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Risk Factors in PTSD and Related Disorders: Theoretical, Treatment, and Research Implications¹

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Contemporary treatment approaches for Posttraumatic Stress Disorder (PTSD) include traditional approaches such as Cognitive-Behavioral therapy, Psychodynamic therapy, Group Therapy, Pharmacotherapy, *et cetera*, as well as experimental approaches such as Body therapies (*e.g.*, Sensorimotor Processing) (Ogden & Minton, in press) and other Asian-based approaches (often termed “Energy Therapies”). These approaches have varying degrees of data in support of their effectiveness, that range from anecdotal case reports to randomized and controlled studies (*e.g.*, see Dietrich, *et al.*, 2000; Foa, Keane, & Friedman, 2000; Shalev, Bonne & Eth, 1996; van der Kolk, McFarlane, & van der Hart, 1996).

Figley (in press) states that desensitization (*i.e.*, de-conditioning the learned association between anxiety and a feared stimulus) through gradual exposure and Reciprocal Inhibition (RI) has received empirical support as an effective anxiety antagonist in the treatment of anxiety disorders. From this perspective, the learned response to the *original* feared stimulus (traumatic event) results in an anxiety or fear response that requires desensitization, as does the learned response that *maintains* the symptoms.

Note, however, that the view that desensitization and extinction involve unlearning the association between the conditioned stimulus and the unconditioned stimulus has been questioned. There is some evidence that emotional conditioning (*e.g.*, involving the amygdala) is permanent, but can be modulated through contextual evaluation (*e.g.*, see Bouton, & Schwartzentruber, 1991; Bouton, 1994). Thus, although learning principles appear to be effective in the treatment of symptoms of PTSD, it is not clear that the original emotional learning can be completely reversed.

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Gradated Versus Prolonged Exposure

In an early, well-controlled study, Brom, Kleber and Defares (1989) found systematic desensitization (SD) to be effective for the treatment of PTSD. Rothbaum, Meadows, Resick and Foy (2000) acknowledge the study by Brom *et al.*, and also state that “although several studies have found that SD was effective in reducing posttrauma symptoms, the studies [other than that by Brom *et al.*] are not well controlled” (p. 75). Rothbaum *et al.* (2000) claim that, because prolonged exposure appears to result in better outcomes than shorter exposure and because relaxation during exposure does not contribute to treatment effectiveness, SD has fallen into disfavor.

Rothbaum *et al.* have not explicitly cited any empirical data in support of their assertion that relaxation *in combination* with gradual exposure fails to contribute to treatment effectiveness, however, and it is not clear as to how or why they have come to this conclusion. Moreover, they acknowledge that relaxation, *per se*, appears to have some “limited utility” for assisting clients with arousal (p. 79). One difficulty with prolonged exposure is that it is not well tolerated by some individuals (Rothbaum, *et al.*, 2000). For example, clinical data suggest that individuals with complex posttraumatic and dissociative disorders decompensate with prolonged exposure (*e.g.*, Herman, 1992), and studies have also shown that direct exposure techniques result in deterioration for some proportion of clients (*e.g.*, see Kilpatrick & Best, 1984; Litz, Blake, Gerardi & Keane, 1990; Pitman *et al.*, 1991; Pitman, Orr, Altman & Longpre, 1996; Scott & Stradling, 1997; Vaughan & Tarrier, 1992; Watson, Tuorila, Detra, Gearhart & Wickiewicz, 1995). Moreover, dropout rates of participants with simple PTSD when using flooding and prolonged exposure techniques appear higher than with other approaches. Finally, empirical studies that compare prolonged exposure to other techniques frequently screen out those individuals with more complex forms of psychopathology. As such, prolonged exposure might be most effective with some proportion of traumatized people, and less appropriate for others.

As noted by Briere (in press), when exposure is too fast-paced with insufficient time to process the material, clients can easily become overwhelmed and resort to increased avoidance activities to modulate their distress. As such, they may utilize dissociation, cognitive avoidance, abuse of substances, *et cetera*, and may drop out of treatment. In addition, effective treatment is believed to require information that is disparate from the original trauma (Briere, in press; Foa & Kozak, 1986). Disparate information includes the absence of danger, as well as positive components that are antithetical to danger (Briere, in press).

As such, the pacing that occurs with SD can function to prevent the excessive use of avoidance coping, whereas the relaxation response may provide disparate information from the original trauma (*i.e.*, people are not in a relaxed state during a traumatic event). In relation to the latter, the relaxation or other disparate information, when repeatedly presented in conjunction with feared traumatic stimuli, acts in such a way that the original conditioned association is not reinforced (Briere, in press). Moreover, there is some evidence for the possible role of endogenous opioids with elicitation of opposite emotions (Solomon & Corbit, 1974). In other words, relaxation, although not sufficient in itself for the treatment of PTSD (Rothbaum, *et al.*, 2000), may not only help to attenuate arousal in response to exposure, but may function as positive information that is antithetical to the danger of the trauma.

Nijenhuis, Van der Hart, and Steele (Nijenhuis *et al.*, in press; Steele *et al.*, in press) argue that recovery from PTSD and dissociative disorders involves the integration of traumatic memories, encoded and stored in a dissociative system metaphorically labeled the "Emotional Personality" (EP), into the "Apparently Normal Personality" (ANP), which has failed to perform this mental act. According to this theory, the EP is a psychobiological system dedicated to defense to bodily threat and life, and the ANP is a psychobiological system dedicated to fulfilling functions in daily life. Integration of the EP can be interfered with by excessive arousal during exposure, as well as by extreme lowering of the level of consciousness (*e.g.*, entering trance states) of the ANP. If the ANP becomes over-aroused during exposure to the traumatic memories, conditioned dissociative reactions can become reactivated. When the ANP engages in mentally avoiding the traumatic memory during exposure, exposure will be inhibited for that reason.

Therapy from this theory of structural dissociation involves concomitant activation of the dissociated traumatic memories and the ANP. Thus, exposure to these internal stimuli is required, as well as response prevention with respect to re-dissociation or mental avoidance that may hamper or block the effective exposure. Exposure is viewed as a means to an end (*i.e.*, integration of dissociative systems), rather than as a therapeutic agent in itself. According to this theory, ANPs fear and avoidance of traumatic memories maintains the dissociative organization of the personality, and the integration of dissociated material is key to recovery. It should be noted that not all individuals with simple PTSD are dissociative (Putnam *et al.*, 1996), and thus this theory may not be applicable to all traumatized clients. With non-dissociative clients, painful memories may take the form of distinct cognitive schemata (such as in state dependent memory), rather than as dissociated personality structures.

Thus, it is proposed that effective trauma treatment involves assisting clients to overcome their fear of trauma stimuli/memories and integrate them into their existing cognitive² schemas or structures. Gradual exposure/pacing allows clients to better tolerate the memories and affect, without the need for excessive cognitive avoidance or dissociation. The inclusion of disparate information (*e.g.*, relaxation, safety, and so forth) and positive information assists the clients in modulating their memories such that the memories no longer have the power to elicit full-blown affective distress. That is, new information is integrated into memory, which might enable the individuals to modulate their distress more effectively through the use of contextual information that is discrepant from the original trauma.

In the next section, risk factors that are believed to contribute to the development of PTSD are summarized. It is suggested that, with an increase in the number of risk factors present for an individual client, active components of treatment may become increasingly more complex. Treatment interventions may thus need modification, depending on the nature of the risk factors present. Treatment outcome may similarly vary depending on the presence, severity, and nature of various risk factors.

Risk Factors for PTSD: Treatment Implications

The construct of PTSD, as explicated by the Diagnostic and Statistical Manual of Mental Disorders, Fourth edition, Text revision (DSM-IV-TR) (American Psychiatric Association, 2000), includes two major etiological factors implicit in the diagnostic criteria. The first refers to the objective traumatic stressor event (Criterion A1), and the second refers to the subjective response of the individual to that objective event (Criterion A2). In the absence of an extreme stressor that involves, first actual or threatened death or serious injury, or other threat to one's physical integrity; or witnessing an event that involves death, injury, or a threat to the physical integrity of another person; or learning about unexpected or violent death, serious harm, or threat of death or injury experienced by a family member or other close associate" (American Psychiatric Association, 2000, p. 463), and second, the absence of the *specific* emotions of intense fear, helplessness or horror, PTSD, by DSM-IV-TR definition, is not possible.

Research has shown, however, that some proportion of individuals appear to develop clinical symptoms of PTSD following normative life events (such as marital disruptions) (*e.g.*, Burstein, 1985; Helzer, Robins, & McEvoy,

² Note that "cognitive" does not refer *only* to thoughts or beliefs, but also involves affective and other components.

1987), leading some experts to question the wisdom of the A1 objective stressor criterion included in the DSM-IV. For example, Yehuda (2001), generalizing from Lazarus's (1990; 1993) notion of the role of subjective appraisal in *normal* coping, opines that weight should be given to subjectively distressing normative events, in the presence of risk factors, as etiological factors in *traumatic* coping or PTSD. That is, Yehuda appears to view stressful normative life events, in combination with certain risk factors, as etiologically sufficient for PTSD.

An alternative hypothesis, however, is that the risk factors in cases of low magnitude events are most predictive, not of a homogenous disorder of PTSD, but of pre-existing disorder or of another disorder that might mimic symptoms of PTSD (e.g., adjustment disorder, simple phobia). In this sense, symptoms or signs of other constructs (e.g., symptoms of depression, adjustment disorder, stress-induced phobias, complicated bereavement, dependent personality styles, *et cetera*) are possibly being confounded with those of PTSD. These hypotheses require further empirical study. They also point to the importance of thorough assessment prior to treatment in the determination of treatment effectiveness. When clients who have experienced stressful normative events (and the absence of any trauma history) present for treatment with symptoms that resemble those of PTSD, practitioners should consider alternatives to PTSD as diagnoses. Desensitization techniques and trauma-specific techniques may still be appropriate for distress following normative events, however, depending on the specifics of the case and the symptoms in evidence.

The American Psychiatric Association (2000) discusses risk factors that affect the likelihood of developing PTSD, *viz.*, The severity, duration, and proximity of an individual's exposure to the traumatic event are the most important factors affecting the likelihood of developing this disorder. There is some evidence that social supports, family history, childhood experiences, personality variables, and preexisting mental disorders may influence the development of Posttraumatic Stress Disorder. This disorder can develop in individuals without any predisposing conditions, particularly if the stressor is especially extreme (p. 466).

Epidemiological studies (e.g., Kessler, *et al.*, 1999) have shown that event severity constitutes one of the most important risk factors for PTSD. The National Comorbidity Survey (as reported in Kessler, *et al.*, 1999) found that the risk of PTSD is highest for those male and female respondents who report rape, childhood physical abuse, and childhood neglect. Combat is also strongly related to PTSD for males (it should be noted that the effects of multiple traumas were not examined in this study).

McFarlane and deGiolamo (1996) note that traumatic stressors vary in terms of duration, severity of life threat, severity of injury, and loss of life and property, which may also function as risk factors for PTSD; however, there is little prospective research that looks at the role of these event factors in PTSD (McFarlane, 1999) and in dissociation (Ogawa, Sroufe, Weinfeld, Carlson, & Egeland, B., 1997). According to Harvey and Yehuda (1999, p. 2) on the other hand, the finding that not all individuals develop PTSD after a severe traumatic event suggests “the failure to develop such symptoms is likely the result of subjective perceptions at the time of or subsequent to the event and not the impotence of the stressor.” According to Yehuda (1999a, p. xviii),

The dose-response relationship between severity of the trauma and the development of PTSD implies that vulnerability factors may be particularly important as one moves down along the spectrum of horror and catastrophe. Because there is a qualitative difference between being subjected to purposeful torture and being involved in a motor vehicle accident...vulnerability factors may be more relevant in the induction of PTSD in response to the latter (lower magnitude) trauma.

Thus, although the American Psychiatric Association, through the DSM-IV-TR diagnostic criteria, conceptualizes PTSD as a more or less learned response to a severe event that involves a subjective affective component (which is partially borne out empirically by cross-sectional epidemiological studies), empirical research has also suggested that the probability of occurrence is not only related to the severity, duration, and proximity of exposure (direct learning factors), but also to predisposing risk factors (Shalev, 1996; Yehuda, 1999a) that may or may not be learned, as well as to the type of traumatic event experienced (Davidson & Foa, 1991a; Kessler, *et al.*, 1999; McFarlane & de Girolamo, 1996). Moreover, not all individuals who are exposed to extreme stressful events develop PTSD (McFarlane & de Girolamo, 1996; Yehuda, 2001), which also appears to suggest that either (1) there is more involved in PTSD etiology than learning alone or (2) traumatic events can lead to PTSD or to other disorders (e.g., Dissociative Disorders; Mood Disorders; etc). The DSM-IV has included symptoms of other disorders in the “Associated Features” section to PTSD.

In practice, assessment of PTSD criteria A1 (nature and severity of trauma) and A2 (subjective appraisal) during intake sessions can assist the clinician with treatment planning using brief treatments. Gathering data regarding the specifics of the event can assist the clinician in determining whether to utilize a simple exposure-based approach or a more phase-oriented treatment approach, and can assist the clinician when using traumatic memory exposure techniques. With regard to the latter, for example, if a client with simple PTSD uses avoidance

due to fear of traumatic memories, the clinician can gently and carefully introduce specific details about the traumatic event to help trigger or elaborate upon the client's memory (while carefully observing for and respecting the client's use of titration so as to not overwhelm the client) (see Briere, 1997a for a discussion on recognizing and working with client's use of titration).

Similarly, thorough assessment of the client's subjective feelings and thoughts at the time of the event and after the event can assist the therapist with cognitive/affective reprocessing. For example, the therapist can gently challenge cognitive distortions that form during or following the trauma. Moreover, the therapist can utilize information about the client's emotional state at the time of the trauma when doing desensitization (*e.g.*, if the client reports intense helplessness, the therapist can monitor this during exposure, and provide or elicit discrepant information to assist in reprocessing and integration of the traumatic memory).

Traumatic Risk Factors. The following factors have been proposed as playing a role in the development of PTSD following exposure: Prior histories of other DSM disorders and gender, (Kessler, *et al.*, 1999); genetic factors (McEwen, 2000; True, *et al.*, 1993; True & Lyons, 1999); family members who have PTSD or mood or anxiety disorders (Yehuda, 1999b); prior traumatization (Shalev, 1996); acute reactions at the time of the traumatic event, including the possible roles of peritraumatic dissociation, coping, and physiological stress reactions (McFarlane, 1999); biological alterations (Shalev, 1999; Yehuda, 1999b); neurocognitive factors (Orr & Pitman, 1999); impaired affect regulation or self-capacities (Briere, *in press*) and personality factors (Schnurr & Vielhauer, 1999).

Childhood maltreatment also constitutes a risk factor for the development of PTSD in response to a traumatic event (Briere, *in press*; Nijenhuis, van der Hart & Kruger, *submitted*), which may be mediated by physiological sequelae following child maltreatment (*e.g.*, see Glaser, 2000; Ito, *et al.*, 1993; Ito, Teicher, Glod & Ackerman, 1998; Putnam & Trickett, 1997; Teicher, Glod, Surrey, & Swett, 1993). That is, there is some evidence that child maltreatment results in physiological changes, which could increase the risk of exposure to traumatic stressors and/or developing PTSD in response to subsequent stressors.

During intake with new clients, it is suggested that therapists assess for risk factors and consider them as potential treatment targets or as factors that may affect treatment staging, progress, and outcome, as explicated later in this chapter (a checklist is provided in Appendix A for this purpose).

Re-examining PTSD Etiology

Harvey and Yehuda (1999) discuss diathesis-stress models of PTSD and other disorders. Diathesis-stress models posit that outcome is a function of an interaction between pre-dispositional (*i.e.*, risk) factors (including, but not limited to, genetics³) and environmental factors. From this perspective, neither environmental factors (the traumatic event) nor pre-dispositional factors, in isolation, are sufficient for PTSD.

It would appear that there are several possible causal pathways leading to PTSD, each a function of variations in the type and severity of trauma and the degree to which risk and protective factors are present. Some possible pathways are depicted, below.⁴

Low Magnitude Event and Low Risk Factors. With normative life events and low to moderate risk factors, it is hypothesized that the outcome would be temporary emotional distress that attenuates with time and/or supportive interventions. PTSD would not be an expected outcome.

Low Magnitude Event and High Risk Factors. With low magnitude events coupled with high risk factors, possible outcomes could be prolonged distress, adjustment disorder, complicated bereavement, phobia, single depressive episode, and so forth (depending on the specifics of the case), the symptoms of which could mimic PTSD.

High Magnitude Event and Low Risk Factors. With high magnitude events and low risk factors or high protective factors, an expected outcome might be distress with symptoms of acute PTSD or sub-clinical PTSD. In such situations, the distress and symptoms could attenuate with time and support, or might require some degree of exposure, desensitization, and/or cognitive processing (whether through formal treatment, or as a natural response to traumatic events (*e.g.*, Briere, *in press*)).

³ Genetic risk factors may find their active expression in partial response to environmental triggers (McEwen, 2000).

⁴ See McFarlane (1999) for an interesting discussion on possible etiological pathways in terms of interactions between properties of the stressor event (severity of threat and/or severity of injury) and the reaction during the event (*e.g.*, peritraumatic dissociation, acute psychological symptoms, and/or acute biological symptoms).

High Magnitude Event and Moderate Risk Factors. In this scenario, an expected outcome could be simple to chronic PTSD, amenable to exposure and desensitization techniques and cognitive processing (e.g., CBT, EMDR, *etc.*). With more severe symptoms, other treatment modalities could be required, such as Dialectical Behavior Therapy (Linehan, 1993), Sensorimotor Processing (Ogden & Kekuni, in press), and so forth.

High Magnitude Event and High Risk Factors. When high magnitude events are coupled with high risk factors, the expected outcome could be severe and chronic PTSD, or complex PTSD (*i.e.*, PTSD plus the associated features to PTSD). Interventions would consist of much stabilization and support prior to desensitization, and would likely involve other treatment modalities, such as interpersonal therapy, group treatment, skills training, body-focused treatments, and/or pharmacotherapy.

The Role of Risk Factors

The postulated pathways above are simplistic generalities in need of refinement (e.g., in terms of specific hypotheses regarding the interplay of relevant variables), and in need of empirical study. They also do not consider the type of trauma involved, or the explicit role of protective factors.

Risk factors may affect outcome in several ways. The actual outcomes of the above pathways are likely differentially determined, depending on properties of the stressor events, the risk factors present, the protective factors present, and the synergistic effects of their various possible combinations.

First, some risk factors can conceivably act to increase the probability that an individual is exposed to Criterion A1 (traumatic stressor) events (Kessler, *et al.*, 1999). For example, a child who has experienced emotional neglect may be at increased risk of interpersonal traumatization, such as extrafamilial sexual abuse. Similarly, women may be at higher risk of rape than men. A history of significant emotional neglect during childhood will in all probability complicate treatment. The client may have greater difficulty in feeling that s/he is worthy of self-care and/or care from others. Such clients may present with poor hygiene, lack of self-care, and a core belief of self as undeserving. S/he may have difficulty in trusting that the therapist is genuinely interested in his or her well-being. In such situations, the therapeutic relationship could require a significant amount of attention by the therapist, particularly during the early stages of treatment. Validation, support, empathy, and a non-judgmental stance on the part of the therapist can assist in the building of rapport and trust, and should be maintained over the duration of treatment. When the client is treated by the therapist in a

respectful, validating manner, the client's schema of self as undeserving can begin to shift, which can assist the client in taking greater care to avoid possible re-traumatizing situations, even if s/he has a history of neglect or maltreatment. When clients present for brief treatments, an initial focus on assisting the client in solidifying their sense of self as worthy of safety and care can set the stage for later trauma processing. Clients can take a break from treatment when desired, and can practice solidifying their gains in self-care and self-respect, in preparation for trauma treatment at a later date.

Second, as noted by McFarlane and deGirolamo (1996), the properties of the A1 criterion (traumatic stressor) may function as risk factors that increase the probability of PTSD. For example, the severity of injury during a rape could determine the nature and degree of peritraumatic reactions, including the biological stress response (*e.g.*, see Dietrich, 2000; McFarlane, 1999). With extended physical injury and stress, there may be excessive physiological dysregulation (McEwen, 2000). Stressors that result in significant tissue damage, therefore, could result in a longer latency to homeostatic baseline as the body attempts to recover (Dietrich, 2000). As noted by Yehuda (1999a), physiological alterations in response to traumatic stressor events may act to increase the risk of PTSD. With clients who have experienced physical injury at the time of the traumatic event, interventions relating to pain management and coping with loss might become an important treatment component. Adjunctive treatments, such as massage therapy, can also be recommended to clients. When there are significant immunological disruptions based in prior traumas (*e.g.*, see Wilson *et al.*, 1999), treatment interventions can be aimed, in part, at strengthening physical health when possible.

Similarly, repeated occurrences of Criterion A1 events (frequency), the length of time exposed to the event(s) (duration), and the number of different traumatic events experienced, could also function to increase the risk of PTSD, independent of the nature of the subjective response. That is, the data at this point in time may not be conclusive that the events, *per se*, are insufficient to lead to PTSD. Although some studies have reported that experiencing the "same" trauma (*e.g.*, the Holocaust) leads to differential response (*i.e.*, some get PTSD, some do not) (*e.g.*, Yehuda, 1999a), it is not clear that all Holocaust traumas, even when in the same camp, are objectively the same. The same would likely hold with traumas such as rape, combat, and so forth. One rape is not identical to another; one battle is not identical to another. As difficult as it may be to do so in a practical sense⁵, it is my view that well-designed study is needed that looks

⁵ One difficulty includes the retrospective rating of the stimulus properties by respondents, which may be affected by (a) the current level of

carefully at the stimulus properties of the event(s), while statistically controlling for the effects of subjective/risk factors.

Third, subjective factors, such as appraisal, may mediate between the event and outcome, affecting the response (Harvey & Yehuda, 1999). For example, appraising oneself as helpless in response to a traumatic event may lead to increased risk of PTSD, whereas an illusion or belief in control can lead to better outcome (van der Kolk, McFarlane, & Weisaeth, 1996). In this sense, risk factors may act on the subjective experience criterion (A2) of PTSD to mediate outcome. Treatment interventions that incorporate cognitive reframing or restructuring could be helpful. Adjunctive interventions such as self-defense courses can also increase a client's sense of power and control.

Risk factors could also conceivably affect subjective experience in a way that moderates outcome, by affecting the *experience* of the stimulus (traumatic event) properties. For example, McFarlane (1999, p. 176) posits "passive surrender, stoic acceptance, and cognitive-reframing are appropriate in situations where the stressor is uncontrollable." So, for example, a rescue worker who is removing bodies following a disaster might fare better if s/he (temporarily) cognitively re-frames the bodies as something else (e.g., store mannequins). Although such re-framing can assist individuals in coping with the immediacy of the trauma and enable them to function, clients may need to expose themselves to the actual memories if they are troubled by intrusions and other posttraumatic symptoms. The role of the therapist in this regard is to validate and normalize the coping response, while also gently challenging the client's cognitive framing of what happened. For example, if Joe continues to frame the bodies as mannequins for several months after the traumatic event, and experiences intrusive images of bodies with concomitant distress, it would be important to validate and empathize with Joe's experience, but also to expose him to the trauma so that he could integrate the dissociated emotional components into his conscious awareness, such as with the following intervention (when Joe is ready): "In order to get through that horror, you coped by viewing the bodies of those *people* as mannequins. You needed to do that to get through it. [Gently, softly:] I am wondering, Joe, how you feel when you look back and you see that those "mannequins" are human beings?"

Finally, risk and/or protective factors could conceivably affect outcome after a traumatic event, such as degree of tolerance of distress in response to memories of the event, openness to seeking treatment following a traumatic event, as well as to treatment responsiveness. For clients who have difficulties with

symptoms, such that ratings are magnified (Yehuda, 1999a), or (b) by avoidance or suppression, such that they are minimized (Briere, 1997).

tolerating the distress from traumatic memories, skills training (Linehan, 1993) can be advantageous in preparation for later processing. In addition, with chronic trauma (such as incest), normative healing processes (see Briere, in press) may be constantly impeded or interrupted because of repeated violations, resulting in increasing levels of avoidance over time becoming a habitual coping response. Avoidance as a habitual way of coping with trauma-related distress has important treatment implications. Skills training, such as teaching clients to practice mindfulness skills, emotional regulation, distress tolerance, and communication skills (Linehan, 1993), can assist clients with tolerating the painful trauma affect and thus reduce their tendencies toward avoidance coping.

Issues of Classification: Implications for Diagnosis and Treatment

The DSM classifies PTSD as a homogenous anxiety disorder that is distinct from other disorders. However, empirical data concerning the presence of postulated risk factors, the heterogeneity of sequelae following traumatic events (e.g., see Dietrich & van der Kolk, in preparation), as well as the degree of symptom overlap among different diagnostic categories of the Diagnostic and Statistical Manual of Mental Disorders (DSM) (American Psychiatric Association, 2000)⁶, raise the question of whether symptoms of psychological disorder vary as a function of variations in risk factors and trauma type. For example, do different risk factors function to (a) increase the probability of developing PTSD (Yehuda, 1999a), (b) increase the probability, depending upon interactions with the type of trauma experienced, of different *types* of PTSD, or (c) determine the differential likelihood of different disorders (e.g., see Marshall *et al.*, 2000; McEwen, 2000; Pelcovitz, *et al.*, 1996; Putnam, 1997, 2000), depending on the particular risk factors involved in a given case, regardless of the severity or type of stressful event?

Accurate assessment of what ails a given client assists the therapist in choosing a brief treatment approach that best suits the presenting needs and symptoms of the client, and also allows for informed determination of treatment effectiveness. For example, treatment of depression will have some similarities and some major differences relative to treatment of PTSD. Similarly, treatment of Complex PTSD (*i.e.*, the associated features to PTSD) will differ in substantial ways from the treatment of simple PTSD.

For clients who suffer from PTSD and associated features, a treatment plan that involves treating the client in “brief treatment blocks” may be most

⁶ For example, there are several commonalities between symptoms of PTSD and symptoms of depression.

suitable for stabilization and treatment of the complex symptoms. With such clients, phase-oriented treatment approaches are strongly recommended (e.g., see Chu, 1998; Herman, 1992). Such clients require safety and stabilization prior to confronting traumatic memories. Linehan's Dialectical Behavioral Therapy (DBT) skills training modules, provided to patients in "brief treatment blocks" with sufficient time between blocks to practice and reinforce the skills on their own, have therapeutic value for beginning phases of treatment. Some preliminary treatment outcome data (Prisman, Dietrich, & Shercliffe, 2000) on an intensive, time-limited treatment program using DBT principles showed that, of the 21 patients who were followed over four time periods (baseline and 3 follow-up periods), all showed reductions of symptoms on a variety of outcome measures, including the Crime-Related PTSD scale. With reductions in their symptoms and improvements in their coping and functioning, these clients are better equipped to engage in trauma work at a later period of time (*i.e.*, during a later "brief treatment block").

According to Brett (1996) and to Davidson and Foa (1991b), the DSM-IV Task Force was faced with the decision of whether to classify PTSD as an anxiety disorder, a dissociative disorder, or a stress disorder. The final decision, based only partially on empirical evidence, was to classify PTSD as an anxiety disorder. Some of the more substantive reasons for including PTSD as an anxiety disorder included the fact that PTSD includes symptoms of fear and avoidance; PTSD has similarities with panic disorder in terms of symptoms and patterns of autonomic arousal, response to yohimbine challenge, and corticotrophin-releasing factor; and many clients with simple PTSD respond to treatments that were originally developed for the treatment of anxiety (Davidson & Foa, 1991b). Davidson and Foa (1991b) opined that the empirical data favor PTSD as an anxiety disorder.

According to Brett (1996), arguments against classifying PTSD as an anxiety disorder in favor of classifying it as a stress disorder include the unanswered question of whether the arousal in PTSD is simply anxiety and whether it has the same physiological pathway as other anxiety disorders; the arousal and numbing phases of PTSD have similarities to processes of mourning and bereavement; and PTSD appears distinct from other anxiety disorders in terms of memory components (e.g., traumatic amnesia). PTSD also has similarities with dissociative disorders (Brett, 1996).

Brett (1996) argues that classification of PTSD as an anxiety disorder can narrow options in the following ways: Viewing PTSD as one syndrome can prevent study of other forms or variations of PTSD (p. 124); the diagnosis is restricted to essential features, with the concomitant risk of focusing treatment at

only those essential features and failure to consider and treat the associated features to PTSD; and looking at PTSD at only one point in time may result in failure to notice and study fluctuations and variations of the disorder over time. Different treatment approaches may be required for different symptoms and at different points in time as the symptoms of the disorder fluctuate and vary. For example, child sexual abuse survivors may be relatively symptom-free until later normative events (e.g., they have a child of their own) trigger their earlier traumas. Thus, clinicians should remain aware that previously traumatized individuals can become symptomatic during or following normative life events (e.g., delayed PTSD).

Moreover, empirical studies that have looked at the risk factors for PTSD may not have sufficiently taken into account other important variables (such as type of trauma experienced) or the potential confounding effects of third variables. For example, studies may confound symptoms from other DSM constructs with symptoms of PTSD, such as through the failure to assess and rule out differential diagnoses. Thus, when looking at the effectiveness of one's treatment for a given disorder, it is important to correctly assess the problem from the start.

Treatment Targets

Simple PTSD

To the extent that core posttraumatic symptoms are learned responses to overwhelming events, exposure and desensitization/habituation and cognitive restructuring appear sufficient for symptom management. As noted by Briere (in press), desensitization of traumatic distress requires exposure to the trauma stimuli, activation of the conditioned emotional and cognitive response to the trauma, inclusion of disparate information, and cognitive-emotional reprocessing. It is not clear that complete amelioration of the symptoms will result (e.g., Bouton, 1994).

Briere (in press) holds that the intrusive and avoidant symptoms of PTSD represent natural healing processes in response to trauma. That is, he posits that intrusions reflect how the individual is attempting to process the traumatic experience, whereas avoidance is a natural attempt at titrating intense distress. In short, he views intrusions and avoidance as a natural, innate form of systematic desensitization.

Counter-conditioning techniques appear to effectively reduce posttraumatic symptoms of intrusions and hyperarousal. This is borne out by empirical evidence (Rothbaum et al., 2000). Techniques that have clients experientially return to their original traumas through various routes of guided,

gradated exposure (e.g., titrated imaginal exposure), while simultaneously introducing information that is discrepant from the original trauma (e.g., the safety of the therapeutic setting; a relaxation response through muscle relaxation or laughter), assists clients in tolerating the affective distress from the trauma and thus facilitates their habituation to it.

It is less clear as to whether desensitization is key with regard to helping clients overcome their initial avoidance of traumatic memories (e.g., see van der Kolk, McFarlane, & van der Hart, 1996). There is some evidence (Brom, Kleber, & Defares, 1989) that dynamic psychotherapy is helpful for mastering avoidance, after which desensitization and cognitive restructuring can occur. Conversely, body-oriented approaches (Ogden & Minton, in press), can teach clients to feel safer in their bodies and to defend themselves against unwanted intrusions (Ogden & Minton, 2001). This sense of somatic safety can function to reduce tendencies toward subsequent avoidance. Somatic safety can also function as disparate information when exposed to fearful stimuli. If clients engage in a high degree of avoidance when exposed to traumatic memories, desensitization will be precluded because the emotional and cognitive responses to the traumatic memory will be immediately shut down or will not be activated at all. With dissociative clients, avoidance is likely associated with a different dissociative system—*i.e.*, the system that has not integrated the traumatic memory. Thus, it seems that there may be parallel reactivity of two dissociative systems in such situations, which makes treatment more complex.

Chronic and Complex PTSD/Associated Features

Active ingredients involved in effective trauma treatments might become more complex as a function of the presence, nature, number, and role of various risk factors for a given client, as well as the type, severity, and numbers of traumatic events experienced. Moreover, if empirical evidence further supports the notion of PTSD as a stress disorder with particular physiological indices (e.g., cortisol irregularities) (Yehuda, 1999b), as well as the possibility that there are different types of PTSD and variations in course of the disorder over time, then other treatment ingredients are conceivably important. For example, there is some evidence to the effect that prolonged stress and/or trauma leads to hippocampal atrophy and thus impairs memory (e.g., see McEwen, 2000). As such, for clients who have memory impairments or who evidence other neurological disruptions, interventions may need to be modified to take into account cognitive processing deficits, and pharmacotherapy may be an important treatment component.

Thus, it is hypothesized that in more complex cases, treatment targets, and hence active ingredients, might depend on the nature of the event, the

presence and severity of the particular risk factors in a given situation, and the particular form that the resultant symptoms take. Clearly, some of the risk factors (*e.g.*, genetics, family history of disorder, gender⁷, intelligence, biological alterations, and temperament) are not amenable to treatment, or are less amenable to treatment than others. Treatment outcome may thus depend, in part, on the presence of these risk factors.

Some of the other identified risk factors appear to be more amenable to treatment. For example, certain pre-morbid or co-morbid disorders (*e.g.*, substance abuse disorders) can be targets of treatment prior to desensitization of the trauma. When the risk factors consist of (presumed) temperamental factors (*e.g.*, hyper-reactivity to stress) or associated features, safety and stabilization will be the initial focus of treatment, such as through skills training (Linehan, 1993), identification of feeling states (van der Kolk, McFarlane, & van der Hart, 1996), pharmacotherapy⁸, working with and desensitizing transference material (Briere, *in press*), and so forth.

To the extent that disruptions in early attachments lead to non-conscious and dysfunctional schemata of self, others, and relationships, and to the degree that they also constitute a risk factor for PTSD, the therapeutic relationship becomes a source of distress (through transference) that also requires desensitization. This may be particularly important when working with clients whose symptoms and schemata result in significant interpersonal disruptions (see Briere, *in press*, for a thorough discussion of this concept). Fostering the therapeutic alliance may be particularly challenging.

As noted by van der Kolk, McFarlane and van der Hart (1996), treatment targets may need to vary at different stages of the client's life and at different phases of chronic PTSD, and may also need to vary as a function of the predominant symptoms that are currently expressed by the client.

Summary and Conclusions

In summary, it has been argued that exposure, desensitization, and cognitive-emotional processing are active mechanisms of therapeutic change for symptoms of PTSD. It is also argued that exposure, desensitization, and processing are key mechanisms for particularly chronic and complicated forms of PTSD and

⁷ Gender may not constitute a direct risk factor, but may function to increase the risk of exposure to certain traumatic events, such as rape (Kessler et al., 1999).

⁸ SSRIs have been found to be very effective in the treatment of PTSD (Friedman, Davidson, Mellman, & Southwick, 2000).

associated features; however, additional mechanisms of change are likely required for complete and successful treatment in the latter. Finally, for individuals who are severely traumatized and incapacitated by their symptoms, teaching symptom management skills in combination with medication might be the most that can be done, until current techniques are refined, further developed, and subjected to continual study.

To the degree that risk factors function to determine both the development of PTSD and recovