Limitations of Trauma-Focused Therapies for Treating PTSD:

A Perspective

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May 2015

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Many therapeutic approaches developed for treating PTSD focus primarily on reliving the traumatic event or include a trauma-focused component. Focusing on traumatic memories can be harmful to individuals suffering from PTSD, although such therapeutic can at times be very helpful given that prerequisite conditions are in place. Trauma-focused therapies vary in their emphasis on reexperiencing traumatic events and their attitude toward the need to reexperience such dysphoric memories. This article attempts to demonstrate that, although they are highly praised by clinical researchers, highly trauma-focused therapies have more limitations than envisioned by their enthusiastic proponents.

Behavioral therapies focusing primarily on reexperiencing traumatic events view PTSD mostly as a fear response. Consequently, they attempt to de-activate trauma-based fear responses. These therapies provoke very high anxiety in participants, if not panic, expecting that a desensitization process will take place on its own like in prolonged exposure, PE (Foa & Meadows, 1997). Traditionally, immersion (newly renamed prolonged exposure) requires that the therapist perseveres in inviting participants to stay into the traumatic reexperiencing until the neurobiological system of anxiety becomes exhausted and anxiety cannot be produced anymore. Another therapy primarily focusing on traumatic events does not wait for anxiety to subside on its own, namely eye movement desensitization and reprocessing, EMDR (Shapiro, 1995). This technique quickly requires the participant's attention to focus, in parallel to reexperiencing traumatic memories, on a dual task (producing a divided attention) One of the rational offered by proponents of PE and EMDR is that a relaxation response will replace anxiety. These two highly trauma-focused therapies (PE and EMDR) are worrisome to some clinicians who have attempted them because participants can be destabilized and experience negative side effects.

Other trauma-focused therapies do not primarily focus on the traumatic event. Although they entail an experiential revision of a traumatic event, they primarily focus on altering distorted cognitions as in cognitive processing therapy, CPT (Resnick & Schnicke, 1992) or working through intrapsychic issues and relational patterns as in brief dynamic therapy, BDT (Horowitz, 1976, 1984, 1997, 2001).

The quality and the quantity of exposure to traumatic events vary immensely across trauma-focused therapies. PE requires participants to sequentially relive a traumatic event *in vitro* and bear the high anxiety until it decreases spontaneously. EMDR involves reliving the worst moment of traumatic event from the very beginning, as vividly as possible, and then pay attention to a dual task (finger moving or tapping) until anxiety decreases. In both therapies, the described process is repeated again and again until anxiety goes down as much as possible.

More gentle trauma-focused therapies do not entail forcing the participant's attention onto the experiential revision of traumatic events. In CPT, the exposure component consists of writing a description of a traumatic event while experiencing the involved emotions in order to identify salient dysfunctional cognitions. The trauma-related component of BDT involves gradually engaging the participant in an in-depth exploration of the traumatic event and experience in order to identify personal meanings and embedded conflicts.

Despite the numerous clinical trials supporting the efficacy of highly trauma-focused techniques (PE and EMDR), an increasing number of studies have demonstrated the efficacy of CPT and few have examined the efficacy of BDT. Major research questions and clinical concerns remain unanswered. Before looking at efficacy studies, let's describe in more details each one of these therapeutic methods regarding their exposure component.

PE is an exposure technique, an immersion or flooding technique as employed with simple phobias. Rather than being exposed to feared object, the patient is exposed to intensely dysphoric traumatic memories. The patient is exposed to a traumatic event in a sequential fashion. Upon exposure, the patient becomes highly aroused and at times very anxious. The patient is told to keep on attending to any anxiety-provoking stimuli until the anxiety subsides. Such reduction of anxiety (arousal) is supposed to be induced by habituation. By practitioners of this method, habituation is expected to happen spontaneously, allowing a relaxation response to emerge. It is inferred that associations between trauma-related fears and the relaxation response will be established when patients have calmed down. PE is applied from the first or second session of therapy and at every session afterward. The session is audiotaped and the participant is supposed to listen to the recording few times per day between sessions. After many sessions of imaginary exposure, exposure is conducted *in vivo* toward trauma-related stimuli if it is required (Foa & Riggs, 1993).

The technique of EMDR is often applied from the second session of therapy and at every session afterward. In EMDR, therapists do not proceed sequentially in having patients relive traumatic events. At the very beginning of the EMDR technique, therapists invite patients to relive, as fully as possible, the worst moment of the traumatic. Then, they apply a dual task consisting of eye movements, finger tapping, etc. (Shapiro, 1995). Patients end up reexperiencing their worst traumatic moment (what is most dysphoric) as fully as possible (emotions, cognitions, sensations, etc.) and then being invited to simultaneously pay attention to a trivial sensory phenomenon, dividing the attention of the participant. This process is repeated until subjective distress disappears. Afterward, a positive cognitive is provided to replace the previously spontaneous negative cognition and the dual task is applied again to implement the chosen positive cognition. The process is repeated until the positive cognition is fully endorsed.

CPT is a cognitive therapy designed for treating PTSD (Resick & Schnicke, 1992). This cognitive method aims at altering basic schemas disrupted by a traumatic event. After patients are taught about the influence of cognitions over emotions through worksheets, they write by themselves a description of the traumatic event while allowing themselves to experience as

fully as possible the emerging emotions. 'Stuck points' are identified, that is, where participants had difficulties elaborating. Their disruptive cognitions and schemas at those points are identified and work in a cognitive manner. CPT differs from PE and EMDR in that the exposure component is circumscribed and occurs after few sessions. The CPT exposure component is also conducted under patients' control and it involves an in-depth therapeutic work on disruptive cognition schemas.

BDT for PTSD (Horowitz & Kaltreider, 1979) is suggested to be used only with individuals presenting with acute PTSD in well-functioning adults without comorbidity or structural deficits. This is the only trauma-focused therapy described hereby identifying inclusion criteria to prevent any destabilization. At the beginning, BDT involves obtaining a cognitive description of the traumatic event, establishing an alliance, completing the patient's history, and reducing any overwhelming anxiety. From the fourth to the eight sessions, the patient reviews the traumatic event at the usual state of consciousness, with the therapist and only as long as it is tolerable. The defense mechanisms employed toward affect-laden issues are addressed. Conflicts are resolved through interpretations and linkages with present or past relationships. Patient's internal representations of self and others are revised accordingly. Termination involves acknowledging therapeutic gains and making plans for the future. In BDT, emphasizes the need to proceed at tolerable dosage without inducing overwhelming anxiety and addressing peripheral issues before exploring core traumatic experiences. The goal is the integration of traumatic memories into the psychological structure of the patient after the psychological structure has been accommodated sufficiently to allow for the assimilation of the traumatic memories.

In the following sections, we will examine the efficacy of trauma-focused therapies for treating PTSD and the limitations of the obtained findings. The term 'efficacy' is hereby employed intentionally, that is, in contrast to the term 'effectiveness'. 'Efficacy' refers to the capacity of a therapy to induce intended changes as established by a controlled clinical trial. 'Effectiveness' refers to the induction of beneficial changes in clinical practice, the real world.

Efficacy of Trauma-Focused Techniques

Before examining the efficacy of specific trauma-focused therapies, let's start by considering the efficacy of all psychological therapies which were evaluated for treating PTSD. A meta-analysis of 26 controlled clinical trials assessed the efficacy of various psychological therapies for PTSD without comorbid disorders (Bradley, Greene, Russ, Dutra, & Western, 2005).* The authors found that all therapies were equally efficacious (as it is almost always the case in evaluation research). From pre-test to post-test, the effect size was 1.43 for all therapies, indicating a very large effect. The effect size for therapies versus wait-list controls was 1.11, sustaining the very large effect of therapies for PTSD. The effect size for experimental therapies versus supportive therapies was 0.83, indicating a large effect. However, the effect size for exposure versus non-exposure therapies was -0.11, indicating a lack of difference in efficacy. Across therapies, 56% of all participants who entered treatment no longer met criteria for PTSD at post-test (reaching partial PTSD remission) and 67% of completers obtained a partial PTSD

remission. Interestingly, clinical improvement was judged to have occurred in only 44% of all participants who entered treatment and in only 54% of completers. The authors underlined that the observed means of PTSD measures indicated the presence of considerable residual symptoms at post-test. Indeed, all these therapies were time-limited, which is likely to render these therapies restricted in their efficacy.

** Another meta-analysis performed by Bisson, Ehlers, Matthews, Pilling, Richards, and Turner (2007) also compared the efficacy of all types of therapies for PTSD. However, no clinically meaningful results were provided by the authors, rendering one's interpretation of their data and conclusions impossible. With large numbers, differences can be determined as statistically significant, especially without a Bonferroni correction. However, differences can be meaningless clinically. Consequently, these findings are not considered here.*

The influence of various characteristics of controlled clinical trials was also examined by Bradley et al. (2005). Of interest to our purpose, only some results are presented here. Sample size was unrelated to outcome. Completion rate was negatively related to pre- versus post-test effect size, indicating that the more participants dropped out of therapy, the greater was the effect size observed in completers . The number of exclusion criteria was significantly related to pre- versus post-test effect size; studies with more exclusion criteria reported higher effect sizes, thus greater efficacy, because participants were initially less symptomatic and more functional. Bradley et al. (2005) concluded that both exclusion criteria and the failure to address comorbidity impede the generalizability of these meta-analytic findings to the general population of PTSD patients.

It is important to understand here that, when PTSD remission is considered in controlled clinical trials, the reported rate of 'PTSD remission' applies only to partial PTSD remission. Indeed, a person can remain with substantial PTSD symptoms without meeting the diagnostic criteria for PTSD. Bradley et al. (2005) underlined that means on PTSD measures at post-test indicated that improved participants presenting with a PTSD remission continued to present considerable PTSD symptoms. Therefore, when readers peruse abstracts of published articles and see 'PTSD remission', they should not assume that PTSD was fully remitted and considerable symptoms are still present.

Prolonged Exposure. A meta-analysis by Powers, Halpern, Ferenschak, Gillihan, & Foa (2010) aimed at demonstrating the superiority of PE over psychological placebos. Only controlled clinical trials with the PE developed by Foa were considered, leaving only 13 controlled clinical trials to be included (N = 675). Results indicated that PE was as efficacious as other cognitive and/or behavioral therapies (anxiety management as SIT, cognitive therapy, CPT, and EMDR). At post-test, comparisons between PE and wait-list controls yielded an effect size of 1.51. In comparison to psychological placebos (according to authors, the supportive therapies which were used as psychological placebos to control for nonspecific ingredients; therefore, we consider that these therapies were not expected to be efficacious by experimenters), the effect size of PE was 1.08, which represents a very large effect size. The major pitfall of this meta-analysis is that it was conducted by Foa and her colleagues to

demonstrate that her PE therapy is superior using only controlled clinical trials including PE according to Foa's method. Unfortunately, such strategy entails an experimenter bias, especially toward the 'other therapies' (supportive and non-cognitive-behavioral) which had been expected to be less efficacious than PE and any other 'active' therapies (cognitive-behavioral) (see section below). Furthermore, rates of PTSD remission (partial or complete) were not provided, although these are the most significant clinical information to both practitioners and patients.

To understand the efficacy of PE, let's look at one of the controlled clinical trials in which rates of PTSD remission, partial and complete, are provided. Schnurr, Friedman, Engel, Foa, et al. (2007) compared the efficacy of PE to present-focused therapy and wait-list as controls (N = 284). Treatment dropout rate was higher in PE (38%) than in present-focused (21%), almost double. At post-test, partial PTSD remission was observed in 41% of participants of PE versus 28% of participants of present-focused therapy (these percentages are for all participants who entered therapy). Complete PTSD remission was found in 15% of participants of PE and 7% of participants of present-focused therapy. At post-test, scores Clinician-Administered PTSD Scale (CAPS) scores were lower with PE than with present-centered therapy overall (d = 0.46), which is a moderate effect. Of outmost clinical significance, the difference in PTSD severity scores on the CAPS found at post-test vanished at 6-month follow-up (for analyses conducted on all participants and on completers only); rates of PTSD remission were 40% in PE and 33% in present-focused therapy for all participants. Schnurr et al. (2007) concluded in their abstract that "Prolonged exposure is an effective treatment for PTSD in female veterans and active-duty military personnel." Such conclusion is not consistent with the above findings. From these reported results, one could rather conclude that time-limited present-focused therapy and PE are efficacious treatments for treating PTSD but only for some individuals, and most participants remain with considerable symptoms.

