

Adult PTSD and Its Treatment With EMDR: A Review of Controversies, Evidence, and Theoretical Knowledge

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This article provides an overview of selective issues relating to adult posttraumatic stress disorder (PTSD) and its treatment with eye movement desensitization and reprocessing (EMDR). The article begins by providing a historical overview of PTSD, and debates about the etiology and definition of PTSD are discussed. The most predominant theories of PTSD are summarized by highlighting how they have evolved from traditional behavioral accounts based on the assumption that PTSD is an anxiety disorder to theories that now incorporate information-processing models. This article then examines the development of EMDR and the corresponding body of research that clearly demonstrates its efficacy for the treatment for adult PTSD. The underlying mechanisms of EMDR are discussed, with a focus on the importance of the eye movement component and how the therapeutic processes in EMDR differ from those of traditional exposure therapy. Finally, the adaptive information-processing (AIP) model that underlies EMDR is outlined, and evidence for the model is summarized. The article concludes by suggesting future research based on questions raised about PTSD and its treatment with EMDR when the AIP model is compared to other information-based theories of PTSD.

Keywords: EMDR; PTSD; review; theory; mechanisms of action

Brief Extract

The Role of Eye Movements in EMDR

Although the clinical efficacy of EMDR has been demonstrated, the role of the eye movements (EMs)

in EMDR continues to be controversial, with critics arguing that they are superfluous to the method (i.e., Nevid, Rathus, & Greene, 2008). Although findings regarding the role of EMs are inconsistent, it is important to note that much of the research in this area is filled with methodological problems, such as analogue studies with small, nonclinical samples and insufficient use of EMs. To date, no randomized controlled trial has been conducted to compare EMDR with EMs to EMDR without EMs on a large sample of adults with PTSD. Thus, ruling out the need for EMs in EMDR is premature. Furthermore, Perkins and Rouanzoin (2002) highlight that

EMDR has received empirical validation as a treatment for PTSD, and the tested procedure includes the eye movement (or alternative dual-attention) component. Therefore, the removal of these stimuli from the validated procedure requires prior component analyses adequate to rule them out as a significant treatment element. In the absence of such studies, their removal is without empirical justification. (p. 86)

Although the exact role of the EMs in EMDR remains unknown, numerous laboratory studies have examined the effects of EMs on memory and cognitive processes for participants not experiencing PTSD. Research suggests that EMs may contribute to the effectiveness of EMDR through a number of different processes, as they have been found to decrease the vividness and/or emotionality of autobiographical memories (Andrade, Kavanagh, & Baddeley, 1997; Barrowcliff, Gray, Freeman, & MacCulloch, 2004; Kavanagh, Freese, Andrade, & May, 2001; Maxfield, Melnyk, & Hayman, 2008; Sharpley, Montgomery, & Scalzo, 1996; van den Hout, Muris, Salemink, & Kindt, 2001), enhance the retrieval of episodic memories (Christman, Garvey, Propper, & Phaneuf, 2003), and increase cognitive flexibility (Kuiken, Bears, Miall, & Smith, 2001–2002) and may change inter-hemispheric coherence in frontal areas of the brain (Propper, Pierce, Geisler, Christman, & Bellorado, 2007). Research has also demonstrated that EMs produce psychophysiological dearousal when accessing distressing memories (i.e., Barrowcliff et al., 2004). Additional treatment studies that have demonstrated a dearousal effect measured physiological changes during EMDR and indicate that the EMs are associated with physiological responses that are characteristic of an orienting response (Sack, Lempa, Steinmetz, Lamprecht, & Hofmann, 2008) but may also resemble physiological characteristics of REM sleep (Elofsson, von Sche'ele, Theorell, & Söndergaard, 2008).

At present, more research is required to examine the precise causal role of the EMs in EMDR. For example, do EMs enhance the processing of memories, leading to physiological dearousal, or do the physiological effects of the EMs facilitate the processing of memories? For a more thorough review of the role of EMs in EMDR, see Propper and Christman (2008) and Gunter and Bodner (this issue).

