatment mechanisms predict subsequent changes in treatment outcomes. Future clinical trials examining treatments for PTSD should use study designs that enable researchers to establish the temporal precedence of change in treatment mechanisms prior to symptom reduction. Moreover, further research is needed that explores the links between specific treatment components, underlying change mechanisms, and treatment outcomes.

Keywords

Treatment mechanism; Posttraumatic stress disorder; Prolonged exposure therapy; Cognitive processing therapy; Cognitive therapy; Treatment component

Introduction

Several psychotherapies shown to be effective for posttraumatic stress disorder (PTSD) are recommended by practice guidelines as first-line treatments for this disorder [1–6]. These treatments include three trauma-focused cognitive–behavioral therapies[1]: prolonged exposure (PE) [7, 8], cognitive processing therapy (CPT) [9–11], and cognitive therapy for

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Compliance with Ethics Guidelines

Human and Animal Rights and Informed Consent

This article does not contain any studies with human or animal subjects performed by any of the authors.

Conflict of Interest

Alyson K. Zalta receives grant support from NIH (K23 MH103394).

PTSD (CT-PTSD) [12–15]. Two meta-analyses have demonstrated that these treatments result in large reductions in PTSD symptoms (g= 1.09–1.44) [16], and on average, 56–70 % of those who complete treatment no longer meet the diagnostic criteria for PTSD [17]. Moreover, effectiveness research indicates that these treatments not only work in research settings but also lead to large improvements in PTSD symptoms in clinical practice [18, 19].

Given the large body of evidence supporting the efficacy of these treatments, the pressing question at present is not whether, but *how* these treatments work. Determining the key mechanisms of effective treatments has the potential to improve therapeutic outcomes in several ways. Understanding core treatment mechanisms would enable us to refine existing protocols and distill treatments to their most essential elements, thereby enhancing the efficiency of treatment delivery. This would be particularly beneficial for effective treatments in which the purported mechanisms of change are not supported by the empirical evidence, as treatment procedures are likely to be optimized for eliciting change in the purported mechanism rather than the true mechanism. An understanding of treatment mechanisms would also aid in the dissemination of treatments by teaching providers what key elements need to be delivered and helping providers understand how to individualize treatments in a way that will maximize benefit.

To explore how treatments work, it is first important to understand the factors involved in promoting therapeutic improvement. Doss [20] draws a critical distinction between *treatment components* and *change mechanisms* as the primary elements of therapeutic improvement.[2] Treatment components refer to the aspects of therapy that occur during the treatment session or as a result of homework that creates improvements in the change mechanisms. Treatment components can be further divided into *therapy techniques* and *patient events*. Therapy techniques refer to the intervention characteristics that are the "active ingredients" of treatment, and patient events refer to the patient behaviors or experiences that result from the therapy techniques. For example, in exposure therapy, a therapy technique includes in vivo exposures in which the therapist encourages the patient to approach objectively safe, but feared situations in the real world. A patient event that could result from this therapy technique is a reduction in the patients' experience of fear to the approached stimulus over repeated trials. Change mechanisms represent changes in patient characteristics or skills that have generalized in the patient's life and are no longer a direct result of therapy activities. Returning to the example of exposure therapy, a change mechanism could include reductions in the patient's expected probability of harm in previously feared situations. Thus, according to Doss' framework, treatment components (i.e., therapy techniques and patient events) lead to improvements in change mechanisms, which lead to therapeutic outcomes.

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[1]Eye Movement Desensitization and Reprocessing has also been established as an effective treatment for PTSD and is recommended by practice guidelines. To date, no studies have examined the underlying mechanisms of EMDR; rather, research has focused heavily on identifying the importance of specific treatment techniques (i.e., whether bilateral stimulation is a necessary component of treatment). Given that there are no empirical studies on the underlying mechanisms of EMDR, this treatment is not discussed in the current review.

[2]Doss refers to *treatment components* as "change processes," *therapy techniques* as "therapy change processes," and *patient events* as "client change processes" in his article. The terms "process" and "mechanism" are often used interchangeably in the psychotherapy literature. Because Doss' original terms may be confusing for readers, I have modified these terms to more closely reflect the nature of these constructs.

The current review focuses on the psychological change mechanisms that lead to therapeutic improvements in PE, CPT, and CT-PTSD. I begin by outlining the purported psychological mechanisms that are described by the treatment developers, then describe the current empirical evidence highlighting the most recent research. I conclude the review with a discussion of the ongoing controversies in this literature and directions for future research. Although research examining treatment components and biological mechanisms of treatment are equally important, these topics are beyond the scope of this review.

