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

"Experiencing trauma is an essential part of being human; history is written in blood" (van der Kolk & McFarlane, 1996, p. 3). As humans, however, we do have an extraordinary ability to adapt to trauma, and resilience is our most common response (Bonanno, 2005). Nonetheless, traumatic experiences can alter one's social, psychological, and biological equilibrium, and for years memories of the event can taint experiences in the present. Despite advances in our knowledge of posttraumatic stress disorder (PTSD) and the development of psychosocial treatments, almost half of those who engage in treatment for PTSD fail to fully recover (Bradley, Greene, Russ, Dutra, & Westen, 2005). Furthermore, no theory as yet provides an adequate account of all the complex phenomena and processes involved in PTSD, and our understanding of the mechanisms that underlie effective treatment, such as eye movement desensitization and reprocessing (EMDR) and exposure therapy remains unclear.

Historical Overview of PTSD

The psychological effects of trauma have been reported for centuries. The earliest evidence of exposure to a traumatic event leading to trauma reactions was recorded on a cuneiform tablet that described people's reactions to an event involving the death of King Urnamma (2111–2094 B.C.) in battle (Ben-Ezra, 2001). In the 19th century, Hermann Oppenheim (1858–1919) coined the term "traumatic neurosis," and debates began as to what constitutes the main etiological factor of trauma reactions. Neurologist Jean Martin Charcot (1825–1893) argued against Oppenheim's idea that psychic neurosis was caused by organic processes and proposed that the etiology of trauma symptoms were in fact the response of predisposed individuals to a terrifying event. Alternatively, Pierre Janet (1859–1947), who studied under Charcot, suggested that subconscious fixed ideas, or cognitive schemas established earlier in life, were responsible for neurotic trauma symptoms. Janet argued that the event

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itself was not the "cause of the consequent illness, but it was necessary to assign a role to the memories left by the accident" (Janet, 1924, p. 39). He believed that the encoding and retrieval of memories were central organizing factors of the mind. Joseph Breuer and Sigmund Freud (1893) also argued that the event was not the crucial etiological factor but proposed that the main casual factor was the "susceptibility of the person affected" (p. 56).

To some extent, the debate still exists today over what constitutes the core element underlying trauma reactions and whether it is the actual event, the unintegrated memories, the associated meaning, or personal vulnerability. The debate is reflected in the differing core assumptions of theories of PTSD and the focus of therapies used to treat PTSD, and it is also evident across the changing PTSD diagnostic criteria. The *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-I;* 1st ed.; American Psychiatric Association, 1952) called what is now known as PTSD "stress response syndrome," and the main causal factor was stressful environmental events, such as natural disasters or war. The *DSM-II* (APA, 1968) referred to PTSD-like symptoms as "transient situational disturbance" (p. 48), and the etiology involved the individual, not the event, as it was believed that "if the patient has good adaptive capacity his symptoms usually recede as the stress diminishes." The *DSM-III* (APA, 1980) defined PTSD as a syndrome that erupted in response to a "stressor that would evoke significant symptoms of distress in almost everyone" (p. 238), thus implying that the etiological factor was no longer individual weakness but rather the event.

Defining PTSD: Controversies Over Criterion A

PTSD was and remains a unique diagnosis because the diagnostic criteria have always implied the assumption of specific etiology. In contrast to all other DSM psychiatric diagnoses (i.e., depression, schizophrenia, generalized anxiety disorder), there must be a known etiological component, an external event (criterion A: the stressor criterion) that directly relates to the trauma symptoms. However, what constitutes a traumatic stressor has changed across *DSM* revisions. The *DSM-IV-TR* (APA, 2000) currently defines the criterion A(1) stressor as when a "person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others" (p. 467). In addition, the stressor must also meet criterion A(2), which states that the stressor must be accompanied by fear, helplessness, or horror. Using such a definition, the lifetime prevalence of exposure to traumatic events may be as high as 89% (Breslau, 2001). Epidemiological research has consistently revealed that experiencing trauma is relatively common, but many people go on with their lives without becoming haunted by memories of what happened, and only a minority of trauma victims, between 5% and 10%, develop PTSD (van der Kolk & McFarlane, 1996). Such findings stimulated research in to the question of why some people develop PTSD and require treatment while others do not.