The Effects of EMDR Are Different to Exposure

Although some reviewers have suggested that the main effect in EMDR is that akin to traditional exposure (i.e., Benish, Imel, & Wampold, 2008), there are three major differences between the therapeutic processes that distinguish EMDR from traditional exposure. According to a strict exposure definition, these differences should result in EMDR being *ineffective* for treating PTSD as the procedures should sensitize rather than desensitize its recipients (Perkins & Rouanzoin, 2002). First, EMDR is not based on habituation, as it uses short 20- to 50-second, interrupted exposures rather than continuous 20- to 100-minute exposures, traditionally recommended for prolonged exposure (Rogers & Silver, 2002). Second, EMDR is nondirective, allowing for free association. The client often moves quickly through scenes or skips scenes by spontaneously changing to other memories that arise. In EMDR, this is not seen as avoidance but is instead viewed as effective memory processing (Lee & Drummond, 2008; Lee, Taylor, & Drummond, 2006). Third, in EMDR, reliving the traumatic memory in the present tense is not a requirement of therapy. Taking a third-party perspective on the trauma is also not seen as avoidance, and, unlike traditional exposure, reliving is not associated with improvement in EMDR (Lee & Drummond, 2008). According to the assumptions of emotional processing theory (Foa & Rothbaum, 1998), which underlie exposure therapy for PTSD, the type of exposure that occurs in EMDR should result in minimal decreased fear if exposure is the proposed mechanism of change. Yet EMDR is effective in treating adult PTSD and associated symptoms.

Theories Regarding the Underlying Mechanisms of EMDR

Common factors across psychotherapies contribute to their individual efficacy. However, it does not follow that all improvement is due mainly to those factors. EMDR involves many therapeutic elements. Therefore, a number of agents of change may be involved beyond the effects of exposure and the EMs. Yet, like

any therapy, the exact mechanisms of change underlying EMDR are currently unknown, but a number of theories exist. EMDR is currently guided by the AIP model, which is consistent with Foa and Kozak's (1986) information-processing theory. There are, however, four other main hypotheses regarding the theoretical mechanisms of EMDR that have, in the current third wave of research, begun to accumulate a sound empirical base and offer support for the AIP model: orienting response activation, REM-like mechanisms, the theory of increased hemispheric communication, and working memory accounts.

AIP Model

The AIP model offers an explanation for the basis and recovery of trauma symptoms, it guides clinical case conceptualization, and directs treatment. The AIP model, which is consistent with other learning-based theories of PTSD, proposes that new experiences are processed by *assimilating* them with existing memory networks and that adaptive learning takes place (Shapiro, 1995, 2001). Shapiro (2001) states that adaptive learning occurs when information from new experiences are perceived and "the connections to appropriate associations are made and that the experience is used constructively by the individual and is integrated into a positive emotional and cognitive schema" (p. 30). According to the AIP model, pathology arises when memories of an experience are not adequately processed. Rather, the memory is dysfunctionally stored in its own neural network, which, like a fear network (Foa & Kozak, 1986), contains thoughts, images, emotions, and sensations associated with the event that, when triggered, influence perceptions, attitudes, and behavior in the present. Whether the memories are of an event that meets criterion A(1) for PTSD or are memories of "small t" traumas or whether the predominant emotions are criterion A(2) emotions or other emotions such as shame or guilt is irrelevant to the model. The main etiological factor of trauma symptoms is that the memories are unintegrated and dysfunctionally stored.

The AIP model suggests that it is the activation of the information-processing system that leads to the resolution of dysfunctionally stored traumatic memories. However, Shapiro (2001) proposes that information processing is facilitated primarily by three mechanisms in EMDR: (a) deconditioning that proceeds through a relaxation response, (b) neurological changes in the brain that activate and strengthen weak associations, and (c) factors that are involved with the client's dual focus of attention on both the

memory and a concurrent task, such as EMs. Evidence for these proposed mechanisms of action have come out of various research paradigms that have examined how EMDR may work.

Research Examining the AIP Model

Research into the activation of an orienting response (MacCulloch & Feldman, 1996) in EMDR provides support that a relaxation response occurs when the EMs begin that may facilitate treatment by reducing stress to a tolerable level so that processing of memories can occur (Barrowcliff et al., 2004; Elofsson et al., 2008; Sack et al., 2008). Research that has investigated physiological responses created by the EMs in EMDR has also noted that changes characteristic of a REM-like state occur (Elofsson et al., 2008). Stickgold (2002) has proposed a REM hypothesis of EMDR that states that the EMs in EMDR, through repeated orienting responses, may "push-start" memory processing in the brain by inducing a physiological and neurological state that is akin to REM sleep that aids in the transfer and integration of memories. Overall, the EMs in EMDR have an effect on physiology by creating either an orienting response or a REM-like state, but further research is required to clarify the effect and refine related theories.

Research into the theory of increased hemispheric communication provides empirical support for Shapiro's (2001) second hypothesized mechanism that information processing in the treatment of traumatic memories is facilitated by neurological changes in the brain that activate and strengthen weak associations. The theory of increased hemispheric communication proposed that horizontal EMs increase communication between both hemispheres of the brain, thus enhancing one's ability to remember the traumatic event while not becoming aroused (Christman et al., 2003). However, at present, mixed findings characterize the evidence for the increased hemispheric communication account of how EMDR works. For example, recent research by Propper et al. (2007) reported that engaging in bilateral EMs decreased rather than increased interhemispheric coherence. Also contrary to the account, Gunter and Bodner (2008) demonstrated that vertical EMs, which in theory do not increase hemispheric communication, were equally effective as horizontal EMs at reducing ratings vividness, emotionality, and completeness of unpleasant autobiographical memories.

Research has also begun to accumulate to support Shapiro's (2001) third hypothesis, that the client's dual focus of attention on both the trauma memory and a

concurrent task is a mechanism that facilitates information processing in EMDR. What is gaining empirical support are working memory models that can account for the discrepant findings within research that have examined the increased hemispheric communication account. For example, Gunter and Bodner (2008) explained the equivalent benefits for vertical and horizontal EMs by proposing that their finding supported a working memory account, as both tasks taxed the visual spatial sketch pad component of working memory to a similar degree. A working memory account of EMDR proposes that the dual-attention stimuli in EMDR, whether it be EMs or some other task such as tapping or tones, leads clients to attend to both the external stimulus and internally to the trauma-related memories (Maxfield et al., 2008). Baddeley's (1986) model of working memory suggests that each component of working memory has limited memory resource capacity, so when two tasks make demands on the attentional capacity of a component, performance on the primary task deteriorates. That is, in EMDR, when individuals engage in EMs while simultaneously focusing on a memory image, the quality of the image deteriorates, presumably because it gets pushed out of working memory and integrated into long-term memory, where the memory then becomes less vivid and less emotional. Space does not permit an extended discussion on the research that has examined working memory effects; for this and for more in-depth discussions of the orienting response, REM-like mechanisms, and the increased hemispheric communication account of EMDR, refer to Gunter and Bodner (this issue).

Consistent with other information-processing theories of PTSD, AIP theory assumes the existence of an information-processing system that, when working appropriately, incorporates new experiences into pre-existing memory networks, which are the basis of perception, attitudes, and behavior. At the heart of AIP and other information-processing models of PTSD, such as emotional processing theory (Foa & Rothbaum, 1998) and dual-representation theory (Brewin et al., 1996), is that recovery of PTSD is all about the elaboration or processing of memory. The AIP model is consistent with emotional processing theory, as it is assumed that the fear memory of the traumatic event needs to be activated and that corrective information must be provided that is incompatible with the fear structure. Associations are made with existing memory networks, resulting in learning, relief of emotional distress, and material becoming available for future use. All information-processing models assume that dysfunctional trauma reactions result when information relating to a traumatic event is not adequately

processed. There are, however, some distinct differences between AIP and current information-based theories of PTSD, and these differences have important implications for theory and treatment of PTSD.

AIP Contrasted With Other Psychological Models of PTSD

Unlike AIP, dual-representation theory (Brewin et al., 1996) assumes that the concept of a single memory system is inadequate to account for the full range of complex phenomena associated with PTSD. Thus, as previously mentioned, two memory systems are proposed to exist: conscious VAMs and unconscious SAMs, which are unintegrated and triggered by reminders of the trauma and, when triggered, are accompanied by emotional and/or physiological arousal experienced during the trauma. Although dual-representation theory is not linked to any specific treatment protocol, like AIP is linked to EMDR, it is proposed that treatment needs to focus on two pathological processes. One involves resolving conscious negative beliefs and associated emotions, and the other involves managing intrusive, unintegrated memories in the SAM system (Brewin & Holmes, 2003). It is hypothesized that following effective exposure and/or cognitive therapy, the old SAMs remain intact but are no longer triggered and experienced because newly created VAMs become more distinctive and rehearsed and thus have a retrieval advantage when the memory is triggered. In contrast to the assumptions in AIP, it is also proposed that because the old SAMs remain unchanged and are *not integrated* in memory in any way, they retain their potential to be retrieved by the right combination of triggers (Brewin & Holmes, 2003). Also in contrast to AIP, where it is assumed that processing new information in the therapeutic process aids in the *assimilation* of the trauma memory into existing memory networks, it is assumed in dual-representation theory that the new information creates new memories that compete with the old trauma memories. This suggests an *extinction* mechanism over assimilation or reconsolidation of trauma memories.