Theorized Psychological Mechanisms of CBT Treatments for PTSD

PE therapy is founded in emotional processing theory (EPT) [21, 22]. According to EPT, a fear structure is a representation in memory that serves as a program for escaping danger; this structure includes representations of feared stimuli, responses, and the meaning of stimuli and responses. EPT proposes that in PTSD, the fear structure associated with the trauma memory is pathological in two critical ways [23]. First, the fear structure contains a large number of stimulus elements that become erroneously associated with the meaning of danger. Second, the person's reactions during and after the trauma (including PTSD symptoms) become erroneously associated with the meaning of self-incompetence. This pathological fear structure is maintained through a vicious cycle of avoidance and inaccurate perceptions of oneself and the world. Cognitive and behavioral avoidance of trauma reminders prevents the individual from learning disconfirming information that the world is in fact safe and that they, as individuals, are competent. In turn, erroneous perceptions that "the world is completely dangerous" and "I am completely incompetent" further reinforce avoidance. The goal of PE is to modify the pathological fear structure and associated erroneous perceptions by activating the trauma memory and presenting disconfirming information; this process is termed "emotional processing." Thus, the modification of erroneous perceptions is viewed as a key psychological mechanism by which symptom reduction occurs in PE. PE aims to achieve emotional processing via the use of in vivo and imaginal exposures that promote systematic confrontation with trauma reminders. Table 1 outlines the purported therapy techniques, patient events, and psychological change mechanisms of PE.

CT-PTSD is grounded in Ehlers and Clark's cognitive model of PTSD [12]. According to this theory, PTSD occurs when individuals "process the trauma in a way that leads to a sense of current, serious threat" (p. 319). This sense of threat is maintained due to: (1) excessively negative appraisals of the trauma and trauma sequelae, (2) disturbances in autobiographical memory that provoke re-experiencing symptoms (i.e., poor elaboration and contextualization, strong associative memory, and strong perceptual priming), and (3) cognitive and behavioral strategies that prevent change in the appraisals and trauma memory. Threatening appraisals of the traumatic event typically involve an exaggerated probability of future catastrophic events and a sense that the individual cannot trust themselves to respond well to future threat. With respect to the consequences of the event, PTSD symptoms are often seen as a threat to physical and mental well-being, others' reactions are often viewed as being rejecting or blaming, and changes in other life domains are typically interpreted as a sign of permanent damage due to the event. These appraisals bias individuals' recall of traumatic events such that they selectively retrieve information

that is consistent with their appraisals and fail to retrieve information that is inconsistent with their appraisals. As patients attempt to manage this sense of threat, they often engage in maladaptive cognitive and behavioral strategies that ultimately reinforce negative appraisals and disturbances in memory including thought suppression, safety behaviors, avoidance of trauma reminders, and rumination. Thus, in CT-PTSD, the key psychological change mechanisms include the following: (1) modification of problematic appraisals that maintain a sense of current threat, (2) elaboration and integration of the trauma memory into autobiographical memory, and (3) reduction of the use of dysfunctional behavioral and cognitive strategies. These mechanisms are targeted using "reclaiming your life" behavioral activation assignments, reconstructing the trauma account through writing or imaginal reliving, cognitive restructuring, behavioral experiments, pairing corrective information with the trauma memory, and pairing trauma triggers with incompatible thoughts/behaviors. Table 1 outlines the purported therapy techniques, patient events, and psychological change mechanisms of CT-PTSD.

CPT, founded in social cognitive theory (SCT), focuses on individuals' cognitions with respect to the trauma and how these cognitions affect individuals' emotions and behavior. According to SCT, people reconcile new information (i.e., information from the trauma) and prior schemata by doing one of three things: altering the new information to match prior beliefs (assimilation), altering prior beliefs enough to incorporate the new information (accommodation), or altering one's prior beliefs completely to match the new information (over-accommodation). Both assimilation and over-accommodation lead to erroneous beliefs about the trauma, oneself, others, and the world. For example, if an individual previously believed that they could keep others safe (schema), then lost a friend in combat (new information), they might come to believe "I should have saved him; it's my fault he's dead," consistent with their prior beliefs (assimilation). Alternatively, if a person previously believed that others could be trusted (schema), then experienced an assault (new information), they might come to believe "No one can be trusted," completely consistent with the new information (over-accommodation). CPT aims to reduce PTSD symptoms by identifying and directly modifying maladaptive cognitions through the use of cognitive restructuring techniques. The goal for therapists is to help patients achieve more accurate and balanced accommodated beliefs. Thus, a reduction of maladaptive assimilated and over-accommodated beliefs and an increase in adaptive accommodated beliefs are viewed as central psychological mechanisms of CPT. Table 1 outlines the purported therapy techniques, patient events, and psychological change mechanisms of PE.

Current Evidence

When examining treatment mechanisms empirically, two critical issues arise [20]. First, investigators must operationalize how change mechanisms will be measured. For example, all three therapies emphasize the role of perceptual change as a mechanism of treatment (i.e., reduction of erroneous perceptions in PE, maladaptive appraisals in CT-PTSD, and stuck points in CPT), but the types of perceptions differ subtly across these treatments. For example, in PE, erroneous perceptions include not only those that are within the individuals' awareness but also perceptions that are outside of the individuals' awareness. The developers of PE have operationalized erroneous perceptions as negative cognitions of the

self, negative cognitions of the world, and self-blame as measured by the Posttraumatic Cognitions Inventory (PTCI) [24]. The developers of CT-PTSD have also used the PTCI to operationalize trauma-related appraisals [25••, 26]. In contrast, the developers of CPT have designed a coding scheme of patients' impact statements to identify assimilated, accommodated, and over-accommodated statements [27] as well as a measure of maladaptive beliefs related to threat of harm, self-worth and judgment, and reliability and trustworthiness of others termed the Posttraumatic Maladaptive Beliefs Scale [28]. These operationalizations are important as the properties of these metrics may impact research findings.

Second, studies must establish that change in the proposed mechanism precedes and predicts subsequent symptom change. Without establishing change and temporal precedence, a proposed mechanism may merely be a prognostic indicator of treatment success or a correlate or consequence of symptom change. To date, only three studies have examined the temporal relationship between changes in purported treatment mechanisms and changes in PTSD symptoms during PE, CT-PTSD, and CPT.

Zalta and colleagues [29••] examined changes in PTSD-related cognitions as a mechanism of PE in 64 female sexual or non-sexual assault survivors. Participants received a typical course of PE delivered over 10 sessions. PTSD-related cognitions, assessed with the PTCI [24], and PTSD symptoms were assessed weekly during treatment, which allowed for an examination of time-lagged associations between cognition change and symptom change. Results showed that session-to-session reductions in PTSD-related cognitions significantly predicted subsequent reductions in PTSD symptoms. In contrast, the reverse effect of PTSD symptom reduction on reductions in PTSD-related cognitions was much smaller and did not reach statistical significance. These findings indicate that change in PTSD-related cognitions is an important mechanism of PE, consistent with emotional processing theory.

Kleim and colleagues [25••] examined cognition change as a mechanism of CT-PTSD among 268 patients who were drawn from an effectiveness study of referrals to a UK National Health Service outpatient clinic. Participants received an average of 11.6 sessions of CT-PTSD over an average period of 108 days. PTSD-related cognitions, assessed with a brief version of the PTCI, and PTSD symptoms were assessed at each session. Results showed that weekly reductions in PTSD-related cognitions significantly predicted subsequent reductions in PTSD symptoms. Moreover, reductions in PTSD symptom severity did not predict subsequent changes in PTSD-related cognitions. These findings are consistent with the cognitive model of PTSD and indicate that cognition change is an important mechanism of CT-PTSD.

Gilman and colleagues [30] examined changes in hope as a mechanism of CPT treatment among 164 veterans in a Veteran's Administration residential treatment program. The patients received 12 group sessions of CPT and 12 to 13 individual sessions of CPT in a 7-week period. Hope and PTSD symptoms were assessed at pre-treatment, midway through treatment (week 4) and at post-treatment (week 7). Hope was assessed using the Hope Scale [31], which assesses two facets of hope: pathways (i.e., planning to accomplish goals) and agency (i.e., goal-directed energy). Results showed that levels of hope at mid-treatment

predicted changes in PTSD symptoms from mid- to posttreatment; however, hope did not change from pre-treatment to mid-treatment, as expected. Thus, it is unclear from this study whether hope is a mechanism or a prognostic indictor of CPT outcomes.

To date, only one study has compared treatment mechanisms across multiple effective treatments. Gallagher and Resick [32•] examined changes in hopelessness as a mechanism of treatment for both CPT and PE. One hundred seventy-one rape victims were randomized to CPT, PE, or a minimal attention condition. Patients in the active treatments were seen twice weekly for a period of 6 weeks resulting in a total of 13 h of treatment. PTSD symptoms and hopelessness were assessed at pre- and posttreatment. Results showed that both treatments led to significant reductions in hopelessness from pre- to posttreatment, and reductions in hopelessness were significantly associated with reductions in PTSD symptoms. CPT led to greater changes in hopelessness from pre- to posttreatment than PE, and changes in hopelessness mediated the relationship between treatment type and PTSD symptom change. This study was unable to establish the temporal precedence of change in hopelessness, limiting the extent to which conclusions can be drawn. However, these findings are suggestive that changes in hopelessness may serve as a mechanism of both PE and CPT, but that CPT leads to larger changes in hopelessness than PE. Given that PE and CPT lead to equivalent treatment outcomes, this finding may suggest that PE is more effective at targeting other important beliefs that maintain PTSD.

Conclusions from the Current Evidence

Remarkably, few studies have examined the temporal relationship between changes in purported mechanisms and outcomes for effective PTSD treatments, indicating that this research is still in its infancy. The current evidence suggests that the reduction of PTSD-related cognitions is a central mechanism of PE and CT-PTSD. Although two recent studies have examined cognition change in the context of CPT, the design of these studies did not permit firm conclusions. Thus, further research is needed to establish the extent to which cognition change is also a core mechanism of CPT.

Notably, the studies on PE and CT-PTSD collected global measures of PTSD-related cognitions whereas studies on CPT focused on the role of changes in specific cognitions (i.e., hope and hopelessness). The study by Gallagher and Resick [32•] raises the question as to whether these three treatments differentially target some beliefs more effectively than others. For example, PE may be more likely to change the belief that a person cannot handle thinking about the trauma whereas CPT may be more likely to change beliefs related to self-blame. If these treatments do target different beliefs more effectively, it is possible that treatment should be selected to match an individuals' profile of beliefs to maximize treatment benefit. Further research is needed to explore how these treatments lead to cognition change and whether certain treatment procedures have a differential impact on certain types of cognitions.

Ongoing Controversies

A question that has been a source of confusion and exploration for many years is the role of habituation in PE. Foa and Kozak [22] argue that habituation is an indicator that emotional

processing is taking place (i.e., pathological elements of the fear structure have been modified). If patients are no longer fearful of a given stimulus because the fear structure has been modified, we would expect self-reported fear in the presence of the stimulus to decrease (i.e., habituation). Additionally, the PE treatment manual highlights how the experience of habituation can provide corrective information for specific erroneous perceptions such as the fact that anxiety does not last forever and that the person can handle a feared situation, thereby facilitating emotional processing. However, habituation is not proposed to be a mechanism of PE, as some have suggested.

If habituation is an indicator of emotional processing and not a mechanism of treatment, we would expect that habituation is related to treatment outcome but not necessary for treatment benefit to occur. Recent evidence by Bluett and colleagues [33•] is consistent with this hypothesis. Their study showed that patients who did not experience a reliable change in distress during imaginal exposure from pre- to posttreatment had similar rates of PTSD remission as those who did experience a reliable change in distress. However, individuals who experienced a reliable change in distress showed larger symptom reduction than those who did not experience a reliable change in distress. These findings suggest that future research should focus on elaborating how habituation promotes emotional processing so that therapists can capitalize on the presence of habituation when it occurs during treatment.

Another long-standing debate in the psychotherapy treatment literature is whether treatment effects are due to specific or non-specific treatment components and change mechanisms. Although this dispute has largely been focused on treatment components, it also has important implications for treatment mechanisms research. *Specific components* refer to the therapy techniques that are based on the treatment theory, whereas *non-specific components* refer to the treatment factors that are common virtually to all psychotherapies. For example, CPT proposes that symptom reduction occurs because cognitive restructuring leads to cognition change; however, it is also possible that symptom reduction occurs because a positive therapeutic alliance leads to increased self-worth. Investigators have implied that if psychotherapies are equally effective, this means that treatment effects must be due to non-specific treatment components [34]. Additionally, investigators have proposed that if two therapies share the same mechanism (specific or non-specific), then the distinctions between the treatment components that contribute to changes in the shared mechanism are meaningless and “merely repackaging” (p. 751) [33•].