The efficacy of PE for treating PTSD was also tested in borderline personality disorder (BPD). Feeny, Zoellner, and Foa (2002) found that only 11% of PE participants with PTSD and BPD, only 11% achieved good functioning at post-treatment and 22% at 3-month follow-up, even though 56% showed partial PTSD remission. Similar results were obtained in stress inoculation training (SIT) and PE with SIT.

Taken together, findings from controlled clinical trials indicate that PE is not more efficacious to treat PTSD than any other forms of psychological interventions. Other therapies aside from PE are gentler such as supportive therapy, anxiety-management, present-focused therapy, traditional cognitive-behavioral therapy, and brief dynamic therapy (see Brom et al., 1989, included in Bradley et al., 2005). As other time-limited therapies, PE is associated with a reduction of PTSD symptoms in some patients, but rarely a full PTSD remission. Its dropout rate can be high and some have reported severe side effects (see below).

Eye movement desensitization and reprocessing. In a meta-analysis of 34 studies, Davidson and Parker (2001) evaluated the efficacy of EMDR for treating PTSD. Findings indicated that EMDR reduced subjective distress within sessions and PTSD symptomatology at

post-test. In comparison to a wait-list control, an effect size of 0.98 was obtained at post-test, a result somewhat comparable to those of Bradley et al. (2005). In this meta-analysis, EMDR was compared to other exposure techniques and it was found to be equally efficacious, no more. Finally, whether therapists were trained or not by the EMDR Institute had no impact on results. These meta-analytic findings are at variance with claims made by EMDR proponents that this technique is particularly powerful for treating PTSD and should be applied only by therapists trained by the EMDR Institute.

In another meta-analysis (Bradley et al., 2005), EMDR was found to be as efficacious as PE, cognitive-behavioral therapy, and 'other therapies'. EMDR is thus no more efficacious than traditional cognitive-behavioral techniques and supportive therapies. The effect size for the comparison of all treatments versus waiting-list effect was 1.11. When dropout or attrition data was included in statistical analyses, an average rate of 56% of participants presented with partial PTSD at post-test for all therapies, including EMDR. Bradley et al. (2005) concluded that the observed means of PTSD measures at post-test indicate the presence of considerable residual symptoms, a conclusion which also applies to EMDR.

Powers et al. (2010) found similar results. As described above, their meta-analysis indicated an equal efficacy between EMDR, PE, CPT, anxiety management, and traditional cognitive therapy.

According to the meta-analysis by Bisson et al. (2007), the analyses conducted in controlled clinical trials with EMDR were performed only with the data of participants who completed treatment, a strategy inflating results (Bradley et al., 2005).

Cognitive processing therapy. The above conclusion of the meta-analysis of Powers et al. (2010) pertains to the efficacy of CPT for treating PTSD. CPT was found to be as efficacious as other therapies for treating PTSD. CPT is thus as efficacious as traditional cognitive-behavioral therapies and anxiety management therapies which were not trauma-focused. CPT was also found to be as efficacious as other therapies comprising exposure such as PE and EMDR. Therefore, there was no difference between CPT, trauma-focused therapies, and gentler therapies in the meta-analysis of Powers et al. (2010).

One meta-analysis (Barrera, Mott, Hofstein, & Teng, 2013) was specifically performed to compare the efficacy of cognitive-behavioral therapy with and without an exposure component (including CPT) in group settings (N = 651). CPT is a cognitive therapy entailing an exposure component and is conducted within a group setting. While the average pre- versus post-test effect size was 1.13, no difference was found between the two conditions (Barrera et al., 2013). Therefore, the exposure component did not add any efficacy to the treatment of PTSD.

Taken together, these meta-analytic findings indicate that CPT is as efficacious as traditional cognitive-behavioral therapies, including those without an exposure component. Cognitive-behavioral therapy is thus efficacious with or without an exposure component, supporting the need to choose gentler approaches for treating PSTD given the reduced

likelihood of attrition and side effects.

One controlled clinical trial (Galovski, Blain, Mott, Elwood, & Houle, 2012) examined the efficacy of providing CPT in a flexible manner, according to participants' progress, rather than following a strictly manualized format. The number of sessions was determined by participant's progress toward end-state criteria. Trauma-focused sessions were inserted whenever necessary. Treated participants obtained greater PTSD improvements than the wait-list control group although dropout rate was high (28%). Among completers, 58% reached end-state criteria prior to the 12th session, 8% at session 12, and 34% between sessions 12-18. The use of trauma-focused sessions did not result in poorer treatment outcomes. Maintenance of gains was found at the 3-month follow-up, with only 4% meeting criteria for PTSD. If the dropout rate is added, about 32% met PTSD diagnosis criteria at follow-up. Therefore, adding flexibility to gently trauma-focused therapies appears to benefit PTSD sufferers.

Brief dynamic therapy. The efficacy of BDT for PTSD has not been assessed in a controlled clinical trial since the 1990s. Highly competent researchers have submitted grants to the PTSD Division of NIMH, but to no avail. Some valuable findings are, nonetheless, available regarding its efficacy and effectiveness. Data from uncontrolled trials are available, along with findings of one controlled clinical trial.

One study (Krupnick, 2002) indicated that addressing interpersonal problems promotes PTSD remission. This study randomized individuals with PTSD to either group interpersonal therapy or a wait-list control. Significant PTSD improvements were found in those who received the interpersonal therapy. This BDT consisted of a dynamic therapy based on Yalom's (1995) group interpersonal psychotherapy.

Lindy, Green, Grace, MacLeod, and Spitz (1988) conducted an uncontrolled study of this BDT. Therapy focused on consciously addressing traumatic memories instead of repressing them. Despite a 30% dropout rate, completers obtained PTSD improvements in addition to decreases in feelings of alienation, depression, hostility, and substance abuse. Noted improvement was made in their increased capacity to trust and manage traumatic stress, as well as to feel appreciation of being alive, greater personal integrity, less estrangement, more investment in adult roles and constructive activities, and continuity with sense of self before the war (in Schottenbauer, Glass, Arnkoff, & Gray 2008).

At a hospital clinic specialized in PTSD, Horowitz, Marmar, Weiss, Kaltreider, and Wilner (1986) reported that PTSD symptoms decreased on the Impact of Event Scale (IES) after 12 sessions of the BDT developed by Horowitz. Significant improvements were also obtained regarding relational and occupational functioning.

As pointed out by Schottenbauer et al. (2008), the models provided by Lindy (1996) and Horowitz and Kaltreider (1979) are brief dynamic psychotherapies intended for well-functioning individuals having experienced event. Patients with more substantial histories of trauma or with complicating factors may require more sessions or a qualitatively different treatment. Let's

examine findings from a naturalistic setting where no such exclusion criteria were applied and no limit was set regarding therapy duration.

At a clinic specialized in treating PTSD with a dynamic integrative model (Gaston, 1995), an independent and retrospective study was conducted as preliminary data gathering for a neuroimagery study. Brunet (2004) randomly selected files (n = 100) in which PTSD was diagnosed with the Structured Clinical Interview for DSM-IV-PTSD (SCID) before and after treatment. The dynamic integrative psychotherapy was based on Horowitz's model for PTSD, augmented by Masterson and Klein's (1989) and Bowlby 's (1988) models for personality disorders. Cognitive and behavioral interventions as well as pharmacotherapy are sometimes employed to reduce anxiety and depression (Gaston, 1995). In this naturalistic setting, the psychotherapy lasted as long as it is clinically required, with the intention of completely resorbing the PTSD and its comorbidity, reestablishing pre-morbid functioning, and preventing PTSD relapse. At the clinic, most patients present with severe PTSD, comorbid disorders (mostly major depression disorder, panic disorder, and agoraphobia), interpersonal difficulties, and incapacity to work. The findings indicated that the psychotherapy lasted an average of 9 months. The rate of PTSD remission was 96% (48% of complete remission).

At the same clinic, a prospective neurological study (Dickie et al., 2011) found that the sample of participants had the following characteristics: 89% presented with severe PTSD, 78% had at least a comorbid disorder, and 44% took psychotropic medication. Results indicated that, after 6 to 9 months (therapy was still proceeding for most), the rate of PTSD remission was already 65% on the CAPS. PTSD symptoms had decreased in all patients but one over the course of the study (d = 1.25). Using magnetic resonance imaging (fMRI), it was found that, upon the presentation of fearful faces as stimulation, neurobiological changes in the amygdala, hippocampus and prefrontal anterior cingulate cortex were associated with PTSD changes at 6-9 months after beginning of therapy. Together, these neurobiological findings indicated neural systemic efficiency in patients presenting with PTSD remission.

One controlled clinical trial evaluated the efficacy of brief dynamic therapy (Brom, Kleber, & Defares, 1989). The efficacy of three therapies for PTSD were compared to a control group: trauma desensitization (similar to systematic desensitization), hypnosis (similar to PE), and BDT following Horowitz's model, and a wait-list control (n = 112). At post-test, all treatments were equally efficacious and more efficacious than the wait-list. Significant clinical improvements were found in 60% of treated participants in comparison to 25% of controls. At 3-month follow-up, effect sizes regarding PTSD severity (pre-test versus follow-up data) were 1.0 for trauma desensitization, 1.0 for hypnosis, 1.2 for BDT, and 0.3 for the waiting-list condition (Brom et al. did not provide effect sizes, but the means and standard variances provided in the article permitted such calculations).

The study of Brom at al. (1989) is the only controlled clinical trial having included brief dynamic therapy for treating PTSD. Regrettably, there seems to be bias against dynamic therapy in grant agencies. This status is particularly puzzling given that the beneficial effects of any dynamic therapy have been shown to increase over time according to five independent

meta-analyses (Shedler, 2010). Applying such findings to the treatment PTSD by dynamic therapy would be a reasonable but untested generalization.

Experimenter Bias

In any experiment or clinical trial, there is always the possibility of an experimenter bias, a phenomenon working beyond the awareness of researchers hopefully. In experimental science, experimenter bias is also known as research bias. It is a subjective bias toward a result expected by the experimenter. For example, it occurs when scientists unconsciously affect subjects in experiments (Kantowitz; Roediger, & Elmes, 2009). In a review of biases in clinical studies, biases can occur in any stage of research: in reading-up on the field, in specifying and selecting the study sample, in executing the experimental maneuver, in measuring exposures and outcomes, in analyzing the data, in interpreting the analysis, and in publishing the results (Sackett, 1979). For an experimenter effect to occur, experimenters subtly communicate their expectations to the participants, who alter their behavior to conform to these expectations.

Because of possible experimenter biases, findings obtained in controlled clinical trials by proponents of a given therapy need to be replicated by non-supporters of the main hypothesis. Therefore, findings about the efficacy of trauma-focused techniques for treating PTSD need to be replicated by non-enthusiastic researchers. Let's look at studies involving a clear bias.

In a seminal study aimed at demonstrating the efficacy of PE, Foa, Rothbaum, Riggs, and Murdock (1991) assessed whether participants with PTSD (n = 45) showed improvements after PE, Stress Inoculation Training (SIT), supportive counseling (SC) versus a wait-list. At post-test, PTSD remission rates were found to be 40% in PE, 50% in SIT, and 10% in SC. At 3-month follow-up, PTSD remission rates were had become equivalent: 55% in PE, 55% in SIT, and 45% in SC. These findings cannot support any claim of superior efficacy of PE or SIT over supportive counseling. The authors concluded in the abstract that 'At follow-up, PE produced superior outcome on PTSD symptoms', a conclusion in contrast with their own text stating 'The three treatment groups (SIT, PE, SC) did not differ in the percentage of PTSD patients at follow-up' (p. 720). These authors are favourably biased toward PE.

