EMDR and the Adaptive Information Processing Model

Potential Mechanisms of Change

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Eye movement desensitization and reprocessing (EMDR) is a therapeutic approach guided by the adaptive information processing (AIP) model. This article provides a brief overview of some of the major precepts of AIP. The basis of clinical pathology is hypothesized to be dysfunctionally stored memories, with therapeutic change resulting from the processing of these memories within larger adaptive networks. Unlike extinction-based exposure therapies, memories targeted in EMDR are posited to transmute during processing and are then again stored by a process of reconsolidation. Therefore, a comparison and contrast to extinction-based information processing models and treatment is provided, including implications for clinical practice. Throughout the article a variety of mechanisms of action are discussed, including those inferred by tenets of the AIP model, and the EMDR procedures themselves, including the bilateral stimulation. Research suggestions are offered in order to investigate various hypotheses.

Keywords: EMDR; adaptive information processing model; mechanisms of change; information processing; psychotherapy

Eye movement desensitization and reprocessing (EMDR) is a therapeutic approach that emphasizes the brain's intrinsic information processing system and how memories are stored. Current symptoms are viewed as resulting from disturbing experiences that have not been adequately processed and have been encoded in state-specific, dysfunctional form (Shapiro, 1995, 2001, 2007a). The heart of EMDR involves the transmutation of these dysfunctionally stored experiences into an adaptive resolution that promotes psychological health. For EMDR to be applied effectively, the clinician needs a framework that identifies appropriate target memories and order of processing to obtain optimal treatment effects. The adaptive information processing (AIP) model, which informs EMDR treatment, contains a variety of tenets and predictions that implicate various potential agents of change. A comprehensive examination of all the AIP principles is beyond the scope of this article (see Shapiro 2001, 2006). However, because EMDR is a complex approach with many elements, the purpose of this article is to highlight a range of possible agents of change in addition to the eye movement and other bilateral stimulation that have garnered the most attention.

The article begins with a brief overview of the AIP model and the proposed basis of clinical pathology. The observed transmutation of processed memories is discussed, along with conjectures regarding recent research on the reconsolidation of memory, which is a neurobiological process hypothesized to underlie EMDR's effects. As reconsolidation is believed to be different from extinction in terms of the neurobiological processes involved, the similarities and differences between the AIP model and those offered for extinction-based exposure therapies are explored along with implications for clinical practice. Research investigations are proposed to test both the tenets and potential mechanisms of actions. Then the potential mechanisms of action attendant to the EMDR procedures, including the bilateral stimulation, are considered. It should be noted that, although theories abound, the precise mechanisms of change are unknown in *any* form of therapy, and randomized

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studies are necessary for full exploration and delineation. Therefore, suggestions for further research are offered for various hypotheses.

AIP Model

The AIP model explains the basis of pathology, predicts successful clinical outcomes, and guides case conceptualization and treatment procedures. Consistent with other learning theories, the AIP model posits the existence of an information processing system that assimilates new experiences into already existing memory networks. These memory networks are the basis of perception, attitudes, and behavior. Perceptions of current situations are automatically linked with associated memory networks (Buchanon, 2007). For example, the reader can make sense of this sentence because of previous experiences with written English. Similarly, burning one's hand on a stove goes into memory networks having to do with stoves and the potential danger of hot objects. A conflict with a playmate ("me first") and its resolution ("we can share") is accommodated and assimilated into memory networks having to do with relationships and adds to the available knowledge base regarding interpersonal relations and conflict resolution. When working appropriately, the innate information processing system "metabolizes" or "digests" new experiences. Incoming sensory perceptions are integrated and connected to related information that is already stored in memory networks, allowing us to make sense of our experience. What is useful is learned, stored in memory networks with appropriate emotions, and made available to guide the person in the future (Shapiro, 2001).

Pathology According to the AIP Model

Problems arise when an experience is inadequately processed. Shapiro's AIP model (1995, 2001, 2006) posits that a particularly distressing incident may become stored in state-specific form, meaning frozen in time in its own neural network, unable to connect with other memory networks that hold adaptive information. She hypothesizes that when a memory is encoded in excitatory, distressing, state-specific form, the original perceptions can continue to be triggered by a variety of internal and external stimuli, resulting in inappropriate emotional, cognitive, and behavioral reactions, as well as overt symptoms (e.g., high anxiety, nightmares, intrusive thoughts). Dysfunctionally stored memories are understood to lay the foundation for future maladaptive responses, because perceptions of current situations are automatically linked with associated memory networks. Childhood events also may be encoded with survival mechanisms and include feelings of danger that are inappropriate for adults. However, these past events retain their power because they have not been appropriately assimilated over time into adaptive networks.

The AIP model views negative behaviors and personality characteristics as the result of dysfunctionally held information (Shapiro, 2001). From this perspective, a negative self-belief (e.g., "I am not good enough") is not seen as the cause of present dysfunction; it is understood to be a symptom of the unprocessed earlier life experiences that contain that affect and perspective. Attitudes, emotions, and sensations are not considered simple reactions to a past event; they are seen as manifestations of the physiologically stored perceptions stored in memory and the reactions to them. This view of present symptoms as the result of the activation of memories that have been inadequately processed and stored is integral to EMDR treatment. As such, directed belief restructuring and behavioral manipulation are not seen, within the AIP model, to be agents of change because they are considered in other treatments. Research that evaluates the mechanisms for the progressive changes in belief and self-efficacy attendant to EMDR processing compared to other treatments can help shed light on this issue.