The precise mechanism by which memories are processed in the treatment of PTSD remains to be empirically clarified. The AIP model proposes that the mechanism of action in EMDR is "the assimilation of adaptive information found in other memory networks linking into the network holding the previously isolated disturbing event" (Solomon & Shapiro, 2008, p. 316). Thus, EMDR transmutes the dysfunctionally stored memory by *integrating* it with

preexisting memory networks. Other psychological theories propose that treatment of memories in PTSD is based on extinction, whereby the process is believed to be that new memories are created that compete for and attain retrieval advantage over old trauma memories (Suzuki et al., 2004). Thus, original trauma memories are able to be retrieved in their original form if triggered by the right combination of cues in the future (Brewin & Holmes, 2003). Solomon and Shapiro (2008) suggest that research comparing recall of original memories and rates and kinds of retrieval patterns can shed light on whether the primary mechanism of action is based on extinction or on association, assimilation, and reconsolidation. They also suggest that EMDR, because of the process of assimilation, may aid in lowered relapse rates when clients experience a similar trauma in the future. Future research needs to compare extinction and reconsolidation models. Solomon and Shapiro suggest that this could be done by following individuals treated with EMDR and exposure-based treatments to investigate if there is a difference in participants' reactions to similar traumas posttreatment.

Future research could also investigate other differences between AIP and emotional processing models of PTSD. For example, the AIP model assumes that trauma symptoms resolve as a result of processing salient or associated memories related to the traumatic event. Alternatively, emotional processing theory (Foa & Rothbaum, 1998) assumes that it is necessary to focus on and relive the traumatic event, to maintain a level of arousal until habituation occurs. Research supporting the AIP model demonstrates that information processing through *association* leads to changes such as reductions in vividness and emotionality and in appraisals related to the memory. Targeting associated memories in non-EMDR treatment studies has also been found to reduce the vividness, distress, and negative beliefs associated with target memories (Wild, Hackman, & Clark, 2008). EMDR may therefore be particularly well suited for individuals who are either avoidant of therapy for fear of having to relive the trauma or cannot tolerate repeated imaginal reliving of the traumatic event. Future research could focus on clarifying if it is possible to reduce trauma symptoms by targeting memories associated to the trauma memory rather than the specific memory of the event.

Summary and Conclusion

Although trauma reactions have been reported for centuries, controversy remains over how to define

PTSD, and the validity of the diagnostic criteria continues to be challenged. Despite this and the theoretical advances that have occurred as our knowledge about PTSD has improved, procedures for the two most effective treatments for PTSD have changed minimally across time. Exposure procedures have changed very little over the years, and the EMDR protocol has remained unchanged since 1991 (Shapiro, 1991). Since Shapiro's (1989) seminal publication that demonstrated the effectiveness of EMDR, what is now known after 20 years of research is that EMDR is an efficacious treatment for adult PTSD. What is also known is that the EMs in EMDR appear to produce various effects that facilitate memory processing and that the processes involved in EMDR are different from those of traditional exposure. However, although evidence is accumulating in support of the AIP model on which EMDR is based, there is still no empirically supported model that is capable of explaining the precise underlying mechanism of EMDR. One must be reminded, though, that even after years of research, we are still struggling to determine the mechanisms through which many psychotherapeutic treatments operate and create change. In addition, the specific mechanisms through which PTSD develops and resolves are not entirely understood, and, as yet, no theory adequately accounts for and explains all the phenomena involved in PTSD. The success of EMDR has challenged existing contemporary theories of PTSD and has advanced our understanding of the therapeutic processes in PTSD. In turn, current theories of PTSD may facilitate our understanding of how EMDR works to resolve PTSD. Comparing and contrasting EMDR and non-EMDR theories of PTSD has more potential to advance our knowledge of effective treatments.

Note

1. Effect sizes pretreatment to posttreatment and pretreatment to follow-up were calculated for the PTSD measures used in each study using Cohen's *d* statistic. Cohen's *d* is calculated by determining the difference in mean scores for each condition divided by the pooled variance (i.e., $SD_{\text{pooled}} = \sqrt{[(SD^2_{\text{pre}} + SD^2_{\text{post}})/2]}$).

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