Preliminary Evidence for the Efficacy of EMDR in Treating Generalized Anxiety Disorder

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This preliminary study sought to evaluate the potential effectiveness of eye movement desensitization and reprocessing (EMDR) as a treatment modality for generalized anxiety disorder (GAD). Using a single-case design with multiple baselines across four subjects, the effectiveness of 15 EMDR sessions was evaluated. Results indicate that subsequent to targeting the experiential contributors to GAD and the current and anticipated situations that caused excessive worry, the scores of anxiety and of excessive worry dropped to levels below diagnostic threshold and in two cases to full remission of GAD symptoms. At both posttreatment and at 2 months follow-up, all four participants no longer presented with GAD diagnosis. In addition, time-series analyses (ARMA) indicate statistically significant improvement on both daily measures of worry and anxiety over the course of the EMDR treatment.

Keywords: efficacy; EMDR; generalized anxiety disorder; single-case design; time-series analyses; treatment

Eye movement desensitization and reprocessing (EMDR) therapy was initially developed by Francine Shapiro (1989, 1995) as a treatment for posttraumatic stress disorder (PTSD). EMDR is a complex and structured psychotherapy methodology that includes aspects of diverse theoretical orientations (e.g., psychodynamic, cognitive-behavioral, humanistic, psychophysiologic). Its distinctive feature is instructing the client to focus on disturbing material while simultaneously attending to an alternate stimulus, namely, eye movements or other forms of bilateral stimulation (Shapiro, 1995, 2002a, 2002b, 2002c; Shapiro & Maxfield, 2002). The eight-phase treatment protocol of EMDR is guided by Shapiro's adaptive information processing (AIP) model (Shapiro, 1995, 2002a), which posits that human beings possess a physiological/neurological system in charge of assimilating and integrating the many aspects of an experience (somatic, cognitive, and emotional). However, under highly stressful situations, such as traumatic events, this information processing system is thought to become unbalanced, thus impeding the integration of the experience into autobiographical memory. When this occurs, the initial perceptions are understood to be stored in their initial form along with any distortions provoked by the high arousal (Shapiro, 2002a; van der Kolk, 2002).

Shapiro (2002c) proposed that these nonintegrated experiences are at the root of various psychological disorders, such as PTSD, as well as forming the basis for certain personality structures. EMDR treatment is thought to reactivate the natural information processing system and to facilitate adaptive resolution of the previously distorted material (Bergmann, 1998, 2000; Shapiro, 2002a; Stickgold, 2002; van der Kolk, 2002). Standardized protocols are formulated to address the past experiential contributors to the presenting dysfunction, the current triggers that presently reactivate the material related to the past event, and related future situations (Maxfield, 2002; Shapiro, 1995, 2002c, 2004).

The efficacy of EMDR in treating PTSD has been clearly demonstrated in 16 randomized controlled trials. Results generally indicate that (a) EMDR is more effective that no treatment (e.g., Wilson, Becker, &

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Tinker, 1995, 1997), (b) EMDR is more effective than nonstandardized treatment approaches (e.g., Marcus, Marquis, & Sakai, 1997, 2004), (c) EMDR is equally effective as behavioral or cognitive-behavioral methods of treatment (e.g., Rothbaum, Astin, & Marsteller, 2005), and (d) EMDR is more effective than pharmacology alone (van der Kolk et al., 2007). Results of five meta-analyses corroborate these findings (Bisson et al., 2007; Bradley, Green, Russ, Dutra, & Westen, 2005; Davidson & Parker, 2001; Maxfield & Hyer, 2002; Van Etten & Taylor, 1998). Maxfield and Hyer (2002) further showed that treatment fidelity plays an important role in EMDR outcome effect sizes.

Although EMDR was initially developed for treating PTSD, some studies have indicated its potential usefulness to address other disorders and/or psychological conditions. They suggest that EMDR may be useful in treating chronic pain (e.g., Grant & Threflo, 2002; Schneider, Hofmann, Rost, & Shapiro, 2007), body image disturbances (e.g., McGoldrick, Begum, & Brown, this issue), specific phobias with a traumatic origin (e.g., de Jongh, van den Oord, & ten Broeke, 2002), "internalized shame" (Balcom, Call, & Pearlman, 2000), affect dysregulation (Korn & Leeds, 2002), borderline personality disorder (Brown & Shapiro, 2006), and PTSD-related anger and guilt (Stapleton, Taylor, & Asmundson, 2006). So far, no study has addressed the use of EMDR for treating generalized anxiety disorder.

Generalized Anxiety Disorder

Generalized anxiety disorder (GAD) was first introduced in the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.; *DSM-III-R;* American Psychiatric Association [APA], 1987). With the *DSM-IV* (APA, 1994), GAD is now recognized as a disorder in itself. It is characterized by excessive and difficult-to-control worry occurring more than every other day for more than 6 months. It is accompanied by three of the six following somatic symptoms of anxiety: agitation, tiredness, trouble concentrating, irritability, muscle tension, or sleep disturbance (APA, 1994; Ladouceur & Dugas, 1999; Mennin, Heimberg, & Turk, 2004). Studies have indicated that the prevalence of GAD is around 4% to 6% in the general population (APA, 1994; Bruce, Machan, Dyck, & Keller, 2001; Ladouceur & Dugas, 1999; Stanley & Novy, 2000).

Since the 1990s, studies have investigated the efficacy of cognitive-behavioral therapy (CBT) in treating GAD (Borkovec & Costello, 1993; Butler, Fennell, Robson, & Gelder, 1991), and two meta-analyses (Deacon & Abramowitz, 2004; Western & Morrison, 2001) have investigated the overall results of research done in the 1990s. Generally, it was found that CBT seems effective in treating GAD but that studies presented some methodological flaws. These included too many exclusion criteria, thus limiting the ability to generalize the findings, and a rather vague definition of CBT, ranging from exposition, cognitive restructuring, and relaxation training to combinations. Only one treatment study actually targeted the main symptom of GAD, worry (Borkovec & Costello, 1993).

Some authors have proposed that worry is mainly a cognitive phenomenon with negative verbal content (Borkovec, Ray, & Stober, 1998). Worry is thus conceptualized as a way to avoid the negative images related to the perceived threatening situation and its accompanying emotional state. It has then been suggested that since worry is a form of cognitive avoidance, it prevents the emotional processing required to surmount the anxiety or fear pertaining to the anticipated situation (Borkovec, Alcaine, & Behar, 2004; Dugas, Gagnon, Ladouceur, & Freeston, 1998; Roemer, Salters, Raffa, & Orsillo, 2005).

The late 1990s also saw the emergence of a cognitive-behavioral model of GAD that has been validated empirically and has shown clinical usefulness (Dugas, Buhr, & Ladouceur, 2004; Dugas, Gagnon, et al., 1998; Dugas, Marchand, & Ladouceur, 2005; Ladouceur & Dugas, 1999). This model proposes four main variables involved in maintaining the excessive worry in GAD:

1. Intolerance to uncertainty (Dugas, Freeston, & Ladouceur, 1997; Grenier & Ladouceur, 2004; Ladouceur, Gosselin, & Dugas, 2000)
2. Poor emotional orientation to problem-solving (Dugas et al., 1997)
3. Dysfunctional beliefs about the usefulness of worrying (Laberge, Dugas, & Ladouceur, 2000)
4. Cognitive avoidance, as described previously (Borkovec et al., 1998, 2004; Roemer et al., 2005)

This model has proved to be effective in individual treatment (Ladouceur et al., 2000; Dugas, Ladouceur, Brillon, Savard, & Turcotte, 2002) and in group treatment (Dugas et al., 2003a, 2003b).

When it comes to onset, it would appear that GAD generally develops before adulthood, as sufferers report "having always been a worrier" (Hudson & Rapee, 2004; Rapee, 2001). As mentioned by these authors, many variables interact together in developing and maintaining GAD. Their model suggest the presence of (a) a genetic predisposition to anxiety disorders, (b) a temperament style increasing

vulnerability to anxiety, and (c) the interaction of environmental factors leading to an anxiety disorder. In addition, Chorpita and Barlow (1998) have suggested that it is not only the occurrence of stressful or traumatic events but also the individual's belief that he or she does not possess the necessary resources (internal and/or external) to face the situations.