Epidemiological research and meta-analyses of PTSD risk factor research have found that more variance is accounted for by peritraumatic processes, previous trauma and psychological history, and post-trauma factors than the nature of the traumatic event itself (Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2003, 2008). A growing body of literature clearly demonstrates that the PTSD syndrome can result from "small t" events that do not meet criterion A(1) (i.e., Avina & O'Donohue, 2002; Dattilio, 2004). These findings justify recent proposals to remove criterion A from the forthcoming *DSM-V* (Rosen, Spitzer, & McHugh, 2008) and is further justified by research that has verified that stressful life events (chronic illness, marital discord) can be as traumatic as criterion A events and generate just as many PTSD symptoms (Mol et al., 2005). In addition, Bodkin, Pope, Detke, and Hudson (2008) recently demonstrated that the prevalence of the PTSD syndrome was equivalent (78%) among patients who had experienced *DSM-IV* trauma and those who had not. The authors concluded that PTSD may therefore "harbour an uncertain theory of aetiology within its name" (Bodkin et al., 2007, p. 181), and the definition may exclude people who would benefit from PTSD treatment but fail to meet current diagnostic criteria.

Evidence highlights that criterion A is not sufficient or necessary to bring about the PTSD syndrome. Therefore, it is possible that in *DSM-V,* criterion A be removed from the diagnostic criteria of PTSD and the stressor be treated as a risk factor rather than a causative event. However, removing criterion A from the PTSD diagnostic criteria so that it becomes like all other psychiatric diagnoses whereby presumed causative factors, such as precipitating events, are assessed as a risk factor (Rosen & Lilienfeld, 2008; Rosen et al., 2008) raises the question whether removing it keeps PTSD a unique and separate distinct clinical entity (Rosen et al., 2008). Research highlights that comorbidity is the rule rather than the exception for PTSD, and many of the symptom criteria that define PTSD

also define the very disorders with which PTSD most frequently co-occurs (i.e., major depression, specific phobia, generalized anxiety disorder, and panic disorder) (Rosen et al., 2008). Several studies suggest that PTSD and depression arise from similar predictive variables and a shared vulnerability such that the disorders should not be viewed as separate distinct entities (i.e., Breslau, Davis, Peterson, & Schultz, 2000). However, other systematic research indicates that reliving experiences or flashbacks are a unique feature of PTSD. For example, Reynolds and Brewin (1998) interviewed matched patients with either PTSD or major depression and a sample of nonclinical controls about their most prominent intrusive cognition, coping strategies, and emotional responses. Their findings support the claim that flashbacks are distinctive to PTSD, as flashbacks were reported as the most frequent intrusive cognition by 43% of the PTSD group, only 9% of those with depression, and none of the nonclinical controls.