Transmutation of Memory

With pathology viewed as the result of unprocessed experiences, processed experiences are seen by the AIP model (Shapiro, 1995, 2001, 2006) to be the basis of mental health. The EMDR protocol involves accessing the dysfunctionally stored information, stimulating the innate processing system through the standardized protocols and procedures (including the bilateral stimulation), and facilitating dynamic linkages to adaptive memory networks, thereby allowing the characteristics of the memory to change as it transmutes to an adaptive resolution. Session transcripts (Shapiro, 2001, 2002; Shapiro & Forrest, 1997) indicate that processing generally occurs through a rapid progression of intrapsychic connections in the session as emotions, insights, sensations, and memories surface and change with each new set of bilateral stimulation. The proposed mechanisms of action include the assimilation of adaptive information found in other memory networks linking into the network holding the previously isolated disturbing event. After successful treatment, it is posited that the memory is no longer isolated, because it appears to be appropriately integrated within the larger memory network. Hence, processing is understood to involve the forging of new associations

and connections enabling learning to take place with the memory then stored in a new adaptive form.

As noted by Shapiro (2007a), the AIP hypothesis appears consistent with recent neurobiological theories of reconsolidation of memory (Cahill & McGaugh, 1998; Suzuki et al., 2004), which propose that an accessed memory can become labile and restored in an altered form. As indicated by Suzuki and colleagues (2004), it appears that reconsolidation and extinction have distinctly different neurobiological mechanisms. While reconsolidation is thought to alter the original memory, extinction processes appear to create a new memory that competes with the old one. This has particular implications for extinction-based exposure models and therapies (e.g., Brewin, 2006; cf. McCleery & Harvey, 2004). The neurological basis of extinction has been related to activity in a particular receptor in the amygdala, and research was conducted using a certain compound known to activate that receptor and to enhance extinction in order to test the mechanism of extinction in exposure-based therapies for acrophobia and social anxiety (Hofmann et al., 2006; Ressler et al., 2004). Unfortunately, it appears as if the compounds are also known to enhance reconsolidation (Lee, Milton, & Everitt, 2006). However, research has also indicated that "pharmacological antagonism of either cannabinoid receptor 1 or L-type voltage-gated calcium channels blocks extinction but not reconsolidation" (Suzuki et al., 2004, p. 4787). This form of research using such compounds (e.g., Rimonabant) would more definitively determine whether reconsolidation is the primary mechanism underlying EMDR's effects.

Other suggested research involves controlled studies comparing extinction-based therapies and EMDR to investigate (1) the kinds of associations available to the client before and after treatment; (2) differences between the ability to access precise visual recollections of the original memory; and (3) the differences in relapse rates, which may be able to shed more light on these possibilities. In particular, the effects of extinction would not be expected to generalize to a new posttreatment event having great similarity to the original critical incident. However, recent case reports indicate that EMDR treatment does generalize to future events (e.g., Shapiro, Kaslow & Maxfield, 2007), suggesting a reconsolidation, rather than extinction, mechanism. EMDR may help to foster resilience and lack of relapse when clients are confronted by a similar trauma (Rost, Hoffman, & Wheeler, in press; Zaghout-Hodali, Alissa, & Dodgson, 2008). Research is needed to systematically follow individuals treated with both EMDR and prolonged exposure treatments to determine whether there is a difference in participants' responses to posttreatment traumas. This would be a simple way to test and compare the predictions and outcomes of the extinction and reconsolidation models.

Similarities and Differences From Other Information Processing Models

The AIP model is in some ways consistent with the emotional processing model that underlies the most widely used exposure-based treatments. In brief, Foa and Kozak (1986) suggest that, for fear reduction to take place, two conditions must be met. First, there has to be activation of the fear memory. Second, corrective information with elements incompatible with the fear structure must be provided so that a new memory can be formed. The incorporation of the new information results in a reduction in fear responses (through in-session and between-session habituation), enabling changes in the meaning of the experience. The AIP model is consistent to the extent that procedures and protocols facilitate the accessing of emotional networks and the incorporation of new information (Rogers & Silver, 2002). The corrective information in exposure-based therapies such as prolonged exposure (e.g., Foa, Rothbaum, Riggs, & Murdock, 1991; Rothbaum, Astin, & Marsteller, 2005) is viewed as coming from the therapeutic situation and the effect of habituation (Foa & Kozak, 1986; Rothbaum et al., 2005). However, the shifts that take place in EMDR suggest that clients incorporate information not only from the therapeutic context but also from memories of previous life experiences (Shapiro, 1995, 2001, 2007b). The linking in of information within and between memories appears to be spontaneous, without therapist prompt, and not the result of repeated and maintained exposure to the memory. Rogers and Silver (2002) concluded that EMDR appears to be consistent with the process of assimilation and accommodation and information processing, rather than habituation. These observations, though speculative, are consistent with the target memory becoming adaptively stored due to reconsolidation, rather than changes taking place because of the formation of a new memory. Once again, research comparing recall of original memories and rates and kinds of retrieval patterns can shed light on whether the primary mechanisms of action in EMDR are based on extinction or are primarily mechanisms involving association, assimilation, and reconsolidation. In addition, process analyses such as those conducted by Lee, Taylor, and Drummond (2006), Rogers et al. (1999), and McCullough (2002) can help shed light on specific mechanisms.

Models and Clinical Practice

Although other information processing models are also based on concepts of memory networks, each model emphasizes different aspects and considerations. The various models guide the practices of their proposed treatments and consider different elements to be the agents of change. For instance, as previously noted, the AIP model concurs with the notion that processing involves the incorporation of "corrective information" (Foa & Kozak, 1986). However, it does not view the change in cognitive appraisal as the key determinant. Instead, the AIP model views processing as an integration of the dysfunctionally stored memory within already existing networks containing adaptive information. Hence, it emphasizes the need for the existence of positive memory networks in order for processing to occur. Therefore, history taking involves assessing whether the positive networks exist and deliberately incorporating them if they do not. This tenet also guides EMDR clinical practice if processing stalls during a treatment session. In that case, the clinician mimics spontaneous processing by deliberately accessing the next positive network already available in the client's history or infuses the information needed to form a positive network that can be linked in.