A major bias in this controlled clinical trial conducted by Foa et al. (1991) is that supportive counseling (SC) was intended to behave as a control condition, with the expectation that SC should not be effective. The supportive therapy offered to participants with PTSD consisted of the following: (a) teaching of a general problem solving technique (b) the therapist responding indirectly and with unconditional support, and (c) immediate refocus by the therapist to everyday problems when the patient tried to talk about the sexual assault. This experimenter bias was fulfilled by participants who seemed to provide data at post-test in support of the researchers' hypothesis. However, such reactivity was not sustained at follow-up. As indicated by Benish, Imel, and Wampold (2008), such 'control therapies' or 'other therapies' are not *bona fide* therapies, that is, they do not reflect how they are conducted by clinicians in practice.

Another example of enthusiastic conclusions about trauma-focused therapies versus supportive therapies is the study of Schnurr et al. (2007), co-authored by Foa. Based on data of completers, PTSD scores on the Clinician-Administered PTSD Scale (CAPS) were found to be lower in PE at post-test (d = 0.54) and at 3-month follow-up (d = 0.34), but the difference had vanished at the 6-month follow-up (d = 0.29). At 6-month, if dropouts were to be included in the analysis, effect size would drop to 0.15. Surprisingly, Schnurr et al. (2007) concluded in their abstract that 'Prolonged exposure is an effective treatment for PTSD in female veterans and active-duty military personnel. It is feasible to implement prolonged exposure across a range of clinical settings.' No mention was made of the equal efficacy of present-focused therapy after 6 months of follow-up.

A more recent controlled clinical trial compared the efficacy of PE versus BDT for treating adolescents after a single traumatic event (Gilboa-Schechtman, Foa, Shafran, et al., 2010). Incredibly, the clinicians providing the dynamic therapy were trained for only two days. Therefore, this controlled clinical trial is flawed with a serious bias against BDT. Unlike cognitive-behavioral therapies, providing dynamic therapy requires months to years of training. Nonetheless, the results are still interesting because the superior efficacy of PE at post-test (d = 0.45) and 6-month follow-up (d = 0.51) dissipated at a 17 month follow-up (d = 0.21). Again the initial difference in treatment efficacy may well have reflected the experimenters' bias. Nonetheless, the support in favor of the experimental hypothesis dissipated over time, despite the efforts of researchers to use a non *bona fide* therapy.

Some other researchers have compared cognitive-behavioral therapies with 'other therapies' intended to work as control placebos, usually supportive or dynamic therapies. Afterward, they claim that cognitive-behavioral therapies for PTSD have more empirical support than 'other therapies' in terms of efficacy for treating PTSD (Foa, Keane, & Friedman, 2004; Nemeroff, Bremmer, Foa, Mayberg, North, & Stein, 2006). However, the inadequacies enforced onto the delivery of 'other' therapies, the lack of appropriate training in dynamic therapy, and experimenter biases against 'other therapies' might have produced results indicating greater short-term efficacy of the endorsed trauma-focused therapies, but these effects were non-lasting.

To remedy this situation, Benish et al. (2008) conducted a meta-analysis on the efficacy of all *bone fide* therapies for treating PTSD. Therapies were considered 'bona fide' if they were expected by experimenters to be efficacious and if the trial was conducted in a valid fashion. An important aspect of the meta-analysis of Benish et al. (2008) is that the 'other therapies' were disaggregated and those which were intended to be therapeutic were included while those which were not intended to be therapeutic were excluded. Results indicated no efficacy difference between *bona fide* therapies and trauma-focused therapies in treating PTSD.

An experimenter bias can appear under many forms. A recent meta-analysis (Ehring, Welboren, Morina , Wicherts, Freitag, & Emmelkamp, 2014) exclusively focused on studies evaluating the efficacy of psychological interventions for PTSD in adult survivors of childhood abuse. Sixteen randomized controlled clinical trials meeting inclusion criteria could be identified

that were subdivided into trauma-focused cognitive-behavioral therapy (CBT), non-trauma-focused CBT, EMDR, and other treatments (interpersonal, emotion-focused). Their results showed that psychological therapies are efficacious for treating PTSD in adult survivors of childhood abuse. They also indicated that trauma-focused therapies were more efficacious than non-trauma-focused ones. However, this meta-analysis excluded one of the landmark studies in the field (Classen, Palesh, Cavanaugh, et al., 2011) in which no difference was found between trauma-focused and present-focused group therapy. Such exclusion by Ehring et al. (2014) suggests a bias on the part of the researchers.

Experimenters' biases are rarely acknowledged in clinical literature. This is unfortunate because it leads to exaggerated claims by enthusiastic researchers. If we go back to the basic teachings on experimental designs, experimenter bias is a prime issue of concern. At this point in time, so many cognitive-behavioral researchers have shown a lack of adequate representation of non-cognitive-behavioral therapies in their clinical trials that it would be prudent to consider from now on any new study conducted to be biased in favour of PE, EMDR, etc. Unfortunately, the bias of trauma-focused researchers is such that it prevents any consideration of their findings. Although science is useful, empirical findings always have limitations, but biased empirical findings have severe limitations. Regrettably, in this race to prove that trauma-focused therapies are best, it is the individuals with PTSD who are those losing the most.

Subject Reactivity

Reactivity of subjects is a phenomenon that occurs when individuals alter their performance or behavior due to the awareness that they are being observed. The change may be positive or negative, and depends on the situation. It is a significant threat to a research study's internal validity (Heppner, Wampold, & Kivlighan, 2008). As a researcher, it is also possible to see that subjects tend to respond in ways to confirm experimenters' hypothesis (for one reason or another; maybe people enjoy when things work out or they simply do not like to tolerate cognitive dissonance). Despite experimenters' best efforts, the cherished treatments are known by subjects in any controlled clinical trial of psychological therapies, especially if media-praised techniques such as PE or EMDR are included.

Clifford and Maisto (2000) have extensively documented subject reactivity in clinical trials of alcohol treatment. In a study designed to test subject reactivity. There are many types of subject reactivity and factors influencing it. For example, Clifford, Maisto and Davis (2007) have demonstrated that subjects assigned to the infrequent-brief research assessment exposure condition reported the poorest outcomes.There is no reason to assume that such effect is not present in clinical trials for PTSD. Let's look at an example in the field of PTSD where subject reactivity appears to be at play.

In the study of Foa et al. (1991), rates of PTSD remission at post-test were 40% in PE, 50% in SIT, and 10% in supportive counseling (SC). At 3-month follow-up, the rates had become equivalent across treatment conditions: 55% in PE, 55% in SIT, and 45% in SC. Given the

'spontaneous' remission rates in the SC group between post-test and 3-month follow-up (an increase of 35% of PTSD remission), subject reactivity seems to have been at play in this controlled clinical trial. In short, participants provided at post-test what experimenters were expecting. Nonetheless, supportive counseling was dismissed by the authors as inefficacious.

Let's look at data from another field. An important study demonstrated that subjects' suggestibility during and after hypnosis influenced their reports (Laurence & Perry, 1983). While hypnotized, hypnotizable subjects were told that, one night of the previous week, they were awakened by a loud noise. When these subjects were interviewed without being hypnotized the next day, about 50% insisted that their memory was genuine. When the experimenters told them that this was a pseudo-memory generated as part of the experiment, some of the subjects refused to believe the memory was not accurate and true. One week later, subjects maintained that the pseudo-memory was genuine even though they were shown the video of the hypnotic session demonstrating that it was the experimenter who had suggested the memory. In a further study (McConkey, Labelle, Bibb, & Bryant, 1990) using a different paradigm (misinformation effect), a number of subjects accepted the misinformation, with or without hypnosis. When these subjects were interviewed on the phone a few weeks later by another experimenter they did not know, subjects expressed doubts about the veracity of the false information. As confirmed by many later studies, timing of the interview and the context of the interview play a role in the acceptance of false memories. If such phenomenon can occur in the reporting of false memories, it can certainly occur in the reporting of clinical symptoms after participants have quite extensively interacted with researchers, therapists, and interviewers. Participants might also want to delude themselves in reporting that they feel better than they are given the time and energy they spent trying to get better in the context of a clinical trial, as per the phenomenon of cognitive dissonance.

Given, the media emphasis on the efficacy of PE and EMDR, participants in controlled clinical trials are likely to provide answers in support of experimenters' claims, that is, to give answers the experimenters are looking for. This may particularly occur because PE and EMDR therapists have been reported to harshly influence patients into considering these techniques favorably and not report anything negative about them (see patients' reports provided in the section below on negative side effects).

As with experimenter bias, subject reactivity negatively affects the internal validity of clinical trials, and, therefore, their external validity. Such confounding factor should not be ignored or minimized.

Attrition

Attrition or dropout rates are important clinical features of a study. Attrition or dropout rate reflects the applicability and tolerability of a therapy. Schottenbauer, Glass, Arnkoff, & Gray (2008) examined the dropout rates in 55 controlled clinical trials with cognitive-behavioral therapies for PTSD. The authors concluded that dropout rates were often high and varied from one study to another.

According to the meta-analysis by Bradley and colleagues (2005), the dropout rate was in average 20% across all therapies for PTSD. This finding is similar to overall dropout rate of 18% reported by Imel, Laska, Jakupcak, and Simpson (2013) across various PTSD therapies.

In a review of 55 studies, dropout rates of therapies for PTSD were examined by Schottenbauer, Glass, Arnkoff, Tendick, and Gray (2008). The dropout rate ranged widely and seemed to have depended in part on the sample.

Similarly, Imel et al. (2013) examined 42 studies of PTSD treatments. They reported an average dropout rate of 18%. Differences in trauma focus between treatments in the same study did not predict dropout. However, a higher dropout rate (36%) was found in trauma-focused therapies (PE and EMDR) in comparison to present-centered therapy (22%). Interestingly, dropout rates vary greatly across studies but not within studies. Therefore, differences in dropout were driven by differences between studies. The results of Imel et al. (2013) point out toward the presence of experimenter bias in controlled clinical trials.

In a review of 25 controlled clinical trials for PTSD involving cognitive-behavioral therapies for PTSD, Hembree, Foa, Dorfan, Street, Kowalski, and Tu (2003) found no difference in dropout rates across PE, EMDR, cognitive therapy, and anxiety management. In a meta-analysis of cognitive-behavioral therapy for PTSD, Barrera et al. (2013) also found that the dropout rate was higher when a trauma-focused component, as in CPT, was involved (26%) in comparison to cognitive-behavioral therapy without such a component (19%).

With respect to dynamic therapy for PTSD, Brom et al. (1989) reported an equal dropout rate across all therapies: BDT as Horowitz's model (11%), trauma desensitization as systematic desensitization (11%), hypnosis as PE (11%). In the pre-post study by Krupnick (1980), 13% dropped out of group dynamic therapy. In a retrospective study examining the effectiveness of dynamic integrative therapy in a naturalistic setting for 100 files randomly selected (Brunet, 2004), no patient (0%) terminated therapy prematurely.

Attrition matters, empirically and clinically. Attrition/dropout rates tend to be high in controlled clinical trials of therapies for treating PTSD, even though participants have been highly selected by researchers and many applicants chose to not participate after treatments and procedures have been explained to them. In general, at least one participant out of five drops out, 20% (1 out of 5). In trauma-focused therapies, participants drop out more often (36%; 1 out of 3) than in supportive therapy (22%) (Imel et al., 2013).

Furthermore, statistical analyses of efficacy are often computed with the data of completers only. When dropout data are included, differences in efficacy often vanish between 'active' therapies and 'placebo' therapies, especially at follow-up (Bradley et al., 2005). The non-consideration of dropout data in statistical analyses thus creates a favorable bias in favor of therapies associated with higher dropout rates, as demonstrated by Bradley et al. (2005). The higher attrition in trauma-focused therapy suggests restricted applicability and tolerability

of trauma-focused therapies for treating PTSD.

Negative Side Effects

Therapies for PTSD, as any therapies, can produce negative side effects, called iatrogenic effects. Given the neurobiological and psychological vulnerability of individuals developing PTSD in its severe form, iatrogenic effects are at times severe when trauma-focused techniques are employed. These techniques ask participants to intensely relive face what they spend most of their energy avoiding, that is, traumatic memories. In the literature, reports of negative side effects are parsimonious since the 1990s, but such scarcity does not mean that they do not happen in clinical practice.

Severe negative side effects happen as reported by clinicians to clinicians and participants to new clinicians or readers of internet blogs. In an article reviewing the history of side effects in psychological therapies, Barlow (2010) invites clinicians and researchers to take a closer look at iatrogenic effects. We could agree.

As practitioners, we all make mistakes. Hopefully, we get more training and supervision in response to our mistakes. Hopefully, we learn from our mistakes and we correct our assumptions and interventions. As previously mentioned, PTSD experts and general practitioners report that highly trauma-focused techniques such as PE are rarely employed in clinical practice due to attrition and negative side effects (Becker et al., 2004; van Minnen et al., 2010). Therefore, let's look more closely at the negative side effects (iatrogenic effects) of trauma-focused therapies.

Prolonged exposure. Proponents of PE suggest that negative side effects associated with their treatment of choice are quasi-inexistent. Foa et al. (2002) reported that only a minority of PE participants of controlled clinical trials exhibited a reliable exacerbation of PTSD symptoms (Foa has been the leading proponent of PE for treating PTSD for over 20 years). Van Minnen et al. (2002) even claimed that PE can be used safely with individuals presenting a dissociative disorder, suicidal behaviors, substance abuse, major depression, substance abuse, borderline personality disorder, and even psychosis.

Another study examined the attrition, efficacy, and incidence of PTSD worsening in three treatments: PE, EMDR, and relaxation (Taylor, Thordarson, Maxfield, Lovell, & , 2003). These therapies did not differ in attrition rates or incidences of symptom worsening (0 case in PE and EMDR, and 1 case in relaxation). Prior to treatment, a serious process of selection by researchers and self-exclusion by applicants had taken place. A total of 299 prospective participants contacted the clinic and received a telephone screening interview. Of these, 164 passed the interview and were invited to the clinic. Only 60 participants met the inclusion-exclusion criteria and entered treatment, and of those, only 45 participants completed treatment. So, only 20% of applicants entered therapy and only 75 % completed treatment, that is, 15% of applicants. Such selection process and attrition rate seriously limit the generalizability of these results to clinical practice.

Negative side effects have been reported in clinical trials. Pitman et al. (1991) reported that 30% of PE participants developed a variety of negative side effects: depression, suicidal ideation, drug/alcohol relapses, panic attacks, and premature termination. Schnurr et al. (2007) reported adverse side effects were reported: 5 serious adverse events in PE (four psychiatric hospitalizations and one suicidal attempt) and 14 in present-centered therapy (two non-suicidal deaths, nine psychiatric hospitalizations, and three suicidal attempts). These events were regarded as not being related to the therapies, except for the suicidal attempt in PE. Tarrier, Summerfield, Faragher, Reynolds, Graham, & Barrowclough (1999) reported that a greater number of patients who showed PTSD worsening received PE, while such iatrogenic effects were not present in cognitive therapy.

Litz et al. (1990) surveyed numerous psychotherapists regarding the contra-indications for using PE with PTSD. The list included: substance abuse, a history of impulsivity, concurrent stressors, previous failure in using PE, a history of non-adherence to treatments, a recent application for financial compensation, a difficulty to use imagery, lack of reexperience symptoms, an incapacity to tolerate intense arousal, a history of psychiatric disorders, the presence of co-morbid disorders, and the presence of cardiac problems. Allen & Bloom (1994) added other contra-indications: a marked psychological dysfunction, the presence of a personality disorder, suicidal ideations, impulsivity, substance abuse, and resistance to treatment.

Meichenbaum (1994) added that the person needs to be willing to be a collaborator, PTSD symptoms need to be stabilized as well as the living conditions of the person, and the person's self-esteem needs to have been restored. Kilpatrick and Best (1984) caution against treating victims of sexual abuse with techniques involving prolonged exposure.

Beyond clinicians' and researchers' reports, the experiences of patients themselves are of importance. Here is the writing of an ex-Marine, David J. Morris, about his very difficult experience with PE (http://opinionator.blogs.nytimes.com/2015/01/17/after-ptsd-more-trauma/?hpw&rref=opinion&action=click&pgtype=Homepage&module=well-region&region=bottom-well&WT.nav=bottom-well&_r=1).

"My therapist instructed me to select a traumatic event to focus on. As someone who had spent 10 months in some of the deadliest parts of Iraq, I had collected a number of near-death experiences. Would I choose the time I rode in a helicopter that was nearly shot down over Falluja? The I.E.D. ambush I saw near the town of Karma that killed two Pennsylvania National Guardsmen? The week I spent with some Marines from my old battalion when we were shelled for seven days straight? To focus on a single event seemed absurd, the equivalent of fast-forwarding to a single scene in an action film and judging the entire movie based on that.

In the end I chose the story of the I.E.D. ambush I survived in 2007 in southern Baghdad. Over the course of our sessions, my therapist had me tell the story of the ambush dozens of times. I would close my eyes and put myself back inside the Humvee with the patrol from the Army's First Infantry Division, back

inside my body armor, back inside the sound of the I.E.D.s going off, back inside the cave of smoke that threatened to envelop us all forever.

It was a difficult, emotionally draining scene to revisit. This was the work site of prolonged exposure therapy, where the heart's truest labor was supposed to happen. Given enough time and enough story "reps," when I opened my eyes again, I wouldn't feel forever perched on the precipice of a smoke-wreathed eternity. I wouldn't feel scared anymore.

But after a month of therapy, I began to have problems. When I think back on that time, the word that comes to mind is "nausea." I felt sick inside, the blood hot in my veins. Never a good sleeper, I became an insomniac of the highest order. I couldn't read, let alone write. I laced up my sneakers and went for a run around my neighborhood, hoping for release in some roadwork; after a couple of blocks, my calves seized up. It was like my body was at war with itself. One day, my cellphone failed to dial out and I stabbed it repeatedly with a stainless steel knife until I bent the blade 90 degrees.

When I mentioned all this to my therapist, he seemed unsurprised. "You weren't drunk at the time?" he asked. "No. That came later." Following a heated discussion, in which I declared the therapy "insane and dangerous" and my therapist ardently defended it, we decided to call it quits. Before I left, he admonished me: "P.E. has worked for many, many people, so I would be careful about saying that it doesn't work just because it didn't work for you." (this paragraph is particularly worrisome; the present author).

Within a few weeks, my body returned to normal. My agitation subsided to the lower, simmering level it had been at before I went to the V.A. I began once more to sleep, read and write. I never spoke about the I.E.D. attack again.

… My own disappointment is that after waiting three months, after completing endless forms, I was offered an overhyped therapy built on the premise that the best way to escape the aftereffects of hell was to go through hell again.

A month after dropping out of prolonged exposure therapy, I began a treatment of cognitive processing therapy at the V.A. Here, our group was asked to examine our thoughts and feelings about our war experiences without revisiting specific traumas. We were allowed to let sleeping dogs lie. This has helped. As I wrote in my journal at the time, "If P.E. is a kind of emotional chemo, then C.P.T. is a kind of emotional tai chi."

Eye movement desensitization and reprocessing. Let's first acknowledge the position of the EMDR Institute (2015) regarding the effects of EMDR. On the website of the EMDR Institute, it is written: 'As with any form of psychotherapy, there may be a temporary increase in distress: (1) distressing and unresolved memories may emerge; (2) some clients may experience reactions during a treatment session that neither they nor the administrating clinician may have anticipated, including a high level of emotion or physical sensation; and (3), subsequent to the treatment session, the processing of incidents/material may continue, and other dreams, memories, feelings, etc., may emerge.'

Although few side effects have been published (Brunet, 2002; Kaplan & Manicavasagar, 1998), those published indicate that EMDR can provoke severe to extreme side effects. Brunet

(2002) described how a veteran with severe PSD entered a homicidal dissociative episode in the second EMDR session. Despite the numerous attempts by the therapist to calm the patient, the patient withdrew within himself and started to become very agitated, behaving as if he was torture anew. The patient then became acutely homicidal toward the therapist and remained for days. The patient needed to be restrained for days at the psychiatric ward for days and under a sedative.

Because of the popularity of EMDR, it is possible to find ample evidence of its severe side effects on personal blogs on the internet. Here are few examples found on google.com on November 24th, 2014, on the first page of results after entering 'EMDR side effects'.

''So... Yesterday I went for my first EMDR therapy treatment. We made a list of my "Top Ten Traumas" before hand and she told me to put stars around the top two most traumatic events so we could focus more on these. Before I even left to drive to the session, I had worked myself up into a full blown anxiety attack. I was pacing around the house, heart rate through the roof, breathing heavy, mind racing, quickly getting worse and worse. I don't know why all of a sudden I was so freaked out about this therapy because when we previously discussed it I had no problems or concerns doing it.

Anyways, I got there in my panicked frenzy and my therapist suggested that maybe we start off with something a little less severe to start since I was so frazzled. She didn't want me to feel worse than I already felt, not to mention I had a migraine working its way into my brain.

The session seemed to run smoothly and I could feel my progress. The heartache and pain i was feeling in my chest when we first started the session subsided quickly and was completely gone once we finished. My head felt a little cloudy, but I didn't feel like I wanted to curl up into a ball and cry myself to death anymore.

I went home and laid on the couch for a few hours to "just veg" as she suggested. I felt light headed yet my brain felt heavy and "sloshy" in my head. My eyes started to droop and I decided to go to bed (I haven't slept in almost three weeks, so the fact that I felt tired was a GREAT relief) and I fell asleep. Not into a deep sleep, since I know I was awake many times throughout the night, but it was sleep none the less. This is when the nightmares started.

One after the other after the other. I had numerous TERRIFYING nightmares about the apocale, being possessed by a demon, earthquakes, trains on fire full of people screaming to get out, running away from terrible things etc. I NEVER have nightmares, and if I do they do not scare me. I am a huge fan of horror films and nightmares have always excited me. These did NOT excite me. I woke up this morning trembling and dizzy and hysterical.

I turned on the light, ran upstairs crying and almost fell over. My head felt like it weighed more than I did. I felt terror coursing through my whole body. I felt afraid and scared. The nightmares were so REAL. I had to turn all the lights on in my house and open all the curtains because I was afraid of the dark. I was afraid to go back down into my room because it was dark down there. Even when i went into the kitchen throughout the day to get something from the fridge, the thought of even looking towards the top of the stairs sent me into a panic.

All day I have cried. I was in hysterics so badly that my boyfriend left work to come over and check on me. I have never felt so out of control in my whole life. I do not feel like myself. I dont feel like I am even in my own body.

So, the point of this post is to ask if anyone has done EMDR and has had adverse like this. Does it get better? Do you think it will get worse with other treatments? This wasnt even my worst trauma, will it be worse with treating my more severe ones? I did a bunch of research online about the side effects and found a bunch of people stating that it did not multiple traumas. I do not want to live feeling like this, it is unbearable. My … is worsening and I cannot turn my lights off or my heart starts to race and i start to lose it. Oh, not to mention I seem to be getting brain "shocks" or nervous system "ticks" where my head kind of shakes back and forth and sometimes even jerks to the left. Loud noises give me anxiety and hurts my ears. I tried to unload the dish washer this afternoon and the sound of plates clanging together was too much for me to handle. Ugh, hopefully someone reads this, sorry it is so long, ladydawn' (November 24th, 2014; www.healthboards.com/boards/post-traumatic-stress-disorder-ptsd/934679-emdr-side-effects.html).

'Many people have asked about EMDR, so I thought I would share part of what is going on with me.. and it's due to that aweful treatment. I've been triggered and triggered and triggered these past few weeks. I am out of it. and I know it. Anyways, I did around three ? sessions of EMDR last summer. I have multiple traumas stemming from childhood to last year. I did not know when I did the treatment that it is not recommended for someone with numerous traumas. After the first three sessions, I snapped and had to be medicated (I still am medicated now.) The first week or so after that I kept getting this horrid "things." My traumas (not all but way more than I could handle) would flash like a picture book through my mind. I would have my eyes open and the whole room was flashing as if a bulb was going off.. then I would go off the deep end and get violently ill for about a week. Since then I have noticed everytime I get triggered, I have this weird "thing" happen to me. It is not a flashback. It is like the EMDR is burned into my brain.. and it won't stop. It's painful and gives me migraines and my body shuts down.. not to mention the horrid anxiety this event causes. I have no idea if this will ever go away at the present, nor what is causing it. I have no one to ask because this area sucks. I worry that it has damaged me. I just thought that for those of you considering this treatment.. consider this. I don't want anyone else to be stuck where I am from crappy information and a therapist that is too pushy for something that is very dangerous. bec'' (November 24th, 2014; www.myptsd.com/c/threads/emdr-lashback-when-emdr-goes-wrong.1388/

'I had a first session last week and ever since then I just feel dead. I can't feel anything. Nothing. I am a shell, harboring nothing.Trigger- Self harm. I cut myself last night to see if I could feel and I couldn't. I felt nothing. I realize the risk in harming oneself when one cannot feel, so I have chose to not do it for the time being. I can't feel anything. I am so dead inside it makes me sick. Using the word "numb" is an understatement. I am dead. It's like I don't even exist anymore. I've always experienced this in some way, but since the session I have been very bad.' (November 24th, 2014; http://forums.psychcentral.com/psychotherapy/207530-does-emdr-have-side-effects.html).

According to personal clinical experience, EMDR causes severe adverse effects and such effects are not common in psychotherapy. During and after the use of use of EMDR, we have observed the following reactions: a manic flight of ideas unseen beforehand, statements repeated in a loop and leading nowhere (i.e. 'I don't know!'), painful somatic sensations (including sharp bodily pains, migraines and nauseas), and intense anger toward the technique

of EMDR. These reactions have led to our cessation of the EMDR technique in a prophylactic stance in an effort to prevent more iatrogenic effects. Many psychotherapists reported to the present authors that they had received individuals in their practice who has been severely destabilized by EMDR. Severe iatrogenic effects reported by clinicians were: psychotic episodes, suicidal attempts, self-mutilations, alcohol and drug relapses, panic attacks, etc. When patients had reported these damaging side effects, the 'EMDR therapist' responded with statements implying that these were due to the patient and not to the technique; EMDR provoked only side effects such as more traumatic reminiscence afterward. Caution is thus highly suggested with respect to the use of EMDR.

Cognitive processing therapy and brief dynamic therapy. Negative side effects are not reported for CPT or BDT. The exposure component of CPT consists of writing a description of the traumatic event while reliving the emotions then experienced, while allows latitude to patients to engage emotionally to a degree found tolerable to them. With respect to brief dynamic therapy, Horowitz & Kaltreider (1979) were clear that only well-functioning adults with a single-episode PTSD should undergo BDT. Moreover, therapists should always proceed at tolerable dosage and withdraw any exploration of the traumatic event as soon as the patient shows signs of becoming overwhelmed. Nonetheless, it is reasonable to assume that CPT and BDT also carry risks of provoking side effects. Such iatrogenic effects should be investigated and reported.

Lack of Long-Term Maintenance

Maintenance of therapeutic effects is central to the welfare of patients. Therapeutic gains should be maintained over months and years. This is especially true if proponents of a given therapy claim that their technique induces a process of extinction/habituation or a process of integration/assimilation of the traumatic memories.

Nonetheless, the maintenance of therapeutic effects across therapies for PTSD is mostly unknown beyond brief intervals (Bradley, 2005). Follow-up data is usually available at 3-month, 6-month or 12-months. According to Bradley et al. (2005), 54% of completers were judged to be clinically improved, but only 32% met research criteria for clinical improvement. Across therapies, trauma-focused or not, clinical improvements decreased from 54% to 32%, indicating a decay of therapeutic effects over time. Now let's examine maintenance of effects for trauma-focused therapies only.

In their meta-analysis, Powers et al. (2010) found that the effect size of PE had decreased from 1.08 at post-test to 0.68 at follow-up (these effect sizes were calculated in comparison to 'psychological placebos'). Despite such findings, Powers et al. (2010) concluded that 'PE is a highly effective treatment for PTSD, resulting in substantial treatment gains that are maintained over time.' Such conclusion is unwarranted given that the PE effect size dropped from 1.08 to 0.68 in few months after termination.

Only one controlled clinical trial examined the effect of any psychological therapy for PTSD over many years. Macklin, Metsger, Lasko, et al. (2000) assessed the maintenance of EMDR effects after 5 years in veterans with chronic PTSD. At post-test, a modest effect of EMDR had been observed (d = 0.28). At a follow-up, 5 years after EMDR, PTSD severity was similarly deteriorated in the EMDR condition (d = -.82) as in the untreated control group (d = -.83). In previously improved participants, symptoms had not only reemerged, but they had deteriorated in both EMDR and controls conditions. These results suggest that PTSD remitters had regained PTSD 5 years after EMDR as if PTSD had been left untreated. *

** Such complete decay of effect has been also experienced by the author of this article following the practice of EMDR during her EMDR training in 1995 with Francine Shapiro. Ten years later, the same dysphoria reemerged upon remembering a specific childhood moment. In addition, some individuals consulting the clinic of the present author report that they had gone through EMDR therapy, resulting in a decrease of PTSD, but only to regain a PTSD 'worse than ever'.*

Although the EMDR Institute (2015) interprets the finding of Macklin et al. (2000) as being due to a lack of providing of a complete EMDR therapy, we consider that 10 weekly sessions of EMDR represent an adequate dosage given that 3 to 12 sessions are recommended by the EMDR Institute. Such large PTSD relapse in EMDR participants highlights a lack of maintenance of therapeutic effects and, therefore, a lack of integration of traumatic memories. Such results should be worrisome to all clinicians, including therapists employing EMDR.

Long-term follow-up data over years is unfortunately unavailable for PE, CPT, and BDT. The lack of data regarding long-term maintenance of therapeutic gains with PTSD brings us to consider the data reported for other serious mental disorders.

According to Shedler (2010), a major concern with cognitive-behavioral therapies is the lack of maintenance of therapeutic effects over prolonged periods of time. In 5 meta-analyses, the efficacy of cognitive-behavioral therapies was shown to decay over the following months and years after termination. In contrast, the efficacy of dynamic therapy was shown to increase over months and years after termination. These meta-analytic findings do not apply directly to PTSD because controlled clinical trials including dynamic therapy for PTSD are more or less absent from the literature, with the exception of the study of Brom et al. (1989).

Limited Implementation in Clinical Practice

Some clinicians and researchers invite practitioners to be prudent whenever the use of a trauma-focused therapy is considered with a PTSD sufferer, especially techniques enforcing a multi-modal traumatic reexperiencing at the beginning of therapy (PE and EMDR). Many patients with PTSD do not wish to relive their traumatic experiences. Many therapists consider that most PTSD sufferers do not have the capacities to utilize such trauma-focused techniques.

To verify the applicability and acceptability of PE, Scott and Stradling (1997) invited patients to participate in prolonged exposure therapy in a clinical setting. They found that 43%

of patients refused to participate in an exposure-based treatment. Compliance with PE was related to initial severity of PTSD and depressive symptomatology; the less symptomatic patients were, the more likely they would agree to participate in exposure-based therapy. These authors concluded that PE therapy was not a treatment of choice for many PTSD patients.

Many proponents insist on the usefulness of PE and EMDR for treating PTSD, However, general practitioners and PTSD experts do not agree with these academic conclusions, especially regarding PE. Indeed, according to two surveys (Becker, Zayfert, & Anderson, 2004; van Minnen, Hendriks, & Olff, 2010), most clinicians do not use PE for treating PTSD for two main reasons: they are concerned of provoking negative side effects and attrition.

In a large survey, only 4% of psychologists reported using PE to treat PTSD (Becker, et al., 2004). Even psychologists with strong interest and training in behavioral treatments for PTSD perceived PE as comprising significant barriers against its implementation such as possible negative side effects and large dropout rates (Becker, et al., 2004).

Other advocates of prolonged exposure investigated when and why PTSD experts chose to use PE (van Minnen et al., 2010). These authors reported that most experts would not utilize PE for treating PTSD, but no percentage was provided (most likely this percentage is very low). PE was particularly least preferred by PTSD experts when depression was comorbid and experts would be more likely to offer PE as treatment if patients expressed a preference for a trauma-focused technique (van Minnen et al., 2010).

Nonetheless, van Minnen et al. (2010) concluded that PE was simply underutilized by PTSD experts. These authors argued that PTSD experts should be further trained in using PE. However, experts stated valid reasons for not using this potentially destabilizing technique and they did not want to risk exacerbating PTSD and increasing the probability of seeing patients abandon therapy. While PE advocates consider that PE, a highly trauma-focused technique is underutilized by psychologists, PTSD experts view PE as potentially destabilizing to the point of almost never using it. This contrast is of particular interest because academicians are promoting the use of PE for treating all PTSDs. The therapeutic choice of PTSD experts is congruent with the basic contention of this article.

In contrast to PE, EMDR is now a very popular technique among practitioners. EMDR has been repeatedly popularized in the media. Over 20,000 therapists have now been trained in using EMDR for PTSD and other mental disorders (EMDR Institute, 2015). However, no survey is known about the use of EMDR in clinical practice for treating PTSD.

Regarding the implementation of Cognitive Processing Therapy, a survey was conducted by CPT advocates regarding its use with veterans after training (Chard, Ricksecker, Healy, Karlin, & Resick, 2012). In one survey, 642 clinicians were contacted and 34% responded. In a follow-up survey of 1107 clinicians, 541 completed the survey. However, Chard et al. (2012) did not provide data pertaining to the percentage of clinicians using CPT in their practice. Instead, some

clinical data was provided about patients' status, including the number of patients treated with CPT (n = 374). Unfortunately, it was not reported how many clinicians use CPT to treat PTSD after their training, that is, how many clinicians endorse CPT as a therapy of choice for PTSD.

No survey is known to the author regarding the use of BDT in treating PTSD. However, the implementation of BDT in clinical practice might be limited more by the extensive training required on the part of psychotherapists than by a lack of engagement by participants, especially if the dynamic is integrative. For example, in the study of Burnet (2004), no dropout was found in the 100 files randomly selected at the specialized clinic where a dynamic integrative model (Gaston, 1995, 2014, 2014) is employed. Dynamic psychotherapy cannot be learned in one week as PE or few week-ends as EMDR. At the above mentioned clinic, two years are dedicated to training already experienced psychotherapists (at least 5 years of therapy experience plus 2 years of supervision post-diploma).

Predictors of Change

About half of participants of controlled clinical trials of PTSD therapies seem to obtain a symptomatic reduction from trauma-focused techniques as well as other therapies such as present-focused therapies. These patients are likely to present with neurobiological and psychological structures allowing for arousal-modulation and affect-regulation. Such structures need to be already well-developed and readily available for de-activating trauma-based fears or other affective responses. Otherwise, it is our contention that highly trauma-focused techniques (PE and EMDR) will induce defense mechanisms such as repression or dissociation in order to produce anxiety reduction and PTSD decreases. It is also our contention that the gently trauma-focused therapies (CPT and BDT) will be efficacious through the provision of a new perspective and a therapeutic relationship.

Practitioners experienced in treating PTSD know that individuals with good-enough capacities for affect-regulation are not likely to develop PTSD unless a traumatic event is particularly violent. Such individuals present with non-severe PTSD, a condition closer to a phobia than a disorder involving the whole person. These well-structured individuals present with few PTSD symptoms, of mild to moderate severity, and co-morbid disorders are absent.

In support to this contention, pre-therapy PTSD severity has been found to be the major predictor of outcome in trauma-focused therapies such as PE (van Minnen et al., 2002) and in gently trauma-focused therapies such as BDT (Horowitz et al., 1986).

In another study, van Minnen and Hagenaars (2002) indicated that habituation (as reflected by a decrease of subjective distress) in the initial PE session was the only predictor of PTSD reduction. Habituation response indicated by reductions of subjective distress reflects a participant's capacity to modulate arousal and affects.

In line with these findings, a neuroimagery study conducted with patients treated by open-ended dynamic integrative therapy (Dickie, Brunet, Akerib, & Armory, 2013) examined the contribution of a neural site involved in affect-modulation. The thickness of the prefrontal anterior cingulate cortex (ACC) is thought to reflect a structural capacity, namely the capacity to modulate arousal and affects. The thickness of the ACC was found to be associated with the initial severity of PTSD and to predict PTSD reductions in a dynamic integrative psychotherapy (Dickie et al., 2013). A neurologically-based capacity for affect-modulation thus influences how patients respond to psychotherapy.

When personality disorders are considered, PTSD improvements vary in therapy. Hembree, Cahill, and Foa (2004) found that, in PE cohorts, personality disorders were present in 39% of the sample, mostly borderline personality disorder. At the end of treatment, no difference in PTSD improvements was found between personality-disordered participants and not personality-disordered. However, those with a personality disorder were less likely to attain good end-state functioning. Such findings may be partly explained by the fact that PTSD and BPD share many features: greater symptoms of anxiety, anger, avoidance, impulsivity, and suicidality (Giardino, 2009). Such findings are also in line the theory of Masterson (1985) about BPD wherein such individuals are viewed as being better equipped to modulate affects than they appear to be both behaviorally and psychologically whenever they are willing to self-activate (such as fully participating in controlled clinical trial).

In a literature review, Vignarajah and Links (2009) examined studies including patients with PTSD and BPD. Not only were treatments efficacious for reducing PTSD, but effective treatments for one condition appeared to provide some symptom relief in the other. Vignarajah and Links (2009) concluded that, despite promising results of alleviation of certain BPD features when treating for PTSD, treatment of PTSD without the consideration of BPD and related suicidal behavior may be detrimental to patients. These dual treatment effects call into question the validity of the PTSD and BDP diagnoses in these studies Vignarajah and Links (2009). Finally, in some studies (Feeny, Zoellner, & Foa, 2002; Hembree, Cahill, & Foa, 2004; McDonagh, Friedman, McHugo, et al., 2005), patients presenting with PTSD and BPD showed less therapeutic improvements and were more likely to drop out than participants presenting with PTSD only.

The efficacy of a trauma-focused technique such as PE seems to be affected by the presence of anger. Foa, Riggs, Massie, and Yarczower (1995) explored the influence of anger versus fear on PTSD improvements after PE. The more intense were the facial reactions of fear during exposure, the more patients benefitted from PE. Inversely, the more participants reported anger before treatment, less fear reactions were exhibited during exposure and more severe was PTSD at termination. Therefore, PE may be an appropriate therapeutic strategy for fear-based PTSD (similar to a phobia), but PE is ineffective for anger-based PTSD. Clinicians need to differentiate fear-based PTSD from anger-based PTSD.

In general, patients presenting with a milder form of PTSD possess a greater capacity for self-soothing, habituation, and affect-modulation. They are good candidates for any brief

therapy, even trauma-focused techniques. During their childhood, these individuals mostly experienced positive relationships. Their reasoning capacities under stress remain functional. In sum, these individuals are well-equipped for establishing a connection between their higher cortical structures of affect-modulation and trauma-based affective responses. Such connection may allow for integration to occur which dissipates traumatic responses and PTSD symptoms. These individuals may benefit from highly trauma-focused therapies, but they would also benefit from gentler forms of PTSD therapies such as anxiety management (SIT) or brief dynamic therapy (BDT).

Taken together, these findings reflect a well-known therapeutic phenomenon, 'the rich get richer, the poor get poorer'. An empirical examination of the clinical effectiveness of PTSD therapies, rather than their efficacy in controlled clinical trials, should matter to practitioners in order to adequately assess a treatment of choice.

Processes of change

In cognitive-behavioral techniques with a trauma-focused ingredient, the goal is usually to permanently associate an antagonistic response with traumatic memories, that is, to desensitize the trauma-related affective response through habituation (PE) or divided attention (EMDR). It is inferred that traumatic memories will, therefore, remain de-activated permanently, that is, extinguished. In addition, EMDR proponents suggest that this highly trauma-focused technique induces a process of integration of the traumatic memories, that is, the pre-existing psychological structure is accommodated to assimilate the traumatic information, rendering the latter non-disturbing. CPT has been proposed to induce an assimilation process by altering participants' core trauma-related negative beliefs. BDT has been developed by Horowitz (1986) to foster an accommodation of the pre-existing psychological to allow for an integration of the traumatic information.

Prolonged exposure. In PE, the proposed change process is a desensitization of the anxiety associated with the traumatic memories through habituation/extinction of fear responses. Exposure provides these two conditions: eliciting the fear response and providing an opportunity for corrective information, which should lead to habituation (Foa & Meadows, 1997). Foa and Riggs (1993) also consider that PE reorganizes traumatic memories and corrects maladaptive schemas. The decrease of anxiety would permit a re-evaluation of the meanings associated with traumatic memories.

Jaycox, Foa, and Morral (1998) examined the role of habituation toward PTSD improvements in PE. They found that participants displaying high initial levels of engagement and gradual habituation between sessions improved more in PE than participants with high or moderate initial engagement without habituation. Habituation was determined by a decrease in subjective distress.

In support of habituation as the change process in PE, van Minnen and Hagenaars (2002) also reported that participants benefitting from PE showed within-session habituation

and between-session habituation, in terms of decreased subjective anxiety. For improved participants, anxiety decreased within the first session and from the first session to the last. In contrast, non-improved participants showed no signs of anxiety reduction in the first session, even after controlling for initial level of PTSD. Non-improved patients also had higher ratings of anxiety initially, reflecting a higher baseline of anxiety and therefore a more limited capacity for habituation and affect-modulation. These findings highlight the necessity to continue PE after the initial session only if participants report significant decreases in subjective distress.

With heart rate reductions as indication of habituation in PE, findings are not so supportive. In a study by Pitman et al. (1996), heart rate activation, heart rate habituation within sessions, and partial heart rate habituation across sessions were obtained in PE. However, only modest overall PTSD improvements were found. Pitman et al. (1996) concluded that there was limited support for the notion that mobilization of arousal during PE predicts improvement.

Eye movement desensitization and reprocessing. With respect to EMDR, this technique was developed without a theoretical framework. Shapiro (1995) suggests that EMDR induces a therapeutic process of desensitization and/or integration. However, in one study (Macklin et al., 2000), PTSD relapse was found in all improved participants at a 5-year follow-up after PTSD improvements were obtained at post-test. PTSD was similarly deteriorated in the EMDR condition (d = -.82) as in the untreated control group (d = -.83). In improved or remitted EMDR participants, symptoms had not only reemerged, but they had deteriorated. Even though these findings pertain to a small sample, such findings should be highly worrisome to clinicians. These results also counter any inference regarding desensitization or integration of traumatic memories as change process induced by EMDR.

Processes of desensitization or integration would not lead to a resurgence of traumatic memories and PTSD deterioration years later. These findings cannot be overlooked or dismissed because it is just one study. The study of Macklin et al. (2000) is the only one having examined whether EMDR effects withstand the test of time over years. Maintenance of therapeutic effects is paramount to both patients and practitioners. PTSD relapse in all improved participants in EMDR suggest that EMDR might induce a mechanism of dissociation given its dual task.*

** At her EMDR training, the present author has used a dysphoric childhood memory as target event. This event was distressing but not intrusive. After few sets of eye movements, the memory suddenly became non-significant and slight euphoria was present. Some would say that EMDR worked well. However, 10 years later, the same memory resurfaced unaltered, with all its dysphoric affects. Individuals consulting at the clinic of the present author have reported a full-blown resurgence of PTSD after several weeks or months, and an even deteriorated PTSD as in the Macklin et al. study (2000).*

Lanius et al. (2010) described dissociation as a consequence to being confronted with an overwhelming experience from which no escape is possible, an experience which challenges the

individual to find an escape from the external environment as well as their internal distress and arousal. For some EMDR participants, such condition might just well be the reexperiencing their worst traumatic moment at the beginning of this technique, even though they are willing to do so. Human beings are at times willing to push themselves beyond reasonable limits and individuals with PTSD are at times so desperate to get rid of this condition that they are willing to try any technique, even highly anxiety-provoking techniques, especially if such technique is praised in the medias.*

** Individuals with PTSD are at times willing to try almost anything to get rid of their PTSD and to prove to themselves that they are not traumatized. Some patients of the present author, presenting with severe PTSD and a personality geared toward functioning at all costs, were exposing themselves to trauma-related conditions prior to therapy. Victims of assault in public places, these persons went back regularly to public places, such as shopping malls, to sit down and wait until they calm down, which never happened. They persevered in staying in the public place even though they had panic attacks, one after the other. In therapy, these persons were adamant that they had to face their fears. Such self-imposed in vivo exposure had to be addressed in psychotherapy in terms of its detrimental effects on the brain, the body, and the psychological structure of the person. They usually stopped exposing themselves in this way only to protect their brain, not themselves. This was a therapeutic beginning which later led to the consideration of themselves as being valuable of benevolence. Therapeutic work cannot proceed in panic or in very high anxiety (see Yerkes-Dodson Law); otherwise, something else takes place.*

In support of EMDR inducing a dissociative state, Davidson and Parker (2001) reported a spectacular effect size of 2.71 on the reduction of subjective distress from EMDR. Such reduction of subjective distress, associated with eye movements (or any other dual task), parallels how patients with dissociative tendencies suddenly feel in psychotherapy when dissociation is triggered. In the same line, little association between emotional processing and PTSD reductions was found in EMDR (Pitman, Orr, Altman, Longpre, Poiré, & Macklin, 1996). To further explore the idea that EMDR might induce dissociation, let's look at a physiological marker of dissociation.

Individuals with PTSD and a dissociative tendency show a paradoxical response in terms of their heart rate response following an exposure to their traumatic memories. Dissociative individuals with PTSD present a sudden heart rate reduction upon exposure (Griffin, Resick, & Mechanic, 1997; Sack, Cilien, & Hopper,2012; Werner & Griffin, 2012) in contrast to usual PTSD sufferers who present a substantial heart rate increase. Therefore, it is important to examine heart rate changes during the EMDR dual task in order to understand whether EMDR induces dissociation as change process.

One study precisely examined the immediate effects of the EMDR dual task (EM or eye movements) on heart rate (Schubert et al., 2011). Participants were students experiencing distress following a stressor, not PTSD. The experimental conditions involved regular EM or varying EM. The control condition involved simply keeping one's eyes closed. In the first 10 seconds, heart rate reductions were observed with EM, while controls showed no changes in

heart rate. Over the course of a single session, heart rate of control subjects decreased gradually rather than suddenly. At the end, all subjects showed equivalent reductions in heart rate and equivalent reductions in subjective distress. The sudden reduction of heart rate associated with EM is similar to the paradoxical cardiac response of dissociative individuals with PTSD when they are exposed to their traumatic scenario (Griffin et al., 1997; Sack et al., 2012; Werner & Griffin, 2012).

Consequently, it is reasonable to infer that, for individuals with PTSD, the dual task of EMDR demanding a divided attention, could induce a dissociative mechanism, which would explain the sudden and large decreases of subjective distress and the sudden heart rate decreases within seconds in some participants. Some PTSD improvements following EMDR could thus be due to the induction of a dissociative mechanism in participants. This hypothesis needs to be tested with individuals presenting with PTSD to verify whether they show sudden and marked heart rate reductions during the EMDR dual task and if these heart rate reductions predict PTSD improvements.

Cognitive processing therapy. Proponents of CPT suggest that this therapy induces cognitive restructuring, which would allow for the assimilation of traumatic memories (Resick & & Schnicke, 1992).

In line with this hypothesis, a study showed that CPT reduced guilt-related thoughts (Resick, Nishith, Weaver, Astin, & Feuer, 2002). CPT completers also showed significant reductions in cognitive distortions pertaining to safety, trust, power, control, esteem, intimacy, and undoing (Owens, Pike, & Chard, 2001). CPT reduced PTSD symptoms, guilt, shame, and overall cognitive distortions (Resick, Galovski, O'Brien et al., 2008). The percentage of accommodated thoughts was found to be negatively correlated with PTSD symptoms (Sobel, Resick, & Rabalais, 2009). These results are in line with the change process suggested by Resick & Schnicke (1993).

However, one study might suggest that the induction of dissociation is also at play in CPT. The predictive role of dissociative tendency was investigated in a controlled clinical trial designed to compare CPT with its exposure component, CPT without an exposure component and written trauma accounts only (Resick, Suvak, Johnides, Mitchell, & Iverson, 2012). Pretreatment levels of dissociation impacted treatment conditions differently. Participants with low dissociative tendency responded most efficiently to CPT without its exposure component, that is, cognitive therapy. In contrast, participants with high dissociative tendency responded better to CPT with its exposure component (writing by oneself emotionally-charged accounts of traumatic events). A dissociative tendency was particularly predictive of PTSD improvements if participants initially reported experiencing depersonalization. Thus, participants prone to dissociate improve in the usual format of CPT, that is, the trauma-focused CPT. One explanation is that participants with a dissociative tendency might have responded to the exposure component in the third and fourth sessions with more dissociation, rather than a change process of integration or assimilation as suggested by Resick & Schnicke (1993).

Brief dynamic therapy. The change processes examined in BDT for treating PTSD are those proposed by dynamic theoreticians. Horowitz (1976, 1984, 1997, 2001) suggested that PTSD is resorbed if the traumatic information is assimilated after the psychological is accommodated, transformed. Such accommodation happens through the development of an alliance and the use of interpretations of defenses and conflicts related to both the traumatic event and the pre-existing structure of the participant.

A study of BDT with patients presenting with varying disorders, including PTSD, found that interpretations of warded-off wishes, feelings or ideas were significantly related to outcome (Jones, Parke, & Pulos, 1992). Such findings support the inferred mechanism of change proposed by dynamic theorists (Horowitz, Marmar, Krupnick, Kaltreider, Wilson, & Wallerstein, 1984).

In a study of process and outcome of BDT, PTSD reductions were predicted by the initial severity of PTSD and the alliance forged at the beginning of therapy PTSD motivation (Horowitz, Marmar, Weiss, DeWitt, & Rosenbaum, 1984). In addition, an interaction was found between patients' motivation for therapy and two types of interventions, supportive versus exploratory, in predicting PTSD reductions. BDT participants responded better to exploratory interventions if they initially presented higher motivation and they responded better to supportive interventions if they initially presented with low motivation (Horowitz, Marmar, Weiss, Kaltreider, & Wilner, 1986). Such therapeutic responses are in line with psychodynamic theory of change.

Given that the exploration of traumatic event in BDT is gradual and proceeds only at tolerable dosage (Horowitz & Kaltreider, 1979), it is unlikely that dissociation would be triggered by such technique, but it is possible. However, the role of dissociation should be investigated in BDT as well as in other trauma-focused therapies for PTSD. The ultimate test of an integration of traumatic memories is the lack of resurgence of PTSD symptoms after years following termination, especially after a contemporary traumatic event.*

** In a book chapter, the present author described the treatment of Mary (Gaston, 1995). After the murder of a teenager in her bus, Mary had developed a very severe PTSD, a major depressive disorder with suicidal ideations, and a conversion disorder. Within a year of a dynamic and integrative psychotherapy, all symptoms had remitted and Mary was back to work. Within a week, a gang fight suddenly occurred in the bus she was driving. Mary simply put on the 9-1-1 flashing light and left the bus. She waited for the fight to end and for those involved to leave the bus. She then re-entered her bus and drove away. No PTSD symptom was triggered. Five years later, she was working as a subway conductor which is a less stressful job than bus driver. A man threw himself onto her windshield in a suicide attempt. Mary developed a new PTSD, solely in regard to the suicide, and she consulted the Employee Assistance Program at her workplace because she was forced to do so by her employer. Treatment consisted of PE only. After 10 sessions, she went back to work with residual PTSD symptoms. Three years later, she called the present author because she had experienced a severe car accident. She was referred*

to a psychologist specialized in PTSD at the clinic of the present author who was unavailable for therapy. During the referral phone call, Mary recounted the story of the suicide and her subsequent PE therapy. She also reported the content of her following PTSD symptoms. After the car accident, Mary has symptoms pertaining to the car accident and the suicide; she had flashbacks of both the suicide and the car accident. However, Mary had no symptom involving the murder in her bus 10 years earlier; her traumatic memories of the murder had been integrated into her psychological structure. At the end of therapy following the car accident, Mary had lost all of her PTSD symptoms without focusing on the traumatic event per se; her anger toward a loved one was worked through.

Some neuroimagerie data is now available pertaining to the change process of BDT as delivered in a naturalistic setting. A neuroimagery study with fMRI (Dickie, Brunet, Akerib, & Armory, 2011) was conducted with participants in an open-ended dynamic integrative therapy for PTSD (Gaston, 1995, 2014). Neuroimagery data were taken at intake and 6 to 9 months later. Upon a trauma-related stimulation, neurobiological changes in the amygdala, hippocampus, and prefrontal anterior cingulate cortex were found to be associated with PTSD improvements. At 6-9 months within therapy, participants with PTSD remission were less aroused, less reactive to stressors, and more in neurobiological homeostasis upon trauma-related stimulation.

Neurobiological changes in trauma-focused vs. non-trauma-focused therapies. In a recent meta-analysis of 15 studies on the neurobiological changes associated with effective therapies for PTSD, Thomaes et al. (2014) found that almost all therapies were associated with, upon trauma-related stimulation, a decrease in amygdala activation and an increase in ACC activation at post-test from pre-test levels. These results involving more ACC activity might well indicate a top-down regulation of affects, suggesting that the ACC is triggered into activity to suppress affects. To understand these findings, a closer look at the therapies included in the meta-analysis by Thomaes et al. (2014) is warranted.

In the meta-analysis of Thomaes et al. (2014), mostly all psychological therapies for PTSD induced an increase in ACC activation, upon stimulation, in association with PTSD improvements. These therapies were either trauma-focused or they comprised a trauma-focused component (cognitive-behavioral therapies with PE, PE, or EMDR). Only one controlled clinical trial (Thomaes et al., 2012) obtained a decrease in ACC activation in remitted PTSD participants in both evaluated therapies. The therapies were non-trauma-focused: stabilization therapy and treatment-as-usual. These opposing findings (examined by the present author) indicate that non-trauma-focused therapies produced PTSD improvements through a decrease in ACC activity while trauma-focused therapies produced PTSD through an increase in ACC activation.

In remitted PTSD participants of trauma- focused therapies, the increase in ACC activation along with a decrease in amygdala activation upon trauma-related stimulation is the same concomitant response than the one observed in individuals with PTSD and a dissociative tendency (see Lanius et al., 2010). Dissociative individuals also show reduced heart rate

activation upon trauma-related stimulation (Griffin et al., 1997; Sack et al., 2012; Werner & Griffin, 2012). Given that the ACC modulates heart rate and an increased ACC activity reduces heart rate, these findings are in line with the results of Schubert et al. (2011) in which EMDR responders showed a sudden decrease in heart rate during the dual task.

Therefore, opposite change processes may well occur in trauma-focused therapies versus non-trauma-focused therapies for PTSD. It is possible that, in some patients with PTSD remission, the induction of an over-modulation of affect by the ACC allows for temporary PTSD remission. In non-trauma-focused therapies, it is also possible that an integration of affect-laden issues allows for the integration of traumatic memories.

Limited Generalizability

More and more, testing the efficacy of clinical treatments is performed through controlled clinical trials. Randomization is employed and a control for extraneous variables is applied in order to be able to infer that it is the studied treatments per se which are likely to be the cause of symptom reduction, over and beyond the improvement of a control group. In clinical trials, it is also important to control the experimental variable itself (i.e. treatment) by ascertaining the use of standardized manual by which the well-defined treatment is replicable elsewhere. These strategies increase the internal validity of a study, but they do not insure its internal validity. In addition, the more stringent are strategies to augment the internal validity, the more these strategies decrease its external validity, that is, the generalizability of findings to the general population.

The self-selection process selects only individuals willing to participate in all experimental provided because, for ethical reasons, all conditions need to be described in the publicity and they need to be described in details during the intake phone call. Therefore, only individuals interested in trying out trauma-focused therapies and willing to risk being put on a wait-list will apply to participate in a controlled clinical trial aimed at testing their efficacy.

Beyond the self-selection performed by potential participants, the selection process conducted by researchers further impedes the generalizability of findings because many inclusion and exclusion criteria are applied. In addition, treatments are manualized in an attempt to be able to replicate them and thus the obtained results. However, in psychotherapy, it may well be that the more manualized (standardized) a treatment is, the less effective it becomes. Manualized psychotherapies are frameworks into which patients must fit rather than psychotherapists adjusting a therapeutic model to the needs of patients. Let's take an example to illustrate this point.

As previously described, at a clinic specialized in PTSD, the conduct of a naturalistic and retrospective study (Brunet, 2004) was possible because structured diagnostic interviews for PTSD are part of the clinic's procedure, interviews being completed before and after treatment. The treatment provided is a dynamic integrative psychotherapy (Gaston, 1995) based on Horowitz's model plus some cognitive, behavioral and pharmacological interventions if possibly

therapeutic, along with the models of Bowlby and Masterson for personality disorders. Very high rates of effectiveness were revealed (96% of PTSD remission, with 48% of complete PTSD remission) even though very few selection criteria were applied (except for psychosis and severe substance abuse disorders). At this clinic, PTSD is usually severe and associated with serious functional limitations. Depressive, anxious and somatic comorbid disorders are also usually diagnosed, along with personality disorders. In addition, patients are compensated by agencies such as workers' compensation, victims' program, governmental car insurance, veterans' affairs, etc. and they are usually so dysfunctional that they cannot continue working. In such a context, it is usually inferred that odds are against remission. The psychotherapy is open-ended to respond to patients' progress as they occur naturally. Although general guidelines are provided to experienced clinicians trained for 2 years (see Gaston, 1995; Gaston, 2014, Gaston, 2014), psychotherapists have freedom to intervene in the best interest of patients. In contrast to the effectiveness rate of 96% obtained at this clinic, controlled clinical trials using manualized and time-limited therapies usually produce a remission rate of partial PTSD of 50% and rates of complete PTSD remission are usually not even mentioned in articles (see Barrera et al., 2012; Bradley et al., 2005; Powers et al., 2010).

In controlled clinical trials, investigators opt for designs increasing the internal validity as much as possible in order to be able to claim that the treatments are likely to be the factors at play for the observed symptom reduction. However, internal validity does not equate with external validity, or generalizability. On the contrary, these two characteristics of experimental designs are opposite. The more controlled a study is, or they more internally valid it is, the less its findings can be generalized to the general population. This basic reality of experimental science is ongoingly overlooked in the field of mental health. Researchers involved in controlled clinical trials have a propensity to overgeneralize the usefulness of their findings, forgetting that efficacy does not equate effectiveness and ignoring the possibility of the own experimenter bias. Nowadays, researchers even conduct as many statistical analyses as they wish, claiming that they wish to avoid rejecting valid results, and they also provide conclusions beyond any of their own statistical findings.

Controlled clinical trials indicate that highly and gently trauma-focused therapies for PTSD are equally efficacious for reducing PTSD in about 50% of completer as are non-trauma-focused therapies. As described above, these findings of efficacy entail serious limitations to generalizability, which represents a major problem in treating PTSD in clinical settings. Such caveat is not sufficiently acknowledged by researchers. Any claim derived from controlled clinical trials is exaggerated if a given therapy is considered the treatment of choice for PTSD or a treatment of choice for any individual presenting with PTSD. Unfortunately, proponents of PE and EMDR repeatedly claim that their highly trauma-focused therapy is a treatment a choice.

Indeed, clinical researchers claim that results supporting the efficacy of their favored trauma-focused therapies are generalizable to the PTSD population. Many, but not all, of these clinical trials have good internal validity, producing results pertaining to the efficacy of trauma-focused treatments. However, the question of their effectiveness remains open and is even highly questionable, that is, the question of the applicability of these partially empirically-

supported and manualized treatments to the general population of individuals suffering from PTSD. Here, let's remember that 'effectiveness' means how effective a treatment is in a usual clinical setting.

It is well-known that the greater the internal validity of a controlled clinical trial the less generalizable are its findings. Such is the scientific method. Results of controlled clinical trials should be counter-balanced by studies conducted in naturalistic settings. However, there is a bias against data derived from naturalistic settings as if they were untrustworthy or irrelevant. The present author dares to differ. There may well be as many confounding variables in controlled clinical trials as they are in naturalistic settings; they are just different confounding variables.

Thus, practitioners need to keep in mind the limited applicability of research findings derived from controlled clinical trials. Clinicians need to not be swayed by unwarranted conclusions from enthusiastic researchers. Indeed, numerous controlled clinical trials have found that therapies containing a trauma-focused component, whether it is PE, EMDR, hypnosis, or others, were more effective in reducing PTSD symptoms than a waiting-list control group. Nonetheless, whenever their efficacy is compared with anxiety-management therapy, or SIT, this gentler method is found to be equally effective (Barrera et al., 2012; Powers et al., 2010).

The above reflection aims at putting things in perspective and at encouraging practitioners not to readily endorse empirically-supported treatments proposed by clinical researchers. Practitioners can certainly consider trauma-focused methods as a possible component of a comprehensive psychotherapy for PTSD, but no more. Ethically, a rational for using any given technique needs to be developed by practitioners before using it, beyond having been trained in the using the technique, especially if severe side effects have been reported by patients.

Practitioners specialized in treating PTSD need to know about possible iatrogenic effects associated with trauma-focused therapies, especially the highly trauma-focused ones such as PE and EMDR. If we rely on the two surveys regarding the use of PE in naturalistic settings (Becker et al., 2004; van Minnen et al., 2010), practicing psychologists and PTSD experts rarely employ such highly trauma-focused therapy. Clinicians approach individuals presenting with PTSD in an idiosyncratic and comprehensive fashion. Most patients do not want to engage in a trauma-focused technique (Scott & Strandling, 1997) although treatments such as PE and EMDR are now repeatedly praised in the media.

Most patients do not have the affect-modulation capacities to engage in a trauma-focused technique. In clinical trials, individuals accepting to become subjects have been explained all therapeutic methods. They have accepted to readily engage in them, which sets them apart from persons seeking help in the community. Researchers and research assistants are not blind to the treatment of choice and it is quite reasonable to infer that they are enthusiastic toward a trauma-focused therapy for which they spend so much time evaluating in

order to demonstrate its efficacy, creating an unwanted experimenter bias effect inflating results. Furthermore, participants are not blind to the treatments of choice, which is likely to trigger subject reactivity and inflate results.

Life is messier than the laboratory, even if the latter is a clinic. It is thus important to not impose any forms of specific treatments upon individuals with PTSD because they have been reported to be efficacious in reducing PTSD symptoms in controlled clinical trials. Such propensity has led to the endorsement of therapies demonstrated only to be partially efficacious in some patients under very controlled conditions. Generalizing these findings to real-world clinical settings without carefulness and caution can be detrimental to other individuals with PTSD.

Our present neurophysiological knowledge of PTSD supports a cautious clinical perspective. Psychophysiological studies instruct clinicians that individuals with PTSD have a high reactivity to trauma-related stimulation and, therefore, present a limited capacity for affect-modulation and habituation (see the seminal study of Blanchard, Kolb, Pallmeyer, & Gerardi, 1982). Indeed, PTSD is neurobiologically associated with hyperarousal of the amygdala and an under-modulation of arousal of the prefrontal cingulate anterior cortex (AC) when they are exposed to a trauma-related stimulation (Lanius et al., 2010).* The less capacity to habituate (anxiety spontaneously reducing) and the more symptomatic individuals are, the less they are likely to truly benefit from highly trauma-focused therapies, unless a mechanism of dissociation is induced by such techniques in an effort to counter the experience of overwhelming dysphoric affects.

** Let's remember that those with a tendency to dissociate present the inverse neurobiological reactions (Lanius et al., 2010) which sets them apart in a distinct PTSD category as acknowledged in the DSM-5 (APA, 2013).*

In controlled clinical trials, only half of participants obtain PTSD remission across therapies (Bradley et al., 2005). Clinical improvements are reported only in terms of partial PTSD remission in RCTs (see Bradley et al., 2005), with one exception (see Schnurr et al., 2007) reporting rates of 7%-15% of full PTSD remission. The lack of findings regarding complete PTSD remission is not surprising given that treatments in RCTs are time-limited and standardized. To better understand the limitations of controlled clinical trials and the lack of generalizability of their findings, let's look at a naturalistic situation.

At a clinic specialized in PTSD, dynamic integrative therapy is provided to almost all consulting individuals. The therapy is based on Horowitz's model for PTSD, with behavioral, cognitive and pharmacological additions (Gaston, 1995). Since 1995, this therapy was augmented by Masterson's models for treating personality disorders. Therapeutic interventions are adjusted to patients' needs and capacities. Therapy lasts as long as PTSD is not remitted or when a therapeutic ceiling has been reaches (usually due to long-term hypersensitization in patients). A trauma-focused technique is rarely employed, but introspective hypnosis (see Spiegel, 1989) if and only if patients have developed a capacity to continue self-reflection

during highly dysphoric affects, a solid therapeutic alliance has been established, and the experiential revision of the traumatic event at the usual waking state has not led to the identification of significant personal meanings and/or reactivated conflicts. Most patients present with severe PTSD, co-morbid disorders, personality disorders, and serious functional limitations (e.g. inability to work). Patients receive compensation from a governmental agency and psychotherapy is covered by the latter. PTSD diagnoses are established before and after therapy using a structured interview. Within this context, an independent retrospective study was conducted by Brunet (2004) as a preliminary step for a neuroimaging study (Dickie et al., 2011). One hundred files were randomly selected and examined by a research assistant. Results revealed that, in average, psychotherapy lasted 9 months. On the Structured Clinical Interview for DSM-V (SCID), the rate of PTSD remission was 96% at termination, with 48% of complete PTSD remission. Thus, full PTSD remission is possible for many individuals presenting with PTSD, even with severe PTSD complicated by comorbid clinical disorders and personality disorders within a compensation context.

Prerequisite for Using Trauma-Focused Techniques

Only focusing on traumatic memories can be therapeutically very effective, they are many impediments to using trauma-focused experiencing techniques. These are effective without risking severe negative side effects if preliminary conditions are in place.

Firstly, there needs to be a well-established alliance between patient and therapist. Although therapists can offer an empathic relationship to patients, many patients do not readily form an alliance with therapists. Secondly, the patient needs to possess sufficient capacities for good-enough arousal-modulation and affect-modulation. Trauma-focused techniques provoke intense affective neurobiological arousal in patients and can well be whenever a sound alliance is not established yet and patients do not present yet with a capacity to sustain intense dysphoric affects without becoming overwhelmed.

The therapeutic and working alliances presented by patients are also a reflection of their capacity to form secure attachments and to modulate dysphoric affects successfully. Patients who cannot readily form a good-enough alliance in psychotherapy present with limited capacities to establish secure attachments and modulate affects successfully, which renders them vulnerable to internal disruption during and after the use of re-experiencing techniques. Too often psychotherapists assess the alliance with their patients as strong while the patient could not address distressing issues in a transformative way. It is not because patients address with us difficult issues collaboratively and reflect on their problems that there is a good-enough alliance. The alliance is a deeper reality which depends on the internalization of the psychotherapist.

Insecure attachment patterns render individuals vulnerable to developing PTSD, given both their reduced capacity to modulate affects and their malevolent representations of others. Therefore, trauma-focused techniques are not amenable to treat patients presenting with PTSD and personality disorders. At the clinic specialized in PTSD which effectiveness was examined by

Brunet (2004) and Dickie et al. (2011), the majority of individuals presenting with PTSD usually also present with co-morbidity, an attachment disorder, and a personality disorder (diagnosed according to Masterson's model). Such dispositions require adjusting the psychotherapy designed for treating PTSD because these individuals need to build new structures, psychological and neurobiological, before they can reexperience traumatic memories associated with intense dysphoric affects and states (hopelessness, helplessness, etc.). Furthermore, a fully successful psychotherapy of PTSD may not involve re-experiencing traumatic events because individuals may have sufficiently built new inner representations and structures to allow for the spontaneous assimilation of traumatic memories.

Conclusion

Many therapeutic approaches developed for treating PTSD are primarily trauma-focused (PE and EMDR) or contain an exposure component (CPR and BDT). Despite repeated findings of partial efficacy in about 50% of participants, major questions remain unanswered. In response, many scientific and therapeutic concerns have been outlined in this article.

A first consideration for practitioners is that trauma-focused techniques are not more efficacious than other therapies. A second concern is that PTSD experts and some participants consider highly trauma-focused techniques to induce at times severe side effects and attrition. Mental health relies on a core ethical principle, 'Above all, do no harm'.

Our ethical principles demand that the least disturbing treatment should always be applied before attempting any treatment known to produce iatrogenic effects. Although iatrogenic effects are known by PTSD experts and patients regarding highly trauma-focused techniques (PE and EMDR), researchers insist on their quasi-absence in controlled clinical trials. This discrepancy is interesting, not to say puzzling.

Over the last 25 years, there has been a favourable bias toward trauma-focused technique for PTSD in research, especially highly anxiety-provoking techniques such a PE and EMDR. Controlled clinical trials are conducted by highly enthusiastic proponents and their conclusions are at times exaggerated. The possibility of experimenter bias and subject reactivity are not considered. Claims of high efficacy, acceptable dropout rates, and absence of negative side effects are to be taken with caution. Practitioners and PTSD experts have relied mostly upon their own clinical experience and judgement to treat individuals presenting with PTSD and comorbid conditions. From the present overview, it appears that practitioners should continue to do so, rather than relying on empirical findings which are not so generalizable and on efficacy claims offered by enthusiastic researchers. Clinical experience, knowledge of various models, therapeutic flexibility, and a primary concern for patient's welfare is likely to produce more beneficial effects than an adherence to any therapeutic method.

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