In addition to the controversy surrounding criterion A(1) for PTSD, debate also exists regarding the validity of criterion A(2), which requires emotional responses to the stressor that involve "intense fear, helplessness, or horror" (APA, 2000, p. 467). Research that has examined retrospective reports of peritraumatic fear has found that fear is generally positively correlated with the presence and severity of PTSD symptoms (i.e., Brewin, Andrews, & Rose, 2000), and since some fear is generally present with PTSD, it is often assumed that it is the predominant emotion that maintains PTSD symptoms. Most theoretical accounts of PTSD have emphasized that experiencing intensive fear is important in the development of PTSD (i.e., Foa & Kozak, 1986; Keane, Fairbank, Caddell, Zimering, & Bender, 1985). However, the evidence is mixed with regard to the role of other criterion A(2) emotions: horror and helplessness. Although some authors have found significant correlations between PTSD symptoms and peritramuatic helplessness and horror (i.e., Brewin, Andrews, & Rose, 2000), Roemer, Orsillo, Borkovec, and Litz (1998) found no significant correlation between PTSD and reports of horror, and Palmer, Kagee, Coyne, and DeMichele (2004) found no effects of either horror or helplessness. It has also been noted that PTSD can develop without experiencing any criterion A(2) emotions during the trauma (Brewin, Andrews, & Rose, 2000) and that nonfear emotions, such as shame, anger, and guilt, are often predominant emotions experienced and involved in maintaining PTSD (Andrews, Brewin, Rose, & Kirk, 2000; Lee, Scragg, & Turner, 2001; Resick, 2004). Resick (2004) has proposed that the *DSM-V* PTSD criteria be expanded so that emotions beyond fear (i.e., shame, anger, and guilt) are included.

Controversies of PTSD: An Anxiety-Based or Information-Processing Disorder

Resick (2004) also proposed that for the forthcoming *DSM-V* PTSD be moved out of the supraheading of "Anxiety Disorders" and into a new classification of "Stress-Related Disorders" that would include the adjustment disorders, acute stress disorder, traumatic grief, and dissociative disorders. This reclassification would return PTSD, or the study of trauma reactions, back to the broad field of stress research from where it originated. The assumption currently implied by the *DSM-IV* that PTSD is an anxiety disorder does, however, fit with early behavioral theories of PTSD (i.e., Keane et al., 1985) that developed from conditioning and learning principles and were based on Mowrer's (1960) two-factor model of anxiety. These behavioral theories lead to the development of treatments for PTSD such as exposure, flooding, and implosion where the main aim is to alleviate *fear* by preventing avoidance of the feared stimuli so that habituation and extinction take place. While exposure treatments are effective in reducing fear and anxiety, there is no compelling evidence that nonfear emotions (i.e., shame, guilt, and anger) habituate to exposure alone when they are predominant (Grunert, Weis, Smucker, & Christianson, 2007). In fact, Grunert et al. (2007) demonstrated that when nonfear emotions are associated predominantly with PTSD, treatment based on habituation (i.e., prolonged exposure) fails to lead to improvement and recovery from PTSD symptoms.

Alternatively, theorists have argued that the core issue in the development and maintenance of PTSD is not anxiety or fear reactions that stem from experiencing a criterion A(1) event. Rather, it is argued that PTSD is an information-processing disorder whereby it is the way that *memories* of the traumatic event are processed, integrated, and represented that is the central mechanism that creates anxiety states and drives the PTSD syndrome (van der Kolk, 1994; van der Kolk & McFarlane, 1996). Theorists also propose that processing the memory of the event in a way that resolution of meaning takes place is central to the therapeutic recovery process from PTSD (Horowitz, 1976; Janoff-Bulman, 1992). Recent theories of PTSD support the idea that PTSD is an information-processing disorder. It is beyond the scope of this article to discuss all the psychological theories relating to PTSD (for an overview of PTSD theories, refer to Brewin &

Holmes, 2003); however, at present, the most predominant psychological theories of PTSD are emotional processing theory (Foa & Rothbaum, 1998), Ehlers and Clark's (2000) cognitive model of PTSD, and dual-representation theory (Brewin, Dalgleish, & Joseph, 1996). All theories can be referred to as information-processing theories of PTSD, as they initially draw on classic cognitive network models of memory and provide accounts of how trauma-related information is represented in "fear networks" (Foa & Kozak, 1986) within the cognitive system and is integrated with existing semantic memory networks. A predominant theory that has also aided in our understanding of PTSD is the adaptive information-processing (AIP) model (Shapiro, 2001). This theory is based on the assumption that PTSD is an information-processing disorder, and it is the theory on which EMDR is based. The AIP model has guided EMDR clinical practice for the treatment of PTSD whereby the processing of traumatic memories is seen as the key element in treatment.