The two models also differ in the view of current symptomology. The AIP model does not view the primary source of the client's dysfunction to be conditioned responses, current emotional reaction to past event, nor a cognitive appraisal of past event. Rather, the AIP model views the problem as caused by the physiologically stored perceptions (images, thoughts, beliefs, emotions, sensations, smells, etc.) of the past event. Therefore, unlike other models, a prediction based on AIP tenets would be that processing salient memories eliminates the dysfunctional perceptions from storage. For instance, the AIP model predicts that many of the sensations that compose phantom limb pain are actually stored in memory and can be eliminated by processing the salient memories (e.g., Russell, 2007; Schneider, Hoffman, Rost, & Shapiro, 2007, 2008; Shapiro, 2001; Wilensky, 2006). The primary agent of change is not thought to be prolonged exposure, extended focused attention to the event, nor changes in cognitive appraisal. Instead, the change is viewed as a by-product of the processing, which is caused by the internal association process.

This is not to imply that conditioning does not exist, nor that cognitive appraisals are not significant. For instance, current disturbance is addressed in EMDR therapy through first processing the earlier trauma. Indeed, clinical reports indicate that, subsequent to processing the past event, the initially identified trigger is often no longer disturbing (Shapiro et al., 2007). However, the second prong of EMDR treatment involves processing the trigger directly, because new stimuli can become autonomously disturbing through second-order conditioning. However, it is assumed that these conditioning events have themselves been stored in memory and can be adequately treated through processing. It should not be assumed that each therapeutic approach will have completely different mechanisms of change, nor only one. The complexity of any treatment increases the potential number of mechanisms of change interacting to cause positive treatment effects. However, research that investigates the predictions of the various models can not only verify the tenets, but may be able to provide information about possible mechanisms of change.

Treatment Evaluations

Determining the mechanisms of action of any therapy is a complex process, because it involves multiple levels of observation and analysis. Hypotheses may range from constructs such as mind states (see section on "Mindfulness"), specific characteristics of information processing in general (e.g., conjectures regarding the configuration of memory networks and their interaction); the specific kinds of procedures used to evoke change (e.g., prolonged exposure, bilateral stimulation); the underlying processes that have been posited (e.g., extinction, transmarginal inhibition, orienting response, disruption of working memory); the specific physiological concomitants (e.g., decreases in specific neurotransmitters); or the interaction of various brain structures. Research evaluations of a variety of AIP's tenets provide a fruitful springboard for these investigations. Although preliminary research has offered support for various hypotheses, controlled research is needed.

As previously noted, a primary premise of the AIP model is that the source of pathology and health are the physiologically stored memory networks. Pathology is viewed as unprocessed memories, and it is predicted by the AIP model that processing will result in a decrease or elimination of symptomology. Although this has been widely accepted in the treatment of PTSD by addressing the critical (Criterion A) event, the AIP model posits that most forms of pathology are also based on unprocessed memories and can be resolved by allowing the memories causing the complaint to come to an adaptive resolution. This prediction has been supported by a wide range of case studies indicating that problems as diverse as body dysmorphic disorder (Brown, McGoldrick, &

Buchanan, 1997), phantom limb pain (Russell, 2007; Schneider et al., 2007, 2008; Wilensky, 2006), olfactory response syndrome (McGoldrick, Begum, & Brown, 2008), and deviant sexual arousal (Ricci & Clayton, 2008) have been resolved through the processing of core memories. However, controlled research is needed to address these issues by comparing treatment of presenting issues with treatment of core memories.

Mechanisms Suggested by EMDR Procedural Elements During Assessment Phase

As noted by Shapiro (2001), all complex forms of psychotherapy have a range of procedures, and their interactions are responsible for the overall treatment outcome. Hence, as previously noted, it would be too simplistic to assume that any one mechanism of action is responsible for EMDR effects. For instance, there are procedural elements that are consistent with the AIP model that occur during all phases of EMDR that deserve research attention in component analyses to ascertain their relative contribution and measure the potential contributing mechanism of action (see Shapiro, 2001, for a more comprehensive discussion).

Selection of Treatment Targets

Research by Mol and colleagues (2005) compared a range of experiences and reported that events that do not meet the criterion for the designation of Criterion A for PTSD were the cause of trauma symptoms, similar to those in PTSD. This finding provides further evidence of the AIP prediction that the more ubiquitous disturbing events of life ("small *t* trauma") are dysfunctionally stored and the basis of pathology. Hence, on one level of observation, the core mechanism of action inherent in EMDR is posited to be the adaptive processing of the memory. Functionally, this processing is achieved by accessing the stored memory, stimulating the information processing system in a way that permits other memory networks to link into the dysfunctional network, thus transforming the targeted memory, plus targeting memories in a sequence that maximizes therapeutic effect and psychological health. In other words: (a) structured memory access with sequential targeting, (b) stimulation of the information processing system through the procedural elements, and (c) fostering the dynamic integration of other relevant information.

To maximize adaptive information processing, the dysfunctionally stored memories that appear to underlie the presenting symptoms must be identified. These include both large and small *t* traumas and present triggers. In addition, the clinician ensures that there are related relevant memory networks containing positive and/or adaptive information. These are posited to be essential for appropriate linkages to be made during processing. A structured protocol is utilized that prepares the client, comprehensively activates the distressing memory, and elicits relevant aspects of the dysfunctionally stored information.