There are several problems with these arguments. When two treatments achieve an equivalent outcome, it does not necessarily imply that the same treatment components and mechanisms lead to the outcomes [35]. Moreover, even if two treatments do share the same mechanisms, this does not necessarily mean that differences between the treatment components are inconsequential. For example, it is possible that important moderators may affect whether one therapy technique is superior to another in promoting improvement in the treatment mechanism within certain populations or contexts. This is not to deny that non-specific treatment components likely have a role in treatment outcomes. In fact, it is quite likely that non-specific treatment factors and specific treatment components interact to produce change in treatment mechanisms (e.g., cognitive restructuring may be more effective in producing cognitive changes when the therapeutic alliance is high compared to

when the therapeutic alliance is low). Thus, investigators should aim to assess both specific and non-specific treatment components and mechanisms to elaborate how these factors lead to therapeutic benefit.

Future Directions

As the field continues to develop a better understanding of the underlying mechanisms of effective PTSD treatments, it will be important for investigators to explore the links between treatment components and change mechanisms. To date, research on treatment components and treatment mechanisms has largely been conducted separately with different research camps and study methodologies [20]. For example, there is a substantial research literature examining the extent to which completion of homework assignments improves treatment outcome in cognitive–behavioral therapy [36, 37]. To my knowledge, however, there is no research examining the mechanisms that mediate the relationship between homework completion and reduction in PTSD symptoms. Understanding how specific therapy techniques and patient events lead to improvements in established treatment mechanisms will enable clinicians to provide patients with a clear rationale for treatment procedures, focus on procedures that lead to maximal benefit, and individualize treatment delivery without compromising outcome.

If we hope to truly maximize the efficacy, effectiveness, and efficiency of PTSD treatment, we cannot focus solely on how treatment change comes about for the average individual. It is well established that certain patient characteristics (i.e., moderator variables) affect the extent to which individuals benefit from treatment. These moderators may affect the extent to which treatment components in different psychotherapies impact change mechanisms. For example, it is possible that for certain patients, exposures may be more effective than cognitive restructuring at promoting cognition change or vice versa. Within a given treatment, it is also possible that moderating factors may impact which treatment components promote the greatest change in the treatment mechanisms. For example, it is possible that for certain patients, in vivo exposure may be more effective at promoting emotional processing than imaginal exposure or vice versa. Moderator variables may also impact the extent to which change mechanisms impact treatment outcomes. For example, it is possible that for certain patients, a reduction in self-blame beliefs will lead to greater symptom improvement than a reduction in beliefs about distrusting others and vice versa. Understanding these important moderators of treatment components and mechanisms (i.e., moderated mediation) would aid in treatment selection and delivery. Thus, research on the mediators and moderators of treatment must be integrated to maximize treatment benefit.

Conclusions

The research literature on psychological mechanisms of effective treatments for PTSD is still nascent. Two recent studies have conducted rigorous examinations of treatment mechanisms in PE and CT-PTSD [25••, 29••], indicating that the field is moving in the right direction. However, two factors appear to serve as the primary hindrances towards the advancement of this field. First, this review highlights the importance of study design when conducting clinical trials to facilitate an exploration of how these treatments work to reduce

symptoms of PTSD. The National Institute of Mental Health has taken an official stance on this issue by promoting an experimental therapeutic paradigm for clinical trials in which an intervention must demonstrate an impact on the proposed mechanism of action, not just therapeutic benefit [38, 39]. Thus, it will be imperative for researchers to consider and assess treatment mechanisms when designing clinical trials in the future. The second key issue is that researchers have often overlooked the distinction between treatment components and change mechanisms, leading to imprecise conclusions and confusion within the field [20]. Researchers should be mindful of the various factors that lead to therapeutic change, be precise in their terminology when describing these factors, and work towards the integration of treatment component and treatment mechanisms research to best understand how these effective treatments lead to therapeutic benefits.

Acknowledgments

The author would like to thank Nicole Heath and Rebecca Blais for the feedback on a draft of this manuscript.