A number of environmental or experiential contributors in the etiology of GAD have been proposed in the literature: (a) highly or chronically stressful situations, including a traumatic event (Gosselin & Laberge, 2003; Hudson & Rapee, 2004; Roemer, Molina, Litz, & Borkovec, 1997); (b) an overprotective or overcontrolling parental style (Chorpita & Barlow, 1998; Rapee, 2001); (c) being encouraged in an avoidant style by parents or significant adults (Hudson & Rapee, 2004; Rapee, 2001); (d) modeling from a parent suffering from an anxiety disorder (Gosselin & Laberge, 2003; Hudson & Rapee, 2004; Manassis & Hood, 1998; Rapee, 2001); (e) the loss of one parent during childhood or adolescence through either death or divorce (Gosselin & Laberge, 2003; Hudson & Rapee, 2004); and (f) the inversion of parent–child role during childhood or adolescence (Gosselin & Laberge, 2003). When it comes to cognitive styles, Breinholtz, Johansson, and Ost (1999) have suggested that GAD sufferers tend to have more negative beliefs than others in the areas of interpersonal relationships, self-confidence/competence, and various worries.

Given that etiological models of GAD suggest the role of experiential contributors in the development of this disorder and given that its major symptom, excessive worry, seems to impede the processing of the emotional distress related to the cause or trigger of excessive worrying, it seemed logical to see a parallel with Shapiro's AIP model, which proposes that unprocessed life experiences have an impact on personality development and psychological disorders. Nevertheless, no one has investigated the potential usefulness of EMDR in treating GAD and its main symptom of excessive worry. Only one study explored the usefulness of EMDR in targeting the cognitive intrusions related to stressful daily life events (Lytle, Hazlett-Stevens, & Borkovec, 2002). However, it studied the impact of a single EMDR session with a nonclinical sample.

Therefore, the current study sought to investigate the potential efficacy of EMDR in treating GAD by targeting potential past experiential contributors, current triggers of worry, and the possible negative beliefs accompanying these targets. In conducting this study, important considerations were taken into account so as to increase the validity of this single case design: (a) fidelity to the EMDR procedure, as it has been shown that treatment fidelity is associated to stronger effect sizes; (b) the level of experience of the clinician offering the EMDR treatment; and (c) an adequate number of sessions offered so as to increase therapeutic value (Maxfield & Hyer, 2002). The general hypothesis was that EMDR can help to significantly reduce excessive worry and its accompanying anxiety symptoms in GAD sufferers.

Method

This study used a single-case design with multiple baselines across subjects in order to investigate the efficacy of EMDR in treating GAD. This type of design allows maximization of the internal validity by introducing the EMDR treatment at different moments in time for various individuals. This minimizes possible effects of subject maturation or historical factors (Bouvard & Cottraux, 2002; Rivard & Bouchard, 2005; Rubin, 1997). The baseline period varying from 3 to 5 weeks served as a control condition.

Participants

Following institutional and ethical approvals, five participants were recruited in an outpatient psychiatric clinic where the first author worked as a clinical psychologist. Potential participants were referred through colleagues (psychiatrist, psychologists, physician, psychiatric nurses, or social workers) in the same clinic. Potential participants were required to have a principal diagnosis of GAD, with or without a comorbid (axis I or II) disorder. Exclusion criteria were the presence of serious suicidal ideation, active substance abuse, current psychotic features, and/or bipolar disorder. These criteria are generally used in other studies on GAD (Dugas et al., 2003a, 2003b; Ladouceur et al., 2000).

Three participants were already receiving mental health services before entering EMDR treatment. It was agreed either that they would cease receiving these services (one case) or that their therapist would not address any issues related to their GAD (two cases). The five participants selected for the study were randomly assigned to three conditions: 3 weeks of baseline + up to 15 sessions of EMDR, 4 weeks of baseline + up to 15 EMDR sessions, or 5 weeks of baseline + up to 15 EMDR sessions. It should be noted that in the course of the study, one participant had to withdraw because of a car accident resulting in serious injuries. She had just begun the EMDR treatment, receiving one session of EMDR.

Participants were initially screened with French version of the Worry and Anxiety Questionnaire, the *Questionnaire sur les inquietudes et l'anxiété* (QIA), to assess the presence of GAD. If it indicated the presence of GAD, the first author administered the Structured Clinical Interview for DSM-IV (SCID-I & II) to formally assess (a) the diagnosis of GAD, (b) to assess the presence of any comorbid axis I or axis II disorders, and (c) to eliminate the presence of bipolar disorder or psychotic feature.

Measures

Diagnostic and Screening Measures

Participants were administered French versions of the Structured Clinical Interview DSM-IV-Tr Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 2001) and the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II; First, Gibbon, Spitzer, Williams, & Benjamin; 1997) to confirm the presence of GAD and to specify any other concurrent axis I or II disorder. The SCID-I was administered again at the end of treatment and at the 2-month follow-up. The Dissociative Experience Scale (DES-II; Bernstein-Carlson & Putnam, 1993) was also administered to assess the presence of pathological dissociation. This precaution is part of the standard EMDR protocol (Shapiro, 2004; Korn & Spinazzola, 2001). Its authors have indicated that the DES-II presents a good test–retest reliability ($r = 0.79$–0.86) and good internal consistency ($r = 0.83$–0.83).

Daily Measure of Worry and Anxiety. For the purpose of this study, a daily measure of worry and anxiety was designed, consisting of daily ratings of worry and anxiety on a 0% to 100% scale. A previous study had shown the usefulness of such a daily measure (Dupuy, Beaudoin, Rhéaume, Ladouceur, & Dugas, 2001) indicating that daily levels of worry were significantly higher in GAD than in non-GAD participants and significantly correlated with the Penn State Worry Questionnaire. The two questions on the daily measure were (a) "To what extent did you worry excessively in the course of the day?" and (b) "To what extent did you feel anxious today?"

Weekly Measure. A validated French version of the Penn State Worry Questionnaire was used. The *Questionnaire sur les inquiétudes de Penn State* (QIPS; Gosselin, Dugas, Ladouceur, & Freeston, 2001) is a 16-item questionnaire that evaluates the tendency to worry. It shows a good test–retest reliability ($r = 0.86$) and good internal consistency ($r = 0.82$). It has been shown to distinguish between GAD and non-GAD subjects (Ladouceur et al., 1999).

Pre-/Posttreatment and Follow-Up. Four self-report questionnaires were administered at three different times: pretreatment, posttreatment, and at the 2-month follow-up. The first one was a French version of the Worry and Anxiety Questionnaire. The QIA (Dugas et al., 2001) is a self-report measure that contains 11 items corresponding to the *DSM-IV* diagnostic criteria of GAD. Its authors suggest that it possesses a good test–retest reliability, that it successfully distinguishes GAD patients from nonclinical subjects, and that it is a sensitive measure of worry.

A second measure was the French version of the Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1994, cited in Bouvard & Cottraux, 2002). The French version of the initial BDI (Bourque & Beaudette, 1982) has good internal consistency ($r = 0.92$) and good test–retest reliability ($r = 0.062$). This measure was used, as it has been shown that depression is often present with GAD (Butler et al., 1991; Dugas et al., 1998).

The third measure was the French version of the Intolerance to Uncertainty Questionnaire. The *Questionnaire d'intolérance à l'incertitude* (QII; Freeston, Rhéaume, Letarte, Dugas, & Ladouceur, 1994) is a 27-item questionnaire. It possesses good internal consistency ($r = 0.91$) and good test–retest reliability ($r = 0.78$). It was added, as intolerance to uncertainty has been shown to be a central feature of GAD, as previously mentioned.