Mindfulness

The instruction to clients to "let whatever happens, happen" and to "just notice" what is coming up (Shapiro, 1989, 1995, 2001) is consistent with principles of what has come to be known as mindfulness (e.g., Siegel, 2007). Such instructions not only reduce demand characteristics, but may assist clients in noticing what they are feeling and thinking, without judging. Research has shown the therapeutic efficacy of adapting a cognitive set in which negative thoughts and feelings are seen as passing mental events rather than as aspects of self (e.g., Teasdale, 1997; Teasdale et al., 2002). Teasdale (1997) noted the importance of the process of "decentering" or "disidentification," during which the client can move from identifying with the emotion to viewing the thoughts and emotions as passing thoughts and feelings that may or may not be true. This cognitive separation may enable clients to relate to negative experiences with a wider field of awareness, which can increase coping ability (Beck, Rush, Shaw, & Emery, 1979) and enhance the client's sense of efficacy and mastery (Shapiro, 1995, 2001). From an AIP perspective, the increased coping ability and self-efficacy become encoded in the client's memory network. This can enhance the client's ability to stay present with difficult material during processing and provide positive, adaptive information that is available to link into memory networks holding dysfunctionally stored information. Further, the EMDR procedures, including the neurobiological concomitants of the eye movements that result in dearousal (Barrowcliff, Gray, Freeman, & MacCulloch, 2004; Barrowcliff, Gray, MacCulloch, Freeman, & MacCulloch, 2003; Elofsson, von Scheele, Theorell, & Sondergaard, 2008) may produce the state of mind referenced by Teasdale. Controlled research is needed to evaluate these questions.

Alignment of Memory Fragments

Experience that has been insufficiently processed has been posited to be stored in memory fragments (van der Kolk & Fisler, 1995). Therefore, the alignment of memory components appears to be a procedural element that facilitates processing. The EMDR protocol

involves eliciting the image, currently held negative belief, desired positive belief, current emotion, and physical sensation. This procedure, potentially tapping into different parts of the brain, enables activation of different aspects of the dysfunctionally stored information, which have been posited to be encoded in different memory networks, each having different associations and linkages (Buckner & Wheeler, 2001; Gottfried, Smith, Rugg, & Dolan, 2004; Shapiro, 1995, 2001). The assessment phase aligns these primary aspects of the negative memory, which is consistent with the BASK (behavior, affect, sensation, and knowledge) model of dissociation (Braun, 1988). This procedural reconnection of the disturbing material may help the client make sense of the experience and facilitate storage in narrative memory.

Somatic Awareness

Directing the client to attend to the physical sensations after identifying the representative or worst image, the negative cognition, and emotions, may also be a procedural element particularly relevant to positive outcomes. This may help clients identify and separate physical sensations from their negative interpretations that reflect overidentification with their emotions/sensations. Attending to physical sensation and emotion as separate from negative interpretations may help the client recognize the changeability of sensation. For example, the client can shift from identifying with the emotion (e.g., "I am afraid") to recognizing that the experience of sensations in the stomach and chest is associated with fear. This can increase the client's self-efficacy and sense of mastery (Shapiro, 1995, 2001), which, from an AIP perspective, increases the positive information encoded in the brain available to link into memory networks holding dysfunctionally stored information.

Cognitive Restructuring

Cognitive restructuring is a procedural element evident in the assessment phase contributing another possible mechanism of action, with the client identifying both negative and positive cognitions. Identifying the irrational self-belief and restructuring and reframing the belief into an adaptive self-belief can facilitate the therapeutic process (Beck et al., 1979). However, in the EMDR assessment phase, there are no specific attempts to change or reframe the client's currently held belief. It is assumed that the belief will spontaneously shift during the course of the subsequent processing. Nevertheless, from an AIP perspective, forging a preliminary association between the negative cognition with more adaptive information that contradicts the negative experience is believed to facilitate the subsequent processing by activating relevant adaptive networks. Process analyses of spontaneous changes in cognitive content during EMDR treatment can help to evaluate the contribution of the cognitive element to treatment outcome.

Mechanisms Suggested by EMDR Procedural Elements During Desensitization and Installation Phases

Perceived Mastery

Perceived mastery may be another important procedural element. During EMDR, clients may increase a sense of mastery for their ability to mentally circumscribe and manipulate the disturbing material through the ongoing sequences of imagery accessing, attention, and interruption. This can increase coping efficacy, which can enhance the client's ability to manage stress, anxiety, and depression in threatening situations (Bandura, 2004). From an AIP perspective, the client's experience of mastery becomes encoded in the brain as adaptive information available to link into memory networks holding dysfunctionally stored information. It would be interesting for research to compare the effectiveness and efficiency of conditions that utilize the bilateral stimulation, while comparing continuous attention to the traumatic material with interrupted attention, as is done in standard EMDR practice. As evident in this section, it remains an open question regarding whether a sense of mastery increases because of the sequencing, or it is merely the interrupted attention along with the bilateral stimulation, or a combination of both that are primary contributing elements and significant mechanisms of change.

Potential Effects of Eye Movements

Structured procedures are utilized to stimulate the relevant memory networks and engage the associative processing of the brain during the desensitization and installation phases. In accordance with the AIP model, the dysfunctional information is accessed as it is currently stored, and bilateral stimulation is applied to assist in stimulating the brain's intrinsic processing system, allowing information from other neural networks to link in. It is hypothesized that the creation of associations is one of the mechanisms that transmutes the memory. There are a variety of theories regarding how the eye movements contribute to this process.

The AIP model views eye movements and other forms of bilateral stimulation as one of the elements that serve to facilitate the information processing. Unfortunately, existing randomized controlled component analyses using clinical populations and treatment outcome measures were flawed (see Chemtob, Tolin, van der Kolk, & Pitman, 2000; Shapiro, 2001) and need to be conducted under more rigorous conditions. However, there is an expanding body of research that has evaluated the eye movements in isolation with nondiagnosed populations (Gunter & Bodner, 2008). A number of such studies indicate that eye movements produce a desensitization effect during the accessing of disturbing memories. For instance, in a laboratory study, Barrowcliff et al. (2004) reported that eye movements lowered physiological arousal on skin conductance electrodermal measures.