References

Papers of particular interest, published recently, have been highlighted as:

• Of importance

•• Of major importance

1. American Psychiatric Association Work Group on ASD and PTSD. Practice guideline for the treatment of patients with acute stress disorder and posttraumatic stress disorder. Washington, DC: Author; 2004.
2. Australian Centre for Posttraumatic Mental Health. Australian guidelines for the treatment of adults with acute stress disorder and posttraumatic stress disorder: Information for people with ASD and PTSD, their families and carers. 2007.
3. National Institute for Health and Clinical Excellence. [Accessed 19 Dec 2014] Posttraumatic stress disorder (PTSD): The management of PTSD in adults and children in primary and secondary care (NICE Clinical Guideline 26). 2005. Retrieved from http://guidance.nice.org.uk/CG26
4. Stein DJ, Cloitre M, Nemeroff CB, Nutt DJ, Seedat S, Shalev AY, et al. Cape Town consensus on posttraumatic stress disorder. CNS Spectr. 2009; 14(1 Suppl 1):52–8. [PubMed: 19169194]
5. Department of Veterans Affairs and Department of Defense. Management of post-traumatic stress. Washington, DC: Department of Veterans Affairs; 2010. Retrieved from http://www.healthquality.va.gov/Post_Traumatic_Stress_Disorder_PTSD.asp
6. Foa, EB.; Keane, TM.; Friedman, MJ.; Cohen, J. Effective treatments for PTSD: practice guidelines from the International Society for Traumatic Stress Studies. 2. New York: Guilford Press; 2009.
7. Foa, E.; Rothbaum, BO. Treating the trauma of rape: cognitive-behavior therapy for PTSD. New York: Guilford Press; 1998.
8. Foa, E.; Hembree, E.; Rothbaum, B. Prolonged exposure therapy for PTSD: emotional processing of traumatic experiences, therapist guide. New York: Oxford University Press; 2007.
9. Resick PA, Schnicke MK. Cognitive processing therapy for sexual assault victims. J Consult Clin Psychol. 1992; 60(5):748–56. [PubMed: 1401390]
10. Resick, PA.; Schnicke, MK. Cognitive processing therapy for rape victims: a treatment manual. Newbury Park, CA: Sage; 1993.
11. Resick PA, Nishith P, Weaver TL, Astin MC, Feuer CA. A comparison of cognitive-processing therapy with prolonged exposure and a waiting condition for the treatment of chronic posttraumatic stress disorder in female rape victims. J Consult Clin Psychol. 2002; 70(4):867–79. [PubMed: 12182270]