The fourth and final self-report measure was the *Questionnaire d'évitement cognitif* (Cognitive Avoidance Questionnaire) (QEC; Gosselin et al., 2002). This 25-item scale measures the tendency to resort to cognitive avoidance, yet another associated feature of GAD. It possesses both good internal consistency ($\alpha = 0.92$–0.95) and good test–retest reliability ($r = 0.81$).

Treatment

The therapist was the first author. At the time of the study, he was a master's-level licensed psychologist with more than 10 years of experience in the field and a doctoral-level student. He was an EMDRIA-certified therapist and an EMDRIA-approved consultant in training. EMDR treatment followed the protocol described in Shapiro's textbook (Shapiro, 1995, 2001) and in the EMDR Institute training manual (Shapiro, 2004), which was manualized for research purposes (Korn & Spinazzola, 2001). In accordance with standard EMDR protocols, past events believed to be

experiential contributors to current GAD were first treated, followed by present and future situations triggering excessive worry. Participants received up to 15 EMDR sessions. That number was determined on the basis of the number of sessions provided in various research studies that investigated the efficacy of CBT in treating GAD (Borkovec & Costello, 1993; Dugas et al., 2003a, 2003b; Ladouceur et al., 2000) and in consideration of Maxfield and Hyer's (2002) recommendations pertaining to a sufficient number of EMDR sessions in more complex situations.

Target Selection for EMDR Processing

Participants were educated about the various environmental or experiential contributors in the etiology of GAD as proposed in the literature. They were also presented with the model of GAD (Dugas et al., 1998, 2004) that suggests the role of the four main variables involved in maintaining the excessive worry in GAD: intolerance to uncertainty, poor emotional orientation to problem solving, dysfunctional beliefs about the usefulness of worrying, and cognitive avoidance. With this in mind, they were asked to identify past events or situations that may have played a role in fostering their anxious disorder and/or where they felt uncomfortable levels of uncertainty. They were then asked to rate their current level of discomfort when thinking about those events. Those past events with the highest levels of distress were identified as targets for EMDR. Participants were then asked to identify current situations or potential future situations that activated their excessive worry. Again, those situations with the highest self-rated level of distress were used as "present" and "future" targets for EMDR. Targets were then treated with EMDR in chronological order, from the earliest memory to the current triggers of worry to the possible future events also triggering worry.

Results

Participant 1

Participant 1 was a woman in her early 30s. She was referred to the study through a social worker. The initial SCID interview revealed that she presented only with a diagnosis of GAD and no evidence of an axis II disorder. At the time of her participation in this study, she had not received prior treatment for her GAD and was not taking medication. She was assigned to a 3-week baseline and treatment was terminated after 12 sessions of EMDR since her symptoms had subsided after that number of sessions.

Different targets from the past were identified. They were related to themes (negative cognitions) of being alone and/or not being competent or good enough, such as being all alone in a difficult situation as a child and a difficult situation at work a number of years ago. Current triggers of worry were focused on her youngest child, fearing that some accident may happen to him when he plays in the backyard. Potential future situations that triggered her excessive worry were the fear that GAD would resurface or, again, fearing that harm would come to family members. In both present and potential triggers of worry, the negative cognitions revolved around not being able to handle it or believing in not being a good mother/wife if she does not worry.

When assessed at both posttreatment and follow-up, participant 1 no longer suffered from GAD and did not present with any residual symptoms. Results were similar on both the clinician-administered SCID-I and on the self-report measures.

Participant 2

Participant 2 was a man in his early 40s. He was referred by his treating psychologist. The initial SCID interview indicated that he suffered from GAD as well as social anxiety disorder and major depressive disorder. On axis II, he presented many traits of avoidant personality disorder. During the EMDR treatment, he continued to meet with his treating psychologist with the understanding that they would not address/treat his GAD. He was assigned to a 4-week baseline and received all 15 sessions of EMDR.

Participant 2 identified past situations that still bothered him today: a period of sexual abuse during his teen years, suffering from a heart attack in his early 30s, and experiences of being criticized. These events activated negative beliefs around being at fault and not being able to set limits (sexual abuse) and about his life being over (heart attack) and being "incapable" (being criticized). Present triggers of his worry revolved around taking steps to reintegrate a course he was interested in and the accompanying negative cognition of "not being competent enough" and of being "unable to face change and new situations." Potential future triggers of worry focused on not feeling competent enough to handle changes and unpredictable situations.

At posttreatment, Participant 2 no longer met diagnostic criteria for GAD, major depression, and social anxiety. However, he identified some residual symptoms for all three disorders. At the 2-month follow-up, diagnostic criteria were met for social anxiety but not

for GAD and major depressive disorder, although he still presented with a few residual symptoms of these disorders.

Participant 3

Participant 3 was a woman in her mid-40s. In the past, she had participated in a study on the treatment of panic disorder with agoraphobia and had been treated successfully. Her current initial SCID interview indicated that she suffered from GAD as well as major depressive disorder and specific phobia (snakes). She did not present any distinctive features of a particular axis II disorder. Physically, she was also suffering from multiple sclerosis and was therefore living with chronic pain. She was assigned to a 3-week baseline and received all 15 sessions of EMDR. She had been referred by her social worker, who had been seeing her in the context of helping her adapt to her illness. She continued to see her over the course of the EMDR treatment.

Again, after having been educated in the etiological model of GAD and about the four variables involved in maintaining excessive worry, participant 3 identified scenes from her past related to feelings and negative beliefs about "being useless," "not being able to do anything," or "being helpless," such as a witnessing a severe car accident as a child and a situation where her father was physically violent toward one of her siblings. Present triggers of her worry involved a conflictual situation within her family, her illness, and her finances. These were related to negative beliefs around "not being able to handle it" and "being incompetent." Targets concerning the future were also related around potential family conflicts along with negative beliefs around her sense of "incompetence" and of "not being able to handle it."

The results for participant 3 at both posttreatment and 2-month follow-up indicated that her symptom pattern no longer met criteria for either GAD or major depressive disorder. She did have some residual symptoms of depression.

Participant 4

Participant 4 was a man in his late 40s. He was referred by his social worker. At the time of beginning EMDR treatment, he chose to stop his meetings with her. The initial SCID interview revealed that he suffered from GAD. Although he presented with certain symptoms of major depression, these were insufficient for a diagnosis. On axis II, he presented substantial features of obsessive-compulsive personality disorder. He was assigned to a 5-week baseline and received all 15 EMDR sessions for his GAD.

Targets from the past focused on moments of verbal abuse and intense expressions of anger by his father or excessive demands made by his anxious mother when he was a child and adolescent. Another past situation involved a verbal aggression in the workplace a number of years previously. These situations were related to negative beliefs about "not being good enough," "not being allowed to express or have emotions," and "not being able to count on others." Various situations from his daily present life activated the same negative beliefs about himself and his excessive worrying and the fear of having to face similar situations in the future.

At posttreatment evaluation, participant 4 no longer met diagnostic criteria for GAD and reported a few residual symptoms. At follow-up, he had no residual symptoms of GAD. However, at posttreatment evaluation, he did meet criteria for major depression. At the 2-month follow-up, the major depressive disorder was in remission. It should be mentioned that 10 days or so prior to his follow-up interview, his physician had begun an antidepressant treatment, venlafaxine (Effexor).

Intervention Time-Series Analyses

Since this study is based on a single-case design, time-series analyses (also referred to as intervention timesseries analyses, or ARMA testing; Box & Jenkins, 1970; Wei, 1990) were used as the primary tool to test the statistical significance of the EMDR intervention. Time-series analyses are known to be more sensitive and robust than a simple visual analysis of the impact on the baseline level of introducing the intervention (Kazdin, 1982, 1984; Matyas & Greenwood, 1990). With the possibility to test changes in level and slope following the introduction of the intervention, time-series analyses are more appropriate than typical *t* tests and analyses of variance. The later are not adequate for several reasons, including the small number of participants and the lack of independence of the data recorded over time (autocorrelation). The time-series analyses were performed according to the procedures outlined by Box and Jenkins using the SCA software, version 7.1a (Scientific Computing Associates, 2005). The procedures were performed independently for each participant and can be summarized as follows: (a) finding an adequate ARMA model that describes the baseline data; (b) building the ARMA model over the entire series and including a function that describes either an abrupt change in *level* when the treatment is introduced or a gradual change, referred to as a *slope,* following the introduction of the treatment;

(c) testing the statistical significance of the function (change in level or in slope); and (d) confirming that the revised ARMA model is adequate.

Visual Inspection and Time-Series Analyses

Daily measures of worry and anxiety are reported in Figures 1 and 2. The moment that the intervention was introduced is indicated with a vertical line crossing each graph. A visual analysis clearly suggests that introducing the intervention had an immediate impact on worry and anxiety for participant 1 (reflecting a *change in level*) and a gradual impact for participant 3 (reflecting a *change in slope*). For participants 2 and 4, the results are harder to interpret visually.

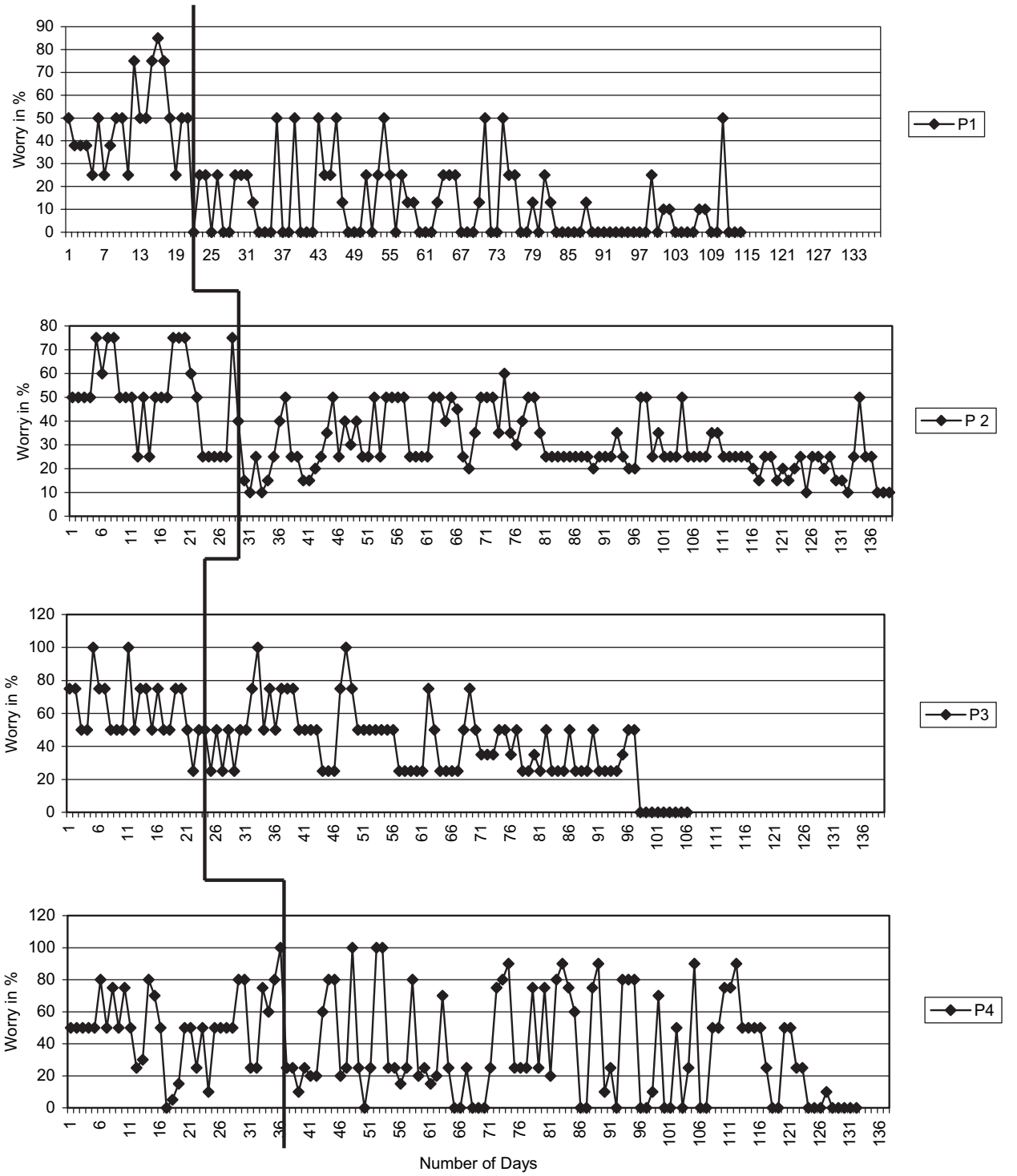

FIGURE 1. Daily levels of worry for all participants.

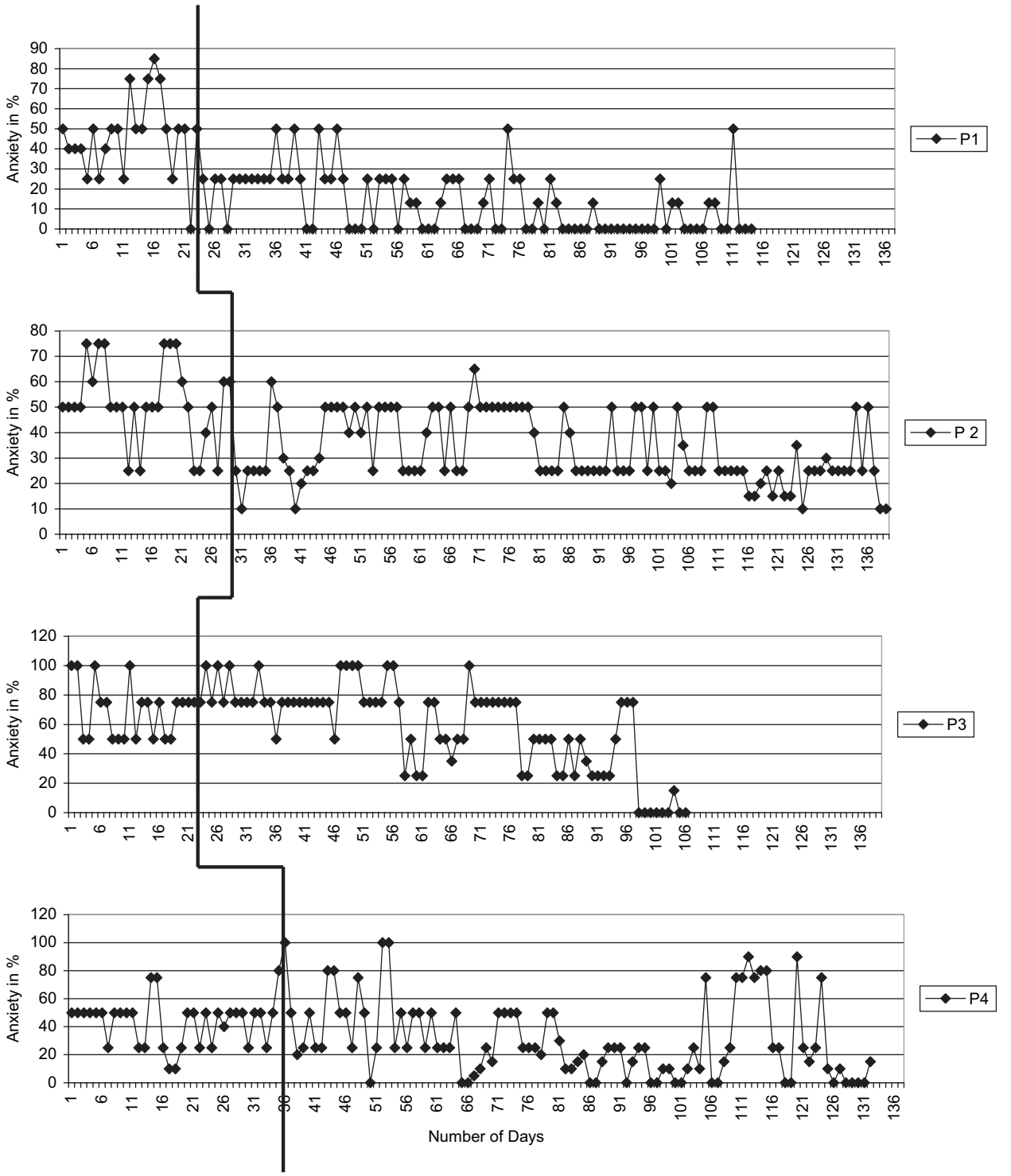

FIGURE 2. Daily levels of anxiety for all four participants.