Information-processing theories of PTSD have facilitated our understanding of EMDR and the processes involved in recovering from PTSD. Emotional processing theory (Foa & Rothbaum, 1998) aids in our understanding of EMDR, as it expands on Foa and Kozak's (1986) information-processing, "fear network" theory to account for beliefs and appraisals that exist prior to or that occur during and after trauma and how they can reinforce schemas and maintain PTSD. Ehlers and Clark's (2000) cognitive model provides what is currently considered one of the most detailed accounts of the maintenance and treatment of PTSD. They suggest that PTSD develops and persists when individuals process the trauma in a way that leads to a sense of threat. Treatment involves the elaboration of the trauma memory to increase associations and facilitate adaptive processing (i.e., processing the meaning of the event) and integrating it into one's autobiographical memory base. Dual representation theory raises questions about how EMDR may work, as, unlike other theories of PTSD, it proposes that there are two memory systems: conscious verbally accessible memories (VAMs), which are autobiographical memories that can be deliberately retrieved, and unconscious situationally accessible memories (SAMs), which are triggered by reminders of the trauma. PTSD results when VAM representations of the trauma event fail to form, and only SAMs of the trauma are experienced as intrusive images that are triggered by cues and are accompanied by emotional and/or physiological arousal experienced during the traumatic event. Treatment involves the construction or transfer of detailed consciously accessible memories (VAM) that previously existed only in an unintegrated form in the SAM system. All three psychological theories of PTSD propose that PTSD develops when *memories* of the traumatic event are poorly elaborated, are often difficult to verbalize, and are unintegrated with preexisting memory networks.

In summary, over the past 30 years, theories of PTSD have evolved from traditional behavioral accounts of PTSD that were based on conditioning and learning principles and models of anxiety to current theories of PTSD that have incorporated information-processing models. These information-processing theories of PTSD emphasize the idea that unprocessed trauma memories leads to the development and maintenance of PTSD (Brewin & Holmes, 2003). Current theories also provide more comprehensive explanations of a wide range of complex processes involved in the development, maintenance, and recovery from PTSD. For example, they account for a range of emotions associated with PTSD beyond fear and consider cognitive elements, such as incorporating the meaning of the event into preexisting schema networks. Currently, meta-analyses that have examined the efficacy of treatments for PTSD indicate that trauma-focused exposure therapies, based on the idea that PTSD is an anxiety disorder, are effective (Bisson et al., 2007; van Etten & Taylor, 1998). However, as traditional theories of PTSD and exposure treatments have developed, simultaneously EMDR has evolved, and meta-analyses reveal it to be equally effective as exposure-based therapies for the treatment of PTSD. Although EMDR treatment of PTSD is based on the assumption that PTSD is an information-processing disorder, EMDR has evolved from AIP theory (Shapiro, 2001), which is an information-processing theory that is separate yet comparative with those incorporated into current theories of PTSD. The remainder of this article examines the development of EMDR and corresponding research. The AIP model is then discussed and evidence for the model summarized. The article concludes by highlighting questions raised about PTSD and its treatment when the AIP model is compared to other information based theories of PTSD.

EMDR Treatment of Adult PTSD: History of Research and Current Status

EMD was initially developed by Shapiro (1989) to resolve trauma symptoms by desensitizing traumatic memories. EMD evolved to become EMDR (Shapiro, 1991, 1995, 2001), which is an integrative, comprehensive treatment approach that contains many elements of

effective psychodynamic, cognitive-behavioral, experiential, interpersonal, and physiological therapies. Marquis and Marquis (in press) present a historical account of EMDR, but what is interesting to note is that the challenges EMDR has faced since its conception have, in many ways, been similar to those that arose with the inception of PTSD. EMDR received divergent reactions from scientists and professionals; it challenged existing ideas about how trauma was being treated, it was not initially accepted, and it was criticized because of the impression that it was being proposed as a one-session cure for PTSD rather than a structured eight-phase treatment approach that aims to access and process past, present, and future aspects of dysfunctionally stored memories that form the basis of current pathology. EMDR created a vocal group of concerned skeptics who influenced the progression of the field as it drove proponents to produce exceptional amounts of evidence to justify claims. Just as PTSD has been the most researched anxiety disorder in the past 20 years (Boschen, 2008), EMDR is one of its most extensively researched treatments.