12. Ehlers A, Clark DM. A cognitive model of posttraumatic stress disorder. Behav Res Ther. 2000; 38(4):319–45. [PubMed: 10761279]
13. Ehlers A, Clark DM, Hackmann A, Grey N, Liness S, Wild J, et al. Intensive cognitive therapy for PTSD: a feasibility study. Behav Cogn Psychother. 2010; 38(4):383–98. [PubMed: 20573292]
14. Ehlers A, Clark DM, Hackmann A, McManus F, Fennell M, Herbert C, et al. A randomized controlled trial of cognitive therapy, self-help booklet, and repeated early assessment as early interventions for posttraumatic stress disorder. Arch Gen Psychiatry. 2003; 60(10):1024–32. [PubMed: 14557148]
15. Ehlers A, Clark DM, Hackmann A, McManus F, Fennell M. Cognitive therapy for post-traumatic stress disorder: development and evaluation. Behav Res Ther. 2005; 43(4):413–31. [PubMed: 15701354]
16. Watts BV, Schnurr PP, Mayo L, Young-Xu Y, Weeks WB, Friedman MJ. Meta-analysis of the efficacy of treatments for post-traumatic stress disorder. J Clin Psychiatry. 2013; 74(6):e541–50. [PubMed: 23842024]
17. Bradley R, Greene J, Russ E, Dutra L, Westen D. A multidimensional meta-analysis of psychotherapy for PTSD. Am J Psychiatry. 2005; 162(2):214–27. [PubMed: 15677582]
18. Tuerk PW, Yoder M, Grubaugh A, Myrick H, Hamner M, Acierno R. Prolonged exposure therapy for combat-related posttraumatic stress disorder: an examination of treatment effectiveness for veterans of the wars in Afghanistan and Iraq. J Anxiety Disord. 2011; 25(3):397–403. [PubMed: 21131170]
19. Schulz PM, Resick PA, Huber LC, Griffin MG. The effectiveness of cognitive processing therapy for PTSD with refugees in a community setting. Cogn Behav Pract. 2006; 13(4):322–31.
20. Doss BD. Changing the way we study change in psychotherapy. Clin Psychol (New York). 2004; 11(4):368–86.
21. Foa, EB.; Kozak, MJ. Treatment of anxiety disorders: implications for psychopathology. In: Tuma, EH.; Maser, JD., editors. Anxiety and the anxiety disorders. Hillsdale: Laurance Erlbaum Associates; 1985. p. 421-52.
22. Foa EB, Kozak MJ. Emotional processing of fear: exposure to corrective information. Psychol Bull. 1986; 99(1):20–35. [PubMed: 2871574]
23. Zalta, AK.; Foa, EB. Exposure therapy: promoting emotional processing of pathological anxiety. In: O'Donohue, W.; Fisher, JE., editors. Cognitive behavior therapy: core principles for practice. Hoboken: Wiley; 2012. p. 75-104.
24. Foa EB, Ehlers A, Clark DM, Tolin DG, Orsillo SM. The posttraumatic cognitions inventory (PTCI): development and validation. Psychol Assess. 1999; 11(3):303–14.
25••. Kleim B, Grey N, Wild J, Nussbeck FW, Stott R, Hackmann A, et al. Cognitive change predicts symptom reduction with cognitive therapy for posttraumatic stress disorder. J Consult Clin Psychol. 2013; 81(3):383–93. In this effectiveness study of cognitive therapy for PTSD, session-to-session reductions in PTSD predicted subsequent reductions in PTSD symptoms. [PubMed: 23276122]
26. Ehlers, A. Trauma-focused cognitive behavior therapy for posttraumatic stress disorder and acute stress disorder. In: Simos, G.; Hofmann, SG., editors. CBT for anxiety disorders: a practitioner book. West Sussex, UK: Wiley-Blackwell; 2013. p. 161-89.
27. Sobel AA, Resick PA, Rabalais AE. The effect of cognitive processing therapy on cognitions: impact statement coding. J Trauma. 2009; 22(3):205–11.
28. Vogt DS, Shipherd JC, Resick PA. Posttraumatic maladaptive beliefs scale: evolution of the personal beliefs and reactions scale. Assessment. 2012; 19(3):308–17. [PubMed: 20660470]
29••. Zalta A, Gillihan S, Fisher A, Mintz J, McLean C, Yehuda R, et al. Change in negative cognitions associated with PTSD predicts symptom reduction in prolonged exposure. J Consult Clin Psychol. 2014; 82(1):171–5. In this study of prolonged exposure, session-to- session reductions in PTSD related cognitions predicted subsequent reductions in PTSD symptoms. [PubMed: 24188512]
30. Gilman R, Schumm JA, Chard KM. Hope as a change mechanism in the treatment of posttraumatic stress disorder. Psychol Trauma. 2012; 4(3):270–7.

31. Snyder CR, Harris C, Anderson JR, Holleran S, Irving LM, Gibb J, et al. The will and the ways: development and validation of an individual differences measure of hope. J Pers Soc Psychol. 1991; 60(4):570–85. [PubMed: 2037968]

32•. Gallagher MW, Resick PA. Mechanisms of change in cognitive processing therapy and prolonged exposure therapy for PTSD: preliminary evidence for the differential effects of hopelessness and habituation. Cognitive Ther Res. 2012; 36(6):750–5. This study showed that cognitive processing therapy led to greater reductions in hopelessness than prolonged exposure and reductions in hopelessness mediated the relationship between treatment type and treatment outcome.

33•. Bluett EJ, Zoellner LA, Feeny NC. Does change in distress matter? Mechanisms of change in prolonged exposure for PTSD. J Behav Ther Exp Psy. 2014; 45(1):97–104. This study showed that patients receiving prolonged exposure who did not demonstrate reliable changes in distress during imaginal exposure from pre to post treatment still showed marked improvement in PTSD symptom severity.

34. Luborsky L, Singer B, Luborsky L. Comparative studies of psychotherapies: is it true that "everyone has one and all must have prizes"? Arch Gen Psychiatry. 1975; 32(8):995–1008. [PubMed: 239666]

35. DeRubeis RJ, Brotman MA, Gibbons CJ. A conceptual and methodological analysis of the nonspecifics argument. Clin Psychol-Sci Pr. 2005; 12(2):174–83.

36. Mausbach BT, Moore R, Roesch S, Cardenas V, Patterson TL. The relationship between homework compliance and therapy outcomes: an updated meta-analysis. Cognit Ther Res. 2010; 34(5):429–38.

37. Huppert JD, Ledley DR, Foa EB. The use of homework in behavior therapy for anxiety disorders. J Psychother Integr. 2006; 16(2):128–39.