Several PTSD treatment studies (Elofsson et al., 2008; Sack, Hofmann, Wizelman, & Lempa, this issue; Sack, Lempa, & Lemprecht, 2007; Sack, Lempa, Steinmetz, Lamprecht, & Hofmann, 2008; Wilson et al, 1996), examined the specific physiological effects of eye movements during EMDR treatment sessions. The results suggested that eye movements resulted in an increase in parasympathetic activity and a decrease in psychophysiological arousal. Similar physiological results were found following one session of EMDR, evidenced by lowered heart rate and skin conductance (Aubert-Khalfa, Roques, & Blin, 2008).

In other studies, the eye movements have been found to decrease vividness and emotionality of negative and positive memories (Barrowcliff et al, 2004; Gunter & Bodner, 2008; Kavanagh, Freese, Andrade, & May, 2001; Maxfield, this issue; Sharpley, Montgomery, & Scalzo, 1996; van den Hout, Muris, Salemink, & Kindt, 2001). At this time, it is unknown whether the change in vividness precedes or follows the physiological dearousal and whether these occur together or are separate elements. Nevertheless, a variety of hypotheses have been advanced regarding the mechanism of action related to the bilateral stimulation. These include the orienting response (MacCulloch & Feldman, 1996), rapid eye movement sleep (Stickgold, 2002, this issue), and working memory (Andrad, Kavanagh, & Baddeley, 1997). The apparent desensitization effects reported in various studies are predicted by all these hypotheses. Additional research is needed to identify actual mechanism of actions and to determine whether there is an interaction of various mechanisms. Further, studies are needed to evaluate the relationship between the reported changes and treatment outcome. In other words, we do not yet know the sequential order of these effects and cannot assume causality. Does heart rate decrease because the memory is becoming less distressing due to processing, or does the decreased arousal facilitate processing of the memory so that it becomes less distressing? Only randomized controlled research under the appropriate conditions can settle these questions (see Shapiro, 2001).

With the lowering of arousal and decrease in vividness and emotionality of negative memories, information from other memory networks may be able to link into the network holding the dysfunctionally stored information (see Shapiro, 1995, 2001). Stickgold (2002) proposes that the eye movements utilized in EMDR produce a repetitive redirecting of attention that induces a neurobiological state similar to REM sleep, which increases access to less dominant associations and could result in a cortical integration of disturbing memories into semantic networks, reducing the strength of the distressing memories. The transmutation of the memory appears to include a shift of the sensory information from implicit to episodic and then semantic memory (Siegel, 2002; Stickgold, 2002).

Preliminary support for changes in memory retrieval comes from Christman, Garvey, Propper, and Phaneuf (2003) and Propper and Christman (this issue) showing that eye movements enhanced retrieval of episodic memories in laboratory studies with right-handed nonclinical participants. Propper, Pierce, Geisler, Christman, and Bellorado (2007) posited that eye movements may change interhemispheric coherence in frontal areas. A study by Kuiken, Bears, Miall, and Smith (2001–2002) found that eye movements were related to increased attentional flexibility. Research is needed to replicate these studies in clinical settings with diagnosed left- and right-handed participants. Additional research should evaluate the premise that the quality of the targeted memory is correlated with an increased number of associative nontraumatic memories. This would provide an opportunity to test the hypothesis generated from the Suzuki et al (2004) animal research. They suggested that, when a memory is activated, it appears to become more labile, so that the memory can reconsolidate in an altered form. Hence, it is possible that reconsolidation provides the capacity, as Przybyslawski, Roullet, and Sara (1999, p.) pointed out, to permit "reorganization of the existing memory as a function of new information in the retrieval environment."

Summary and Conclusion

The AIP model (Shapiro, 1995, 2001) specified that the dysfunctionally stored memory was changed through the linking up of networks containing adaptive information. This memory was then posited to

be restored in adaptive form. The implication from an AIP perspective is that new associations link into the previously isolated network, causing a transmutation of the memory itself. This, in turn, supports the use of procedures that encourage an internal associative process. Although the field of neurobiology is currently unable to specify the mechanism by which this would be achieved, the theory of reconsolidation and the recent research supporting it seems to suggest such a mechanism.

When viewing EMDR effects through the lenses of other dominant information processing models (e.g., Foa & McNally, 1996), the elicitation of associations as conducted in EMDR would be considered antithetical to positive treatment effects because it would foster avoidance and simply reinforce negative behavioral and cognitive outcomes. While Foa's information processing model specifically states the need to access the dysfunctional memory network and the need to incorporate corrective information, the emphasis is on an alteration of the cognitive appraisal of the experience through the exposure to the disturbance in a safe therapeutic environment. As previously noted, AIP considers the change of maladaptive beliefs to be a by-product of the processing, not the agent of change. The mechanism of change is considered to be incorporation of adaptive information through the internal associations to information already stored in the brain. However, as previously noted, many theorists (e.g., Foa & Kozak, 1986; Foa & McNally, 1996; Marks, Lovell, Noshirvani, Livanou, & Thrasher, 1998; Rothbaum & Foa, 1996) presume that extinction is the underlying mechanism of prolonged exposure therapy. And, as indicated by Suzuki et al. (2004), the process of extinction is believed to produce another competing memory, not alter the original one. Hence, studies that compare EMDR and exposure therapies based on extinction can shed light on the underlying process and determine whether EMDR is indeed based on memory reconsolidation effects. In addition to studies that evaluated effects in conditions that would be hypothesized to cause relapse in extinction-based treatments (see Suzuki et al., 2004), it would be useful to see whether there are differential effects in studies of deviant arousal and phantom limb pain, which have been previously considered to be intractable conditions but which appear to be positively impacted by EMDR (e.g., Schneider et al., 2007, 2008).