First and Second Phases of Research

The history of research into EMDR for the treatment of adult PTSD can be divided into three main phases: (a) demonstrating EMDR's effectiveness in treating PTSD, (b) demonstrating EMDR's effectiveness against other trauma-focused treatments for PTSD, and (c) focusing on understanding the underlying mechanisms of EMDR. In the early phase of EMDR research (1989–1998), strong evidence arose demonstrating that EMDR was consistently superior to wait-list or delayed treatment controls. As seen in Table 1, average effect sizes[1] for EMDR and control conditions pre- to posttreatment are 1.19 and 0.07, respectively. Effect size is a measure of the change in mean scores between conditions after controlling for the variance in each condition. The effect size of 0.07 for control conditions is below what is considered a small effect, which is generally between 0.2 and 0.3. Around 0.5 is referred to as a medium effect, and the effect size of 1.19 for EMDR is considered a large effect, which is generally anything above 0.8.

In the second phase of EMDR research, beginning a decade after Shapiro's (1989) seminal publication, four randomized controlled trials examined the effectiveness of EMDR compared to nonspecific therapies for PTSD, and again EMDR was consistently more effective in treating adult PTSD than other nonspecific treatments. As shown in Table 2, average effect sizes for EMDR compared to nonspecific treatments are 1.61 and 0.88, respectively. In this phase of research,

TABLE 1. RCTs of EMDR Versus Wait-List Control for the Treatment of Adult PTSD

						Effect Size	
Study	Conditions	Population Type	*N*	No. of Sessions	% Dropout	Pretreatment to Posttreatment	Pretreatment to Follow-Up
Boudewyns and Hyer (1996)	EMDR	Combat veterans with PTSD	21	5–7[a]	[b]	0.67	—
	Standard care		22	8 group		0.38	—
Devilly et al. (1998)	EMDR	Combat veterans with PTSD	13	2	32	0.37	0.11
	Psychiatric support		6	—	63	–0.01	0.12
Hogeberg et al. (2007)	EMDR	Occupation-based PTSD	12	5	8	0.93	—
	WL control		9	—	18	0.35	—
Jensen (1994)	EMDR	Combat veterans with PTSD	13	3 in 10 days	—	–0.50	—
	WL control		12	—	—	–1.01	—
Rothbaum (1997)	EMDR	Female rape victims with PTSD	10	3	9	2.43	3.19
	WL control		8	3	20	0.51	—
Wilson et al. (1995, 1997)	EMDR	Trauma memory, 46% with PTSD	37	3	8	1.61	1.63
	WL control		37	—	8	—	—

Note. Effect sizes pre- to posttreatment and pretreatment to follow-up were calculated for the PTSD measures used in each study using Cohen's *d* statistic and were based on completer rather than end-point or intent-to-treat analyses. *N* = number of participants who completed therapy at posttreatment.

[a]This condition also received eight sessions of the standard group treatment program offered. [b]Four participants chose not to complete the study; however, which condition they were in was not specified.