38. Insel T. NIMH's new focus in clinical trials. National Institute of Mental Health; 2013. http://www.nimh.nih.gov/funding/grant-writing-and-application-process/concept-clearances/2013/nimhs-new-focus-in-clinical-trials.shtml [Accessed 19 Dec 2014]

39. Insel T. Director's blog: a new approach to clinical trials. National Institute of Mental Health; 2014. http://www.nimh.nih.gov/about/director/2014/a-new-approach-to-clinical-trials.shtml [Accessed 19 Dec 2014]

Table 1

Proposed treatment components and psychological change mechanisms of effective CBT treatments for PTSD

Treatment	Therapy techniques	Patient events	Psychological mechanisms
Prolonged exposure [8]	• Therapist teaches breathing retraining • Therapist helps the patient develop an exposure hierarchy and assigns in vivo exercises for homework • Therapist encourages the patient to engage in imaginal exposure in session • Therapist promotes engagement with the trauma memory during imaginal exposure using encouragement and prompting questions • Therapist helps patient process reactions to imaginal exposure	• Patient practices breathing retraining and experiences relaxation • Patient approaches feared situations, places, and people for homework and experiences a reduction in distress over repeated trials • Patient repeatedly recounts the trauma memory aloud with a description of his/her thoughts and feelings at the time and experiences a reduction in distress over repeated trials • Patient listens to tape of imaginal exposure daily for homework and experiences a reduction in distress over repeated trials • Patient experiences activation of the trauma memory during in vivo and imaginal exposures • Patient is confronted with disconfirming information during in vivo exposures, imaginal exposures, and processing of imaginal exposures	• Emotional processing: pathological elements of the fear structure and associated erroneous perceptions are modified
Cognitive therapy for PTSD [13, 15]	• Therapist helps patient identify appraisals by recalling the trauma • Therapist uses Socratic dialogue to gather updating information • Therapist uses behavioral experiments to gather updating information and test maladaptive behavioral/cognitive strategies • Therapist helps patient recall the trauma while reminding themselves of updating information • Therapist helps patient identify triggers • Therapist helps patient explore how current triggers differ from past trauma	• Patient reinstates pleasant activities/social contacts for homework and recognizes their life is not permanently damaged by trauma • Patient recalls the trauma and recognizes idiosyncratic appraisals • Patient engages in Socratic dialogue/behavioral experiments and gathers updating information • Patient completes behavioral experiments in which they do not use maladaptive cognitive/behavioral strategies and recognizes how these strategies lead to further problems • Patient develops a narrative account of the trauma using imaginal reliving, writing, and revisiting the site • Patient practices pairing narrative account with updating information • Patient identifies trauma triggers • Patient experiences/induces triggers and focuses on how the present is different from the past	• Patient reduces use of maladaptive cognitive/ behavioral strategies • Patient's appraisals of trauma and trauma sequelae become more accurate/ adaptive • The trauma memory is elaborated and integrated into autobiographical memory
Cognitive processing therapy [10]	• Therapist helps patient identify stuck points in their thinking	• Patient writes about why they think the trauma happened and how the trauma has affected their thinking in critical domains	• Patient's daily cognitions about the trauma, themselves, others, and the world become more

Treatment	Therapy techniques	Patient events	Psychological mechanisms
	• Therapist teaches patient how thoughts affect feelings • Therapist encourages the patient to write about the meaning of the trauma and asks patient to read in session • Therapist uses Socratic dialogue to challenge stuck points in session • Therapist teaches patient to challenge stuck points, identify maladaptive thinking patterns, and generate alternative thoughts	• Patient identifies stuck points in session and between sessions and is able to recognize maladaptive beliefs • Patient completes ABC sheets and understands the connection between thoughts and feelings • Patient talks with the therapist about stuck points in session and acknowledges evidence against their beliefs • Patient challenges stuck points for homework by asking themselves a series of questions and recognizes thinking errors • Patient practices labeling stuck points with patterns of problematic thinking and understands common patterns in their thinking • Patient practices generating alternative beliefs and experiences a reduction in the extent stuck points are endorsed	balanced, accurate, and adaptive as characterized by a greater number of accommodated beliefs and a reduced number of assimilated and over-accommodated beliefs

These treatment components and change mechanisms reflect those proposed by the treatment developers, not those established by empirical research. The components and mechanisms listed are not exhaustive; they are meant to highlight the components and mechanisms thought to be most central to each treatment