The AIP model posits that the simultaneous accessing of the traumatic memory network combined with the reduction in distress caused by eye movements and the procedures used to guide the client's attention leads to a comprehensive memory shift, with new associations being able to link into the disturbing memory. The structured procedural elements as well as the bilateral stimulation are viewed as having additive effects in the adaptive processing. As noted by Smyth, Rogers, and Maxfield (2004), results from unpublished studies suggest that eye movement appears to add to treatment effects that are produced by the remainder of the procedures alone. For that reason, large samples of suitable diagnosed populations are necessary to ascertain the relative contribution of the various elements (see Shapiro, 2001, for a more comprehensive discussion). Previous component analyses using treatment outcomes have been flawed because of the choice of population, treatment dose, and outcome measures (Chemtob et al., 2000). While studies of eye movement in isolation have shown pronounced and significant effects, these studies need to be replicated with clinical participants. Determining the value and neurobiological concomitants of the bilateral stimulation in relation to treatment outcome is a necessary next step. Further, as previously mentioned, it is presently undetermined by research whether the decrease in memory image vividness is related to direct changes caused by the effect of eye movements on working memory (Andrade et al., 1997) or whether the image changes because of the eye movements' direct effect on physiological arousal (Elofsson et al., 2008; Sack et al., 2007, 2008; Wilson et al., 1996).

During the past twenty years, EMDR has evolved from a desensitization technique to an integrative psychotherapeutic approach. The AIP model is the theoretical foundation that integrates the many procedural elements that contribute to EMDR effects. Present-day problems, unless physically or chemically based, are due to past experiences that have not been adequately processed and are dysfunctionally stored. Although the AIP model is not tied to a specific neurobiological mechanism, it provides an understanding of therapeutic change as achieved through the processing of dysfunctional memories and their integration within larger adaptive networks. Functionally, this is achieved by accessing the dysfunctionally stored memory and stimulating the information processing system in a way that permits other memory networks to link into the dysfunctional network, which transforms the targeted memory. Although the precise mechanisms of change are unknown, studies show that the eye movements utilized in EMDR are correlated with a desensitization effect, an increase in parasympathetic activity, and a decrease in psychophysiological arousal. Consistent with research showing increased attentional flexibility and memory

retrieval, the lowering of arousal may enable information from other memory networks to link into the network holding the dysfunctionally stored information. However, further research is needed to determine the causality of such effects and the biological concomitants of eye movements and other types of stimulation utilized in EMDR.

Ultimately, the mechanisms of action are viewed as facilitating reorganizations of memory networks, with the AIP model guiding the EMDR procedures needed to orchestrate the clinical attitudes, client awareness, and neurobiological connections of encoded memories needed to achieve these ends. For this reason, it is suggested that component analyses be conducted with diagnosed populations and treatment conditions and doses consistent with the clinical complaint (see Shapiro, 2001, for a comprehensive discussion of research parameters). Testing the predictions of AIP is a useful step in determining the appropriate clinical conditions for comparing the mechanisms of change in various psychotherapy approaches.

References

Andrade, J., Kavanagh, D., & Baddeley, A. (1997). Eye-movement and visual imagery: A working memory approach to the treatment of post-traumatic stress disorder. *British Journal of Clinical Psychology, 36,* 209–223.

Aubert-Khalfa, S., Roques, J., & Blin, O. (2008). Evidence of a decrease in heart rate and skin conductance response in PTSD patients after a single EMDR session. *Journal of EMDR Practice and Research, 2,* 51–56.

Bandura, A. (2004). Swimming against the mainstream: The early years from chilly tributary to transformative mainstream. *Behaviour Research and Therapy, 42,* 613–630.

Barrowcliff, A. L., Gray, N. S., Freeman, T. C. A., & MacCulloch, M. J. (2004). Eye-movements reduce the vividness, emotional valence and electrodermal arousal associated with negative autobiographical memories. *Journal of Forensic Psychiatry and Psychology, 15,* 323–345.

Barrowcliff, A. L., Gray, N. S., MacCulloch, S., Freeman, T.C.A., & MacCulloch, M. J. (2003). Horizontal rhythmical eye movements consistently diminish the arousal provoked by auditory stimuli. *British Journal of Clinical Psychology, 42,* 289–302.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression.* New York: Guilford Press.

Braun, B. G., (1988). The BASK model of dissociation. *Dissociation, 1,* 4–23.

Brown, K. W., McGoldrick, T., & Buchanan, R. (1997). Body dysmorphic disorder: Seven cases treated with eye movement desensitization and reprocessing. *Behavioural & Cognitive Psychotherapy, 25,* 203–207.

Buchanon, T. W. (2007). Retrieval of emotional memories. *Psychological Bulletin, 5,* 761–779.

Buckner, R. L., & Wheeler, M. E. (2001). The cognitive neuroscience of remembering. *Nature Reviews Neuroscience, 2,* 624–634.

Brewin, C. R. (2006). Understanding cognitive-behaviour therapy: A retrieval competition account. *Behaviour Research and Therapy, 44,* 765–784.

Cahill, L., & McGaugh, J. L. (1998). Mechanisms of emotional arousal and lasting declarative memory. *Trends in Neuroscience, 21,* 294–299.

Chemtob, C. M., Tolin, D. F., van der Kolk, B. A., & Pitman, R. K. (2000). Eye movement desensitization and reprocessing. In E. A. Foa, T. M. Keane, & M. J. Friedman (Eds.), *Effective treatments for PTSD: Practice guidelines from the International Society for Traumatic Stress Studies* (pp. 139–155, 333–335). New York: Guilford Press.

Christman, S. D., Garvey, K. J., Propper, R. E., & Phaneuf, K. A. (2003). Bilateral eye movements enhance the retrieval of episodic memories. *Neuropsychology, 17,* 221–229.

Elofsson, U.O.E., von Scheele, B., Theorell, T., & Sondergaard, H. P. (2008). Physiological correlates of eye movement desensitization and reprocessing. *Journal of Anxiety Disorders,* 622–624.