TABLE 2. RCTs of EMDR Versus Other Nonspecific Treatments Used to Treat Adult PTSD

Study	Conditions	Population Type	*N*	No. of Sessions	% Dropout	Effect Size: Pretreatment to Posttreatment	Effect Size: Pretreatment to Follow-Up
Carlson et al. (1998)	EMDR	Combat veterans with PTSD	10	12	0	1.44	2.31
	Relaxation		13	12	7	0.60	0.75
	Routine care / WL		12	6	0	0.63	—
Edmond and Rubin (2004); Edmond (1999)	EMDR	Trauma memory of sexual abuse	20	6	0	1.52	2.41
	Routine care		20	6	0	1.60	0.60
	WL control		19		0	0.50	0.31
Marcus (1997); Marcus et al. (2004)	EMDR	Civilians with PTSD	33	Unlimited	0	2.03	2.69
	Standard care		33	Unlimited	3	0.57	1.16
Scheck et al. (1998)	EMDR	Trauma memory, 77% with PTSD	60	2 2	30	1.45	N/A
	Active listening					1.02	

nine randomized controlled trials also compared the effectiveness of EMDR to other trauma-focused therapies, such as cognitive-behavioral therapy (Devilly & Spence, 1999), exposure (Ironson, Freund, Strauss, & Williams, 2002; Rogers et al., 1999; Rothbaum, Astin, & Marsteller, 2005; Taylor et al., 2003), and exposure with cognitive restructuring (Power et al., 2002) or stress inoculation (Lee, Gavriel, Drummond, Richards, & Greenwald, 2002). Average effect sizes for EMDR and other trauma-focused treatments are similar with the change from pre- to posttreatment being 1.74 and 1.52, respectively (see Table 3). With the exception of Devilly and Spence (1999) and Taylor et al. (2003), EMDR has been found to be roughly equal in its effectiveness with exposure-based therapies. However, others have found a slight trend toward greater efficiency for EMDR over exposure therapy (Ironson et al., 2002; Lee et al., 2002; Power et al., 2002). Compared to exposure therapy, EMDR was found to result in a more rapid reduction of symptoms (Ironson et al., 2002; Rogers et al., 1999), was reported to require fewer treatment sessions (van Etten & Taylor, 1998), and resulted in fewer dropouts (Ironson et al., 2002; Rothbaum et al., 2005; Taylor et al., 2003). The first meta-analysis to examine the comparative effectiveness of EMDR to exposure-based therapies found that randomized controlled trials did not reveal any significant difference in effect (van Etten & Taylor, 1998), yet the authors did note that EMDR required fewer sessions. Subsequent meta-analyses over the past 10 years have also found equivalent effect sizes for EMDR and exposure therapy for adult PTSD (Bisson et al., 2007; Bradley et al., 2005; Davidson & Parker, 2001). However, Rothbaum et al. (2005) has noted that EMDR achieved its results without the use of the 30 to 60 hours of homework often used in exposure therapies. As yet, only one randomized controlled trial (Ironson et al., 2002) has compared the effectiveness of EMDR and exposure therapy and controlled for treatment time and the amount of homework between sessions. Although homework is not part of the EMDR protocol, all participants were required to do in vivo exposure homework. The authors found that EMDR led to a more rapid reduction in symptoms, as 7 out of 10 EMDR participants had a 70% reduction in PTSD symptoms after 3 sessions, compared to only 2 out of 12 in the prolonged exposure group. However, further studies comparing EMDR to exposure therapy that control for treatment time and homework are required.

EMDR Research: Variability in Methodological Strengths and Limitations in Knowledge

Although effect sizes are equivalent between traditional exposure-based treatments and EMDR for the treatment of PTSD, it is worth noting that there are varying degrees of methodological strengths between the nine randomized controlled trails that have examined their comparative effectiveness (see Table 3). For example Devilly, Spence, and Rapee (1998) did not meet basic requirements for randomization, the majority of participants were treated by the same