Results of the statistical time-series analyses provide a clearer picture. Table 1 summarizes results of the time-series analyses for all four participants on both daily worry and anxiety levels, including residual standard errors, the order of the ARMA model, and the *t* value for the function describing the impact of introducing the treatment.

For all four participants, the intervention had a statistically significant impact either in terms of an immediate *change in level* of worry and anxiety or via a progressive change in worry and anxiety over time (*change in slope*). As can be seen for participant 1 (pure GAD), results indicated a statistically significant change in the mean levels of worry ($t = -1.78$, $p = .05$) and anxiety ($t = -2.98$,

TABLE 1. Summary of Results for the Time-Series Analyses Performed on the Level of Worry and Level of Anxiety for All Four Participants

Variable and Participant	Order of the Final ARMA Model	RSE	Change in Level (*t* test)	Change in Slope (*t* test)	$p <$
Level of worry					
P1	MA-1	15.68	–1.78		0.05
P2	AR-1	11.44		2.42	0.01
P3	AR-1	17.88		6.20	0.01
P4	AR-1	28.86		4.00	0.01
Level of anxiety					
P1	MA-1	15.03	–2.98		0.01
P2	AR-1	12.45		6.18	0.01
P3	AR-1	22.22		5.74	0.01
P4	AR-1	22.44		5.70	0.01

Note. AR = autoregression model; MA = moving average model; RSE = residual standard error. For all four participants on both worry and anxiety variables, levels of RSE were within acceptable levels based on the visual inspection of the time-series analyses models.

$p = .01$) after introduction of EMDR treatment. For the other participants, who all presented with comorbid axis I and/or axis II disorders, results revealed a statistically significant change in the slope ($p = .01$), indicating a gradual but significant change in worry and anxiety after the introduction of EMDR treatment.

Diagnosis (SCID-I) at Posttreatment and at Follow-Up

All four participants were readministered the SCID at the end of the EMDR treatment and after a 2-month follow-up period (see Table 2). SCID results for all four participants indicated that they no longer met the diagnostic criteria for GAD at either posttreatment or follow-up. Further, participants 1 and 3 reported no residual symptoms of GAD at either posttreatment or follow-up. Participant 4 had no residual symptoms of GAD at follow-up. Comorbid diagnoses also responded to treatment. With regard to major depressive disorder, participants 2 and 3 no longer met diagnostic criteria at posttreatment and follow-up, and participant 4 lost his diagnosis at follow-up. Some residual depressive symptoms remained for these participants. Participant 2 was diagnosed with a recurrence of his social anxiety disorder at follow-up.

Self-Report Measures at Pretreatment, Posttreatment, and Follow-Up

Table 2 presents a summary of the results for the self-report measures. Results on the Worry and Anxiety Questionnaire are presented in a way that reflects the presence or absence of GAD diagnosis based on the *DSM-IV* diagnostic criteria. Like the SCID interviews, results indicated that for all four participants, they no longer met diagnostic criteria for GAD at both posttreatment and follow-up. For other measures, results generally indicated an improvement for intolerance and uncertainty (QII), for cognitive avoidance (QEC), and for depression (BDI-II) at the time of posttreatment and at the 2-month follow-up.

Weekly Scores on the Penn State Worry Questionnaire

Figure 3 presents the weekly scores (from pretreatment to posttreatment) for all four participants on the QIPS. As can be observed, all participants had initial scores indicating the presence of GAD (60 or more). At the end of the treatment phase, all had scores under the threshold, indicating the presence of GAD. Time-series analyses cannot be performed in this case given the small number of data collected in the course of the baseline period. However, visual inspection shows a general and gradual decrease in QIPS scores as treatment progressed.

Discussion

This study investigated the application of EMDR in treating GAD, and the findings provided preliminary support for its efficacy and usefulness with this disorder. As results indicated, participant 1, who suffered from pure GAD, was successfully treated with 12 sessions of EMDR, and the effects remained at the 2-month follow-up evaluation. The other participants who presented with GAD and comorbid axis 1 and/or

TABLE 2. Results on the Self-Report Measures Across the Three Evaluation Times (Pretreatment, Posttreatment, and Follow-Up)

	Participant 1			Participant 2			Participant 3			Participant 4			
Measures	Pre	Post	Follow-Up	Pre	Post	Follow-Up	Pre	Post	Follow-Up	Pre	Post	Follow-Up	Cutoffs
QIA	Dx	no	no	Dx	no-r	no-r	Dx	no	no	Dx	no-r	no	
QII	71	38	54	97	60	75	103	47	50	33	30	30	71+ = GAD
QEC	60	37	45	45	50	52	98	44	49	68	57	30	67+ = GAD
BDI-II	1	5	1	35	9	15	39	16	9	16	24	0	12+ = dep

Note. QIA = Worry and Anxiety Questionnaire (French Version); QII = Intolerance to Uncertainty Questionnaire (French Version); QEC = Cognitive Avoidance Questionnaire (French Version); BDI-II = Beck Depression Inventory II. Dx = GAD diagnosis; No = absence of GAD diagnosis; No-r = residual GAD symptoms; dep = symptoms of depression.

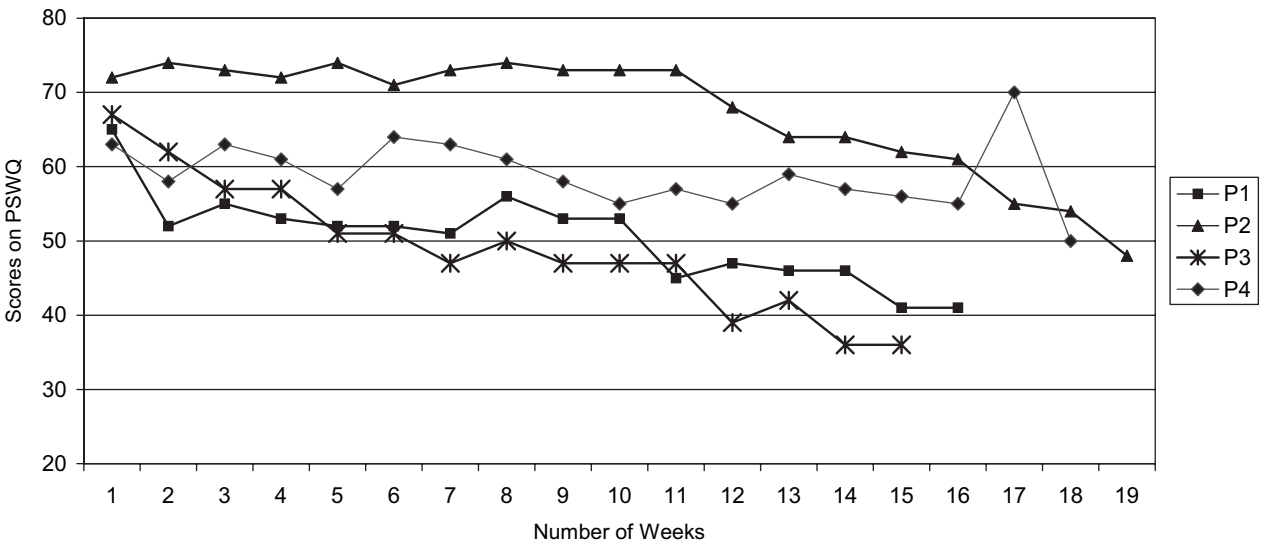

FIGURE 3. Scores on the Penn State Worry Questionnaire.
Scores above 60 indicate the presence of GAD. EMDR treatment began at week 3 for P1, week 4 for P2, week 3 for P3, and week 5 for P4.

axis 2 disorders no longer met diagnostic criteria for GAD following 15 EMDR sessions. Participants 2 and 4 did have some residual symptoms of GAD at posttreatment, and participant 2 had some residual symptoms at follow-up. A possible explanation for these results is that participant 2 had a complex initial presentation, with comorbid major depressive disorder, social anxiety disorder, and axis II avoidant personality features. It is likely that 15 sessions of EMDR were insufficient to resolve all his related issues. Participant 4 also had a more complex presentation, with comorbid major depressive disorder and axis II obsessive-compulsive features. It may be that the presence of comorbid axis II disorder mitigates the therapeutic effects of a treatment aimed directly or targeting uniquely one disorder, in this case GAD.