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin, 99*(1), 20–35.

Foa, E. B., & McNally, R. J. (1996). Mechanisms of change in exposure therapy. In R. M. Rapee (Ed.), *Current controversies in the anxiety disorders* (pp. 329–343). New York: Guilford Press.

Foa, E. B., Rothbaum, B. O., Riggs, D. S., & Murdock, T. B. (1991). Treatment of posttraumatic stress disorder in rape victims: A comparison between cognitive-behavioral procedures and counseling. *Journal of Consulting and Clinical Psychology, 59,* 715–723.

Gottfried, J. A., Smith, A. P., Rugg, M. D., & Dolan, R. J. (2004). Remembrance of odors past: Human olfactory cortex in cross-modal recognition memory. *Neuron, 42,* 687–695.

Gunter, R. W., & Bodner, G. E. (2008). How eye movements affect unpleasant memories: Support for a working-memory account. *Behaviour Research and Therapy, 46,* 913–931.

Hofmann, S. G., Meuret, A. E., Smits, J. A., Simon, N. M., Pollack, M. H., et al. (2006). Augmentation of exposure therapy with D-cycloserine for social anxiety disorder. *Archives of General Psychiatry, 63,* 298–304.

Kavanagh, D. J., Freese, S., Andrade, J., & May, J. (2001). Effects of visuospatial tasks on desensitization to emotive memories. *British Journal of Clinical Psychology, 40,* 267–280.

Kuiken, D., Bears, M., Miall, D., & Smith, L. (2001–2002). Eye movement desensitization reprocessing facilitates attentional orienting. *Imagination, Cognition and Personality, 21,* 3–20.

Lee, C., Taylor, G., & Drummond, P. D. (2006). The active ingredient in EMDR: Is it traditional exposure or dual focus of attention? *Clinical Psychology and Psychotherapy, 13,* 97–107.

Lee, J. L., Milton, A. L., & Everitt, B. J. (2006). Reconsolidation and extinction of conditioned fear: Inhibition and potentiation. *Journal of Neuroscience, 26*(39), 10051–10056.

Marks, I., Lovell, K., Noshirvani, H., Livanou, M., & Thrasher, S. (1998). Treatment of posttraumatic stress disorder by exposure and/or cognitive restructuring. *Archives of General Psychiatry, 55,* 317–325.

MacCulloch, M. J., & Feldman, P. (1996). Eye movement desensitization treatment utilizes the positive visceral element of the investigatory reflex to inhibit the memories of post-traumatic stress disorder: A theoretical analysis. *British Journal of Psychiatry, 169,* 571–579.

McCleery, J. M., & Harvey, A. G. (2004). Integration of psychological and biological approaches to trauma memory: Implications for pharmacological prevention of PTSD. *Journal of Traumatic Stress, 17,* 485–496.

McCullough, L. (2002). Exploring change mechanisms in EMDR applied to "small t trauma" in short term dynamic psychotherapy: Research questions and speculations. *Journal of Clinical Psychology, 58,* 1465–1487.

McGoldrick, T., Begum, M., & Brown, K. W. (2008). EMDR and olfactory reference syndrome: A case series. *Journal of EMDR Practice and Research, 2,* 63–68.

Mol, S.S.L., Arntz, A., Metsemakers, J. F. M., Dinant, G., Vilters-Van Montfort, P. A. P., & Knottnerus, A. (2005). Symptoms of post-traumatic stress disorder after non-traumatic events: Evidence from an open population study. *British Journal of Psychiatry, 186,* 494–499.

Propper, R., Pierce, J. P., Geisler, M. W., Christman, S. D., & Bellorado, N. (2007). Effect of bilateral eye movements on frontal interhemispheric gamma EEG coherence: Implications for EMDR therapy. *Journal of Nervous and Mental Disease, 195,* 785–788.

Przybyslawski, J., Roullet, P., & Sara, S. J. (1999). Attenuation of emotional and nonemotional memories after their reactivation: Role of β adrenergic receptors. *Journal of Neuroscience, 19,* 6623–6628.

Ressler, K. J., Rothbaum, B. O., Tannenbaum, L., Anderson, P., Graap, K., et al. (2004). Cognitive enhancers as adjuncts to psychotherapy: Use of D-cycloserine in phobic individuals to facilitate extinction of fear. *Archives of General Psychiatry, 61,* 1136–1144.

Ricci, R. J., & Clayton, C. A. (2008). Trauma resolution treatment as an adjunct to standard treatment for child molesters: A qualitative study. *Journal of EMDR Practice and Research, 1,* 41–50.

Rogers, S., & Silver, S. M. (2002). Is EMDR an exposure therapy? A review of trauma protocols. *Journal of Clinical Psychology, 58,* 43–59.

Rogers, S., Silver, S., Goss, J., Obenchain, J., Willis, A., & Whitney, R. (1999). A single session, controlled group study of flooding and eye movement desensitization and reprocessing in treating posttraumatic stress disorder among Vietnam war veterans: Preliminary data. *Journal of Anxiety Disorders, 13,* 119–130.

Rost, C., Hofmann, A., & Wheeler, K. (in press). EMDR treatment of workplace trauma: A case series. *Journal of EMDR Practice and Research.*

Rothbaum, B. O., Astin, M. C., & Marsteller, F. (2005). Prolonged exposure versus eye movement desensitisation (EMDR) for PTSD rape victims. *Journal of Traumatic Stress, 18,* 607–616.

Rothbaum, B. O., & Foa, E. B. (1996). Cognitive-behavioral therapy for posttraumatic stress disorder. In B. A. van der Kolk, A. C. McFarlane, & L. Weisaeth, (Eds.), *Traumatic stress: The effects of overwhelming experience on mind, body and society* (pp. 491–509). New York: Guilford Press.