therapist, and the trained assessor was not blind or independent. Lee et al. (2002) also used a nonblind assessor who was not independent, and Taylor et al. (2003) failed to discuss intent to treat analysis. This being especially significant because of the high dropout rate in the traditional exposure condition (32%) compared to the EMDR condition (21%). However, a number of randomized controlled trials that have examined the effectiveness of EMDR have very few or no major limitations, for example, Carlson, Chemtob, Rusnak, Hedlund, and Muraoka (1998), Rothbaum et al. (2005), and van der Kolk et al. (2007), all of whom found statistically significant improvement in treatment groups with large effect sizes for EMDR. The average effect size for these three studies are 1.89 (pre- to posttreatment), and 2.10 (pretreatment to follow-up). Maxfield and Hyer (2002) have examined the relationship between effect size and methodology. Interestingly, they found that a significant relationship exists between effect size and treatment fidelity, and the more rigorous the methodology, the greater the effect size for EMDR.

It is also important to note that although EMDR has demonstrated its efficacy for the treatment of adult PTSD, the majority of randomized controlled trials to date have utilized civilian, single-trauma patient groups, and complex trauma cases are often excluded. A number of studies have, however, focused on combat (i.e., Carlson et al., 1998) and sexual abuse (i.e., Rothbaum et al., 2005) trauma that has led to PTSD. Although more studies are needed to establish the effectiveness of EMDR with these populations, what appears to be evident is that that approximately three sessions of EMDR are necessary for comprehensive

TABLE 3. RCTs of EMDR Versus Trauma-Focused Therapies for the Treatment of Adult PTSD

						Effect Size	
Study	Conditions	Population Type	*N*	No. of Sessions	% Dropout	Pretreatment to Posttreatment	Pretreatment to Follow-Up
Devilly and Spence (1999)	EMDR	Mixed PTSD civilians	11	Up to 8	35	1.56	0.32
	CBT variant (TTP)		12	8	20	2.36	1.26
Ironson et al. (2002)	EMDR	Civilians with PTSD	10	1–3	0	1.53	1.43
	PE		9	1–3	25	2.18	3.03
Lee et al. (2002)	EMDR	Civilians with PTSD	12	7	8	1.87	2.17
	SIT + PE		12	7	8	1.45	1.46
	WL control		29	—	0	0.5	—
Power et al. (2002)	EMDR	Mixed PTSD civilians	27	10	31	2.76	—
	Exposure + CR		21	10	43	1.84	—
	Wait list		24	—	17		—
Rogers et al. (1999)	EMDR	Combat veterans with PTSD	6	1	0	0.85	—
	Exposure		6	1	0	0.2	—
							—
Rothbaum et al. (2005)	EMDR	Rape victims with PTSD	20	9	13	1.91	1.46
	PE		20	9	20	2.02	2.30
	WL control		20		17	0.38	—
Taylor et al. (2003)	EMDR	Mixed PTSD civilians	15	8	21	N/A	1.96
	Exposure therapy		15	8	32		2.42
	Relaxation training		15	8	21		1.89
Vaughan et al. (1994)	EMDR	Mixed PTSD civilians, 78% with PTSD	12	3–5	0	1.35	1.34
	Imaginal Exposure		13	3–5	0	0.65	0.72
	Muscle Relaxation		11	3–5	0	0.57	1.00
	WL control					0.23	—
van der Kolk et al. (2007)	EMDR	Mixed PTSD civilians	24	8	17	2.32	2.54
	Fluoxetine		26	8	13	1.95	1.93
	Pill placebo		26	8	10	1.84	—

treatment of single-trauma PTSD (i.e., Ironson et al., 2002; Marcus, 1997; Marcus, Marquis, & Sakai, 2004; Rothbaum, 1997; Wilson, Becker, & Tinker, 1995, 1997); however, complex, multiple trauma populations require many more sessions for the treatment to be complete and robust (i.e., Carlson et al., 1998; Marcus, 1997; Marcus et al., 2004). Further research is needed to systematically examine the effect that the number and type of traumatic memories the individual has on treatment outcome.

Further research is also required to examine the effect the variable of time since the traumatic event has on the effectiveness of EMDR for treating PTSD. Recently, van der Kolk et al. (2007) conducted a randomized control trial that included both adult PTSD participants with child abuse trauma and adult-onset trauma. What was found was that eight sessions of EMDR was insufficient for those with childhood abuse as their response was less robust than those with adult-onset trauma. Although at 6-month follow-up 89% of the child-onset trauma group lost their PTSD diagnosis, only 33% were asymptomatic, compared to 75% of those with adult-onset trauma. Similarly, in a study by Edmond, Rubin, and Wambach (1999) where adult survivors of childhood sexual abuse showed significant reductions in trauma symptoms after six sessions of EMDR, the authors concluded that although this number of sessions helped alleviate symptoms, longer-term treatment was likely to be needed to adequately address all the issues confronting participants. Research is needed to better determine if lengthier EMDR is a requirement for childhood trauma survivors and whether these patients would also benefit from an extended preparation phase or a combination of treatments (i.e., EMDR combined with pharmacotherapy). Only one randomized trial to date has examined the efficacy of EMDR compared to pharmacological treatment for PTSD (van der Kolk et al., 2007). EMDR was found to be more successful than pharmacotherapy in achieving sustained reductions in PTSD symptoms, but this was primarily for adult-onset trauma survivors. It may be possible that childhood trauma responds to a combination of EMDR and pharmacotherapy, which is common in clinical practice, but the efficacy of this is yet to be examined.

The efficacy of EMDR in the treatment of disorders other than PTSD is less established. Clinicians often use EMDR to treat a variety of presenting problems, such as those that stem from criterion A events that frequently do not meet criterion A for PTSD, such as extramarital affairs (Dattilio, 2004), sexual harassment (Avina & O'Donohue, 2002), and complicated grief (Sprang, 2001). A recent randomized control trial by Cvetek (2008) demonstrated that EMDR is effective for treating participants who experience distress as a result of "small t" incidents that fail to meet criterion A for PTSD. Significant reductions in trauma symptoms were found for EMDR over an active listening control. Cvetek's finding supports those of Wilson et al. (1995, 1997), who found that EMDR was equally effective in decreasing symptoms associated with trauma memories for those who met PTSD diagnostic criteria and those who did not and were instead referred to as "partial PTSD participants." Keeping in mind the potential removal of criterion A in *DSM-V* and the knowledge that the PTSD syndrome can develop without exposure to a criterion A event, the expansion of research into the effectiveness of EMDR for treating "small t" traumas is encouraged.

Given the research described previously, it is not surprising that several independent bodies have rated EMDR in the highest category of effectiveness for the treatment of PTSD. For example, in the International Society of Stress Studies practice guidelines (Foa, Keane, Friedman, & Cohen, 2009), EMDR has recently been ranked as an evidence-based level A treatment for PTSD in adults. EMDR is rated in the highest category of research and support in the clinical practice guidelines of the American Psychiatric Association (2004) and the U.S. Department of Veterans Affairs and Department of Defense (2004). EMDR is also acknowledged as an evidence-based treatment for PTSD by the U.K. National Institute for Clinical Excellence (2005) and the Australian Centre for Posttraumatic Mental Health (2007). In addition, a growing number of international guidelines (i.e., Bleich, Kotler, Kutz, & Shalev, 2002; CREST, 2003; INSERM, 2004) also recommend EMDR for the treatment of adult PTSD.

Third Phase of Research

The third and current phase of research into EMDR is now heavily focused on understanding the underlying mechanisms of effective treatment. At present, as discussed in the next section, what is known is that the eye movements in EMDR do contribute to the therapeutic process, that the processes involved are not the same as those in traditional exposure, and, to date, that the most promising theoretical account of EMDR is the AIP model (Shapiro, 2001).

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 interhemispheric 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 preexisting 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., $SD_{pooled} = \sqrt{[(SD^2_{pre} + SD^2_{post})/2]}$).

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