For all participants, daily levels of worry and anxiety were significantly reduced following the introduction of the EMDR treatment. Again, the current results indicated a difference in the effect of the treatment between participant 1, who suffered from "pure" GAD, and the other participants with comorbid axis I and/or axis II disorders, where changes in daily worry and anxiety were much more gradual, as indicated by the changes in slopes on the time-series analyses.

In order to conceptualize EMDR treatment of GAD, target selection was based on both an etiological model of GAD (Hudson & Rapee, 2004; Rapee, 2001; Shapiro, 2001) and a theoretical model of GAD that involves four key variables (Dugas et al., 1998, 2004; Ladouceur & Dugas, 1999). It appears that EMDR treatment is congruent with this theoretical model. It allows participants to process past events believed to be experiential contributors in the development of anxious disorders, like GAD, and to process current and potential feared situations that cause excessive worries. It appears to result in emotional integration and the elimination of excessive worries and their accompanying anxiety. In addition, the negative cognitions elicited during EMDR treatment were quite

similar to the negative cognitive styles proposed in the theoretical models of Breinholtz et al. (1999) and Chorpita and Barlow (1998). These cognitions related to a poor sense of competence or being unable to cope with stressful events, and they were transformed with EMDR treatment.

It is also interesting to see that changes in depression were observed following the EMDR treatment (see Table 2). As mentioned earlier, major depression has been linked to GAD (Butler et al., 1991; Dugas et al., 1998). For participants 2 and 3, who did present comorbid major depressive disorder, both had decreased levels of depression at posttreatment based on clinician administered and self-report measures. At the follow-up, the level of depression had increased to a "mild depressive state" based on the BDI but still appeared subclinical on the SCID for participant 2. For participant 3, her level of depressive symptoms had decreased further at the time of the 2-month follow-up.

For participant 4, however, levels of depression had increased at posttreatment, even if they were below clinical threshold at pretreatment. One explanation may be that in this case, the experiential contributors to GAD may also have been related to his personality features, given that the expression of emotional material was severely judged in his childhood environment. During the course of EMDR, special attention had to be given to that aspect and required the use of resource development and installation, an intervention aimed at ego strengthening, that can be part of the EMDR protocol when necessary (Korn & Leeds, 2002, Korn & Spinazzola, 2001; Shapiro, 1995, 2004). In his case, full EMDR treatment was not possible within the time frame of 15 EMDR sessions, and not all the past, present, and future targets were treated. It may be that he would have benefited from a larger number of sessions. Still, even with the limited number of sessions, posttreatment results do indicate a significant improvement. While it could be argued that the introduction of an antidepressant 10 days preceding his follow-up evaluation may have affected the results, it is known that antidepressants of this category usually take up to 4 to 6 weeks before reaching full therapeutic effects. In addition, results obtained at posttreatment indicated positive effects before the introduction of the medication.

Strengths and Limitations

This initial study on the efficacy of EMDR in treating GAD has certain strengths. It can be qualified as experimental in nature, with the use of a multiple baseline across participants who were randomly assigned to the various baseline periods. The presence of multiple baselines also allowed for the control of extraneous variables, such as improvement in the environment, spontaneous recovery, or other historical factors. In other words, this design allowed for greater internal validity. In addition, having been able to measure daily levels of worry and anxiety, we were able to perform time-series analyses that statistically supported the effects of EMDR on excessive worry and its accompanying anxiety symptoms in these four cases of GAD.

Particular attention was given to using multimodal assessment so as not to solely rely on self-rated measures. The administration of the clinician administered SCID at pretreatment, posttreatment, and follow-up further supported the results. In addition, consideration was given to treatment fidelity of EMDR by using the protocol developed for research purposes (Korn & Spinazzola, 2001). Another advantage was the administration of EMDR by an experienced clinician and an EMDRIA-certified therapist. Another strength of this research was to take into account the number of EMDR sessions in the context of a complex disorder that would obviously require more sessions than a single-trauma treatment.

Finally, this research was applied in an actual clinical environment with limited exclusion criteria (bipolar disorder, current psychotic features, current severe suicidal ideations, or active substance abuse). This application gives support to the ecological validity and the feasibility of using EMDR to treat GAD in clinical settings and not only with "pure" GAD clients. However, given the current results, it may be that special attention would have to be given to the presence of axis II features in treatment planning and treatment length.

This study is not without limitations. Being a single-case design with four participants, the capacity to generalize results to a larger population of GAD sufferers is limited. In addition, this study did not use a blind assessor to administer the various measures. Even though attention was given to treatment fidelity, this study did not use independent rating of treatment adherence. Finally, the follow-up period was rather short, being 2 months. In future studies, it would be necessary to have evaluations at longer follow-up periods, such as 6 and 12 months.

Future research on the potential efficacy of EMDR in treating GAD should, of course, include studies with large samples of GAD sufferers, using limited exclusion criteria like those used in this study. A randomized clinical trial is recommended, using a large sample of GAD patients to compare the efficacy of cognitive-behavioral therapy and EMDR. In conducting future studies, the use of blind assessors

would also add to the external validity of the research conducted.

Summary

In conclusion, this preliminary study investigated EMDR's application in treating GAD using a researched etiological model and an empirically validated theoretical model of the disorder to select past, present, and future targets for EMDR processing. This single-case design study with multiple baselines across participants provided preliminary support for its efficacy and usefulness with this disorder, as indicated by both statistical analyses and results on both self-report and clinician-administered measures. Results of intervention times-series statistical analyses suggest that EMDR treatment was significantly effective in reducing both excessive worry, the main symptom of GAD, and its accompanying anxiety. Self-report measures and clinician-administered measures indicated that after EMDR treatment and at follow-up, all participants no longer met *DSM-IV* diagnostic criteria of GAD, two of which were in full remission. Finally, it would then further support Shapiro's AIP model, which proposes that unprocessed life experiences have an impact on personality development and are at the basis of psychological disorders.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Balcom, D., Call, E., & Pearlman D. N. (2000). Eye movement desensitization and reprocessing treatment of internalized shame. *Traumatology, 6,* 68–83.

Bergmann, U. (1998). Speculations on the neurobiology of EMDR. *Traumatology, 4,* 4–16.

Bergmann, U. (2000). Further thoughts on the neurobiology of EMDR: The role of the cerebellum in accelerated information processing. *Traumatology, 6,* Article 4.

Bernstein-Carlson, E., & Putnam, F. W. (1993). An update on the dissociative experience scale. *Dissociation, 6,* 16–27.

Bisson, J. I., Ehlers, A., Matthews, R., Pilling, S., Richards, D., & Turner, S. (2007). Psychological treatments for chronic post-traumatic stress disorder: Systematic review and meta-analysis. *British Journal of Psychiatry, 190,* 97–104.

Borkovec, T. D., Alcaine, O. M., & Behar, E. (2004). Avoidance theory of worry and generalized anxiety disorder. In R. G. Heimberg, C. L. Turk, & D. S. Mennin (Eds.), *Generalized anxiety disorder: Advances in research and practice* (pp. 77–108). New York: Guilford Press.

Borkovec, T. D., & Costello, E. (1993). Efficacy of applied relaxation and cognitive-behavioral therapy in the treatment of generalized anxiety disorder. *Journal of Consulting and Clinical Psychology, 61,* 611–619.

Borkovec, T. D., Ray, W. J., & Stober, J. (1998). Worry: A cognitive phenomenon intimately linked to affective, physiological, and interpersonal behavioral processes. *Cognitive Therapy and Research, 22,* 561–576.

Bourque, P., & Beaudette, D. (1982). Étude psychométrique du Questionnaire de dépression de Beck auprès d'un échantillon d'étudiants universitaires francophones. *Revue Canadienne des Sciences du Comportement, 14,* 211–218.

Bouvard, M., & Cottraux, J. (2002). *Protocoles et échelles d'évaluation en psychiatrie et en psychologie* (3rd ed.). Paris: Masson.

Box, G. E. P., & Jenkins, G. M. (1970). *Time series analysis, forecasting and control.* San Francisco: Holden-Day.

Bradley, R., Green, J., Russ, E., Dutra, L., & Westen, D. (2005). A multidimensional meta-analysis of psychotherapy for PTSD. *American Journal of Psychiatry, 162,* 214–227.

Breinholtz, E., Johansson, B., & Ost, L. G. (1999). Cognitions in generalized anxiety disorder and panic disorder patients: A prospective approach. *Behaviour Research and Therapy, 37,* 533–544.

Brown, S., & Shapiro, F. (2006). EMDR in the treatment of borderline personality disorder. *Clinical Case Studies, 5,* 403.

Bruce, S. E., Machan, J. T., Dyck, I., & Keller, M. B. (2001). Infrequency of "pure" GAD: Impact of psychiatric comorbidity of clinical course. *Depression and Anxiety, 14,* 219–225.

Butler, G., Fenell, M., Robson, P., & Gelder, M. (1991). Comparison of behavior therapy and cognitive behavior therapy in the treatment of generalized anxiety disorder. *Journal of Consulting and Clinical Psychology, 59,* 167–175.

Chorpita, B. F., & Barlow, D. H. (1998). The development of anxiety: The role of control in the early environment. *Psychological Bulletin, 124,* 3–21.

Davidson, P. R., & Parker, K. C. H. (2001). Eye movement desensitization and reprocessing (EMDR): A meta-analysis. *Journal of Consulting and Clinical Psychology, 69,* 305–316.

Deacon, B. J., & Abramowitz, J. S. (2004). Cognitive and behavioral treatments for anxiety disorders: A review of meta-analytical findings. *Journal of Clinical Psychology, 60,* 429–441.

De Jongh, A., van den Oord, H. J. M., & ten Broeke, E. (2002). Efficacy of eye movement desensitization and reprocessing in the treatment of specific phobias: Four single-case studies on dental phobia. *Journal of Clinical Psychology, 58,* 1489–1503.

Dugas, M. J., Buhr, K., & Ladouceur, R. (2004). The role of intolerance of uncertainty in etiology and maintenance. In R. G. Heimberg, C. L. Turk, & D. S. Mennin (Eds.), *Generalized anxiety disorder: Advances in research and practice* (pp. 143–163). New York: Guilford Press.

Dugas, M. J., Freeston, M. H., & Ladouceur, R. (1997). Intolerance of uncertainty and problem orientation in worry. *Cognitive Therapy and Research, 21,* 593–606.

Dugas, M. J., Freeston, M. H., Provencher, M. D., Lachance, S., Ladouceur, R., & Gosselin, P. (2001). Le questionnaire sur l'inquiétude et l'anxiété: Validation des échantillons non cliniques et cliniques. *Journal de Thérapie Comportementale, 11,* 31–36.

Dugas, M. J., Gagnon, F., Ladouceur, R., & Freeston, M. H. (1998). Generalized anxiety disorder: A preliminary test of a conceptual model. *Behaviour Research and Therapy, 36,* 215–226.

Dugas, M. J., Ladouceur, R., Brillon, P., Savard, P., & Turcotte, J. (2002). *Traitement cognitivo-comportemental du Trouble d'anxiété généralisée: Manuel du thérapeute.* Manual developed following a research grant from the CIHR.

Dugas, M. J., Ladouceur, R., Léger, E., Freeston, M. H., Langlois, F., Provencher, M. D., et al. (2003a). Group cognitive-behavioral therapy for generalized anxiety disorder: Treatment outcome and long-term follow-up. *Journal of Consulting and Clinical Psychology, 71,* 821–825.

Dugas, M. J., Ladouceur, R., Léger, E., Langlois, F., Provencher, M. D., Boisvert, J. M., et al. (2003b, November). *Group CBT for generalized anxiety disorder: Does change in intolerance of uncertainty predict symptom change beyond non-specific therapy factors?* Poster session presented at the annual convention of the Association for Advancement of Behavior Therapy, Boston, MA.

Dugas, M. J., Marchand, A., & Ladouceur, R. (2005). Further validation of a cognitive-behavioral model of generalized anxiety disorder: Diagnostic and symptom severity. *Journal of Anxiety Disorders, 19* (3), 329–343.

Dupuy, J. B., Beaudoin, S., Rhéaume, J., Ladouceur, R., & Dugas, M. J. (2001). Worry: Daily self-report in clinical and non-clinical populations. *Behaviour Research and Therapy, 39,* 1249–1255.

First, M. B., Gibbon, M., Spitzer, R. L., Williams, J. B. W., & Benjamin, L. S. (1997). *Structured Clinical Interview for DSM-IV Axis II Personality Disorders* (SCID-II). Washington, DC: American Psychiatric Press.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (2001). *Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Patient Edition* (SCID-I/P). New York: Biometrics Research Department, New York State Psychiatric Institute.

Freeston, M. H., Rhéaume, J., Letarte, H., Dugas, M. J., & Ladouceur, R. (1994). Why do people worry? *Personality and Individual Differences, 17,* 791–802.

Gosselin, P., Dugas, M. J., Ladouceur, R., & Freeston, M. H. (2001). Évaluation des inquiétudes: Validation d'une traduction française du Penn State Worry Questionnaire. *L'Encéphale, 27,* 475–484.

Gosselin, P., & Laberge, B. (2003). Les facteurs étiologiques du trouble d'anxiété généralisée: État actuel des connaissances sur les facteurs psycho-sociaux. *L'Encéphale, 24,* 351–361.

Gosselin, P., Langlois, F., Freeston, M. H., Ladouceur, R., Dugas, M. J., & Pelletier, O. (2002). Le questionnaire d'évitement cognitif (QEC): Dévelopement et validation auprès d'adultes et d'adolescents. *Journal de Thérapie Comportementale et Cognitive, 12,* 24–37.

Grant, M., & Threflo, C. (2002). EMDR in the treatment of chronic pain. *Journal of Clinical Psychology, 58,* 1505–1520.

Grenier, S., & Ladouceur, R. (2004). Manipulation de l'intolérance à l'incertitude et inquiétudes. *Revue canadienne des sciences du comportement, 36,* 56–65.

Hudson, J. L., & Rapee, R. M. (2004). From anxious temperament to disorder: An etiological model. In R. G. Heimberg, C. L. Turk, & D. S. Mennin (Eds.), *Generalized anxiety disorder: Advances in research and practice* (pp. 51–74). New York: Guilford Press.

Kazdin, A. E. (1982). *Single-case research designs: Methods for clinical and applied settings.* New York: Oxford University Press.

Kazdin, A. E. (1984). Statistical analysis for single-case experimental designs. In D. H. Barlow & M. Hersen (Eds.), *Single case experimental designs: Strategies for studying behavior change* (2nd ed., pp. 285–324). New York: Pergamon.

Korn, D. L., & Leeds, A. M. (2002). Preliminary evidence of efficacy of EMDR resource development and installation in the stabilization phase of treatment of complex posttraumatic stress disorder. *Journal of Clinical Psychology, 58,* 1485–1487.

Korn, D. L., & Spinazzola, J. (2001). *EMDR treatment manual research protocol.* Unpublished manuscript.

Laberge, M., Dugas, M. J., & Ladouceur, R. (2000). Modification des croyances relatives aux inquiétudes après un traitement du trouble d'anxiété généralisée. *Revue Canadienne des Sciences du Comportement, 32,* 91–96.

Ladouceur, R., & Dugas, M. J. (1999). L'anxiété généralisée. In R. Ladouceur, A. Marchand, & J.-M. Boivert (Eds.), *Les troubles anxieux: Approche cognitive et comportementale* (pp. 31–57). Montreal: Gaétan Morin éditeur ltée.

Ladouceur, R., Dugas, M. J., Freeston, M. H., Léger, E., Gagnon, F., & Thibodeau, N. (2000). Efficacy of a cognitive-behavioral treatment of generalized anxiety disorder: Evaluation of a controlled clinical trial. *Journal of Consulting and Clinical Psychology, 68,* 957–964.

Ladouceur, R., Dugas, M. J., Freeston, M. H., Rhéaume, J., Blais, F., Boisvert, J.-M., et al. (1999). Specificity of generalized anxiety disorder symptoms and processes. *Behavior Therapy, 30,* 191–207.

Ladouceur, R., Gosselin, P., & Dugas, M. J. (2000). Experimental manipulation of intolerance to uncertainty: A study of a theoretical model of worry. *Behaviour Research and Therapy, 38,* 933–941.

Lytle, R. A., Hazlett-Stevens, H., & Borkovec, T. D. (2002). Efficacy of eye movement desensitization in the treatment of cognitive intrusions related to a past stressful event. *Journal of Anxiety Disorders, 16,* 273–288.

Manassis, K., & Hood, J. (1998). Individual and familial predictors of impairment in childhood anxiety disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 37*(4), 428–434.

Marcus, S. V., Marquis, P., & Sakai, C. (1997). Controlled study of treatment of PTSD using EMDR in an HMO setting. *Psychotherapy, 34,* 307–315.

Marcus, S. V., Marquis, P., & Sakai, C. (2004). Three- and 6-month follow-up of EMDR treatment of PTSD in HMO setting. *International Journal of Stress Management, 11,* 195–208.

Matyas, T. A., & Greenwood, K. M. (1990). Visual analysis of single-case time series: Effects of the variability, serial dependence, and magnitude of intervention effects. *Journal of Applied Behavior Analysis, 23,* 341–351.

Maxfield, L. (2002). Appendix C: Commonly asked questions about EMDR and suggestions for research parameters. In F. Shapiro (Ed.), *EMDR as an integrative psychotherapy approach: Experts of diverse orientations explore the paradigm prism* (pp. 393–418). Washington, DC: American Psychological Association.

Maxfield, L., & Hyer, L. (2002). The relationship between efficacy and methodology in studies investigating EMDR treatment of PTSD. *Journal of Clinical Psychology, 58,* 23–41.

Mennin, D. S., Heimberg, R. G., & Turk, C. L. (2004). Clinical presentation and diagnostic features. In R. G. Heimberg, C. L. Turk, & D. S. Mennin (Eds.), *Generalized anxiety disorder: Advances in research and practice* (pp. 3–28). New York: Guilford Press.

Rapee, R. M. (2001). The development of generalized anxiety. In M. W. Vasey & M. R. Dadds (Eds.), *The developmental psychopathology of anxiety* (pp. 481–503). Oxford, UK: Oxford University Press.

Rivard, V., & Bouchard, S. (2005). Les protocoles à cas unique: Une autre façon tout aussi intéressante de faire de la recherche. In D. S. Bouchard & C. Cyr (Eds.), *Recherche psychosociale: Pour harmoniser recherche et pratique* (2nd ed., pp. 207–244). Quebec: Presses de l'Université du Québec.

Roemer, L., Molina, S., Litz, B. T., & Borkovec, T. D. (1997). Preliminary investigation of the role of previous exposure to potentially traumatizing events in generalized anxiety disorder. *Depression and Anxiety, 4,* 134–138.

Roemer, L., Salters, K., Raffa, S., & Orsillo, S. M. (2005). Fear and avoidance of internal experience in GAD: Preliminary test of a conceptual model. *Cognitive Therapy and Research, 29,* 71–88.

Rothbaum, B. O., Astin, M., & Marsteller, F. (2005). Prolonged exposure versus eye movement desensitization and reprocessing (EMDR) for PTSD rape victims. *Journal of Traumatic Stress, 18,* 606–617.

Rubin, A. (1997). *Empirically validating EMDR with single-case designs: A step-by-step guide for EMDR therapists.* Unpublished manuscript.

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

Scientific Computing Associates. (2005). SCA Statistical System (Version 7.1a) [Computer software]. River Forest, IL: Author.

Shapiro, F. (1989). Eye movement desensitization: A new treatment for post-traumatic stress disorder. *Journal of Behavioural and Experimental Psychiatry, 20,* 211–217.

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. (2002a). EMDR and the role of the clinician in psychotherapy evaluation: Towards a more comprehensive integration of science and practice. *Journal of Clinical Psychology, 58,* 1453–1463.

Shapiro, F. (2002b). EMDR treatment: Overview and integration. In F. Shapiro (Ed.), *EMDR as an integrative psychotherapy approach: Experts of diverse orientations explore the paradigm prism* (pp. 27–56). Washington, DC: American Psychological Association.

Shapiro, F. (2002c). Introduction: 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.

Shapiro, F. (2004). *Eye movement desensitization and reprocessing: Part 2 training manual of the two part basic training.* Watsonville, CA: EMDR Institute.

Shapiro, F., & Maxfield, L. (2002). Eye movement desensitization and reprocessing (EMDR): Information processing in the treatment of trauma. *Journal of Clinical Psychology, 58,* 933–946.

Stanley, M. A., & Novy, D. M. (2000). Cognitive-behavior therapy for generalized anxiety in late life: An evaluative overview. *Journal of Anxiety Disorders, 14,* 191–207.

Stapleton, J. A., Taylor, S., & Asmundson, G.J.G. (2006). Effects of three PTDT treatments on anger and guilt: Exposure therapy, eye movement desensitization and reprocessing, and relaxation training. *Journal of Traumatic Stress, 19*(1), 19–28.

Stickgold, R. (2002). EMDR: A putative neurobiological mechanism in action. *Journal of Clinical Psychology, 58,* 62–75.

Van der Kolk, B. A. (2002). Beyond the talking cure: Somatic experiences and subcortical imprints in the treatment of trauma. In F. Shapiro (Ed.), *EMDR as an integrative psychotherapy approach: Experts of diverse orientations explore the paradigm prism* (pp. 57–84). Washington, DC: American Psychological Association.

Van der Kolk, B. A., Spinazzola, J., Blaunstein, M. E., Hopper, J. W., Hopper, E. K., Korn, D. L., et al. (2007). A randomized clinical trial of eye movement desensitization and reprocessing (EMDR), fluoxetine, and pill placebo in the treatment of posttraumatic stress disorder: Treatment effects and long-term maintenance. *Journal of Clinical Psychiatry, 68,* 1–9.

Van Etten, M. L., & Taylor, S. (1998). Comparative efficacy of treatments for post-traumatic stress disorder: A meta-analysis. *Clinical Psychology and Psychotherapy, 5,* 126–144.

Wei, W. W. (1990). *Time series analysis: Univariate and multivariate methods.* New York: Addison-Wesley.

Western, D., & Morrison, K. (2001). A multidimensional meta-analysis of treatments for depression, panic, and generalized anxiety disorder: An empirical examination of the status of empirically supported therapies. *Journal of Consulting and Clinical Psychology, 69,* 875–899.

Wilson, S. A., Becker, L. A., & Tinker, R. H. (1995). Eye movement desensitization and reprocessing (EMDR) treatment for psychologically traumatized individuals. *Journal of Consulting and Clinical Psychology, 63,* 928–937.

Wilson, S. A., Becker, L. A., & Tinker, R. H. (1997). Fifteen-month follow-up of eye movement desensitization and reprocessing (EMDR) treatment for posttraumatic stress disorder and psychological trauma. *Journal of Consulting and Clinical Psychology, 65,* 1047–1056.

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