Russell, M. (2008). Treating traumatic amputation-related phantom limb pain: A case study utilizing eye movement desensitization and reprocessing (EMDR) within the armed services. *Clinical Case Studies, 7,* 136–153.

Russell, M. C., Silver, S. M., Rogers, S., & Darnell, J. N. (2007). Responding to an identified need: A joint Department of Defense/Department of Veterans Affairs training program in eye movement desensitization and reprocessing (EMDR) for clinicians providing trauma services. *International Journal of Stress Management, 14*(1), 61.

Sack, M., Lempa, W., & Lemprecht, W. (2007). Assessment of psychophysiological stress reactions during a traumatic reminder in patients treated with EMDR. *Journal of EMDR Practice and Research, 1,* 15–23.

Sack, M., Lempa, W., Steinmetz, A., Lamprecht, F., & Hofmann, A. (2008). Alterations in autonomic tone during trauma exposure using eye movement desensitization and reprocessing (EMDR): Results of a preliminary investigation. *Journal of Anxiety Disorders, 22*(7), 1264–1271.

Schneider, J., Hofmann, A., Rost, C., & Shapiro, F. (2007). EMDR and phantom limb pain: Case study, theoretical implications, and treatment guidelines. *Journal of EMDR Practice and Research, 1,* 31–45.

Schneider, J., Hofmann, A., Rost, C., & Shapiro, F. (2008). EMDR in the treatment of chronic phantom limb pain. *Pain Medicine, 9,* 76–82.

Shapiro, F. (1989). Efficacy of the eye movement desensitization procedure in the treatment of traumatic memories. *Journal of Traumatic Stress Studies, 2,* 199–223.

Shapiro, F. (1995). *Eye movement desensitization and reprocessing: Basic principles, protocols, and procedures.* New York: Guilford Press.

Shapiro, F. (2001). *Eye movement desensitization and reprocessing: Basic principles, protocols, and procedures* (2nd ed.). New York: Guilford Press.

Shapiro, F. (2002) Paradigms, processing, and personality development. In F. Shapiro (Ed.), *EMDR as an integrative psychotherapy approach: Experts of diverse orientations explore the paradigm prism* (pp. 3–26). Washington, DC: American Psychological Association Press.

Shapiro, F. (2006). *New notes on adaptive information processing.* Hamden, CT: EMDR Humanitarian Assistance Programs.

Shapiro, F. (2007a). EMDR and case conceptualization from an adaptive information processing perspective. In F. Shapiro, F. Kaslow, & L. Maxfield (Eds.), *Handbook of EMDR and family therapy processes* (pp. 3–36). New York: Wiley.

Shapiro, F. (2007b, September). *EMDR update: Theory, research, and practice.* Keynote presentation at the EMDR International Association annual conference, Dallas, TX.

Shapiro, F., & Forrest, M. S. (1997). *EMDR: The breakthrough therapy for overcoming anxiety, stress, and trauma.* New York: Basic Books.

Shapiro, F., Kaslow, F., & Maxfield, L. (Eds.). (2007). *Handbook of EMDR and family therapy processes.* New York: Wiley.

Sharpley, C. F., Montgomery, I. M., & Scalzo, L. A. (1996). Comparative efficacy of EMDR and alternative procedures in reducing the vividness of mental images. *Scandinavian Journal of Behaviour Therapy, 25,* 37–42.

Siegel, D. J. (2002). The developing mind and the resolution of trauma: Some ideas about information processing and an interpersonal neurobiology of psychotherapy. In F. Shapiro (Ed.), *EMDR as an integrative psychotherapy approach: Experts of diverse orientations explore the paradigm prism* (pp. 85–122). Washington, DC: American Psychological Association Press.

Siegel, D. J. (2007). *The mindful brain.* New York: Norton.

Smyth, N. J., Rogers, S., & Maxfield, L. (2004, September). *What about eye movements?* Plenary at the annual conference of the EMDR International Association, Montreal, Canada.

Stickgold, R. (2002). EMDR: A putative neurobiological mechanism of action. *Journal of Clinical Psychology, 58,* 61–75.

Suzuki, A., Josselyn, S. A., Frankland, P. W., Masushige, S., Silva, A. J., & Kida, S. (2004). Memory reconsolidation and extinction have distinct temporal and biochemical signatures. *Journal of Neuroscience, 24,* 4787– 4795.

Teasdale, J. D. (1997). The relationship between cognition and emotion: The mind-in-place in mood disorders. In D. M. Clark & C. G. Fairburn (Eds.), *Science and practice of cognitive behaviour therapy* (pp. 67–93). Oxford: Oxford University Press.

Teasdale, J. D., Moore, R. G., Hayhurst, H., Pope, M., Williams, S., & Segal, Z. (2002). Metacognitive awareness and prevention of relapse in depression: Empirical evidence. *Journal of Consulting and Clinical Psychology, 70,* 275–287.

van den Hout, M., Muris, P., Salemink, E., & Kindt, M. (2001). Autobiographical memories become less vivid and emotional after eye movements. *British Journal of Clinical Psychology, 40,* 121–130.

van der Kolk, B. A., and Fisler, R. (1995). Dissociation and the fragmentary nature of traumatic memories: Overview and exploratory study. *Journal of Traumatic Stress, 8,* 505–525.

Wilensky, M. (2006). Eye movement desensitization and reprocessing (EMDR) as a treatment for phantom limb pain. *Journal of Brief Therapy, 5,* 31–44.

Wilson, D., Silver, S. M., Covi, W., & Foster, S. (1996). Eye movement desensitization and reprocessing: Effectiveness and autonomic correlates. *Journal of Behaviour Therapy and Experimental Psychiatry, 27,* 219–229.

Zaghout-Hodali, M., Alissa, F., & Dodgson, P. (2008). EMDR and resilience in children in ongoing trauma. *Journal of EMDR Practice and Research, 1,* 106–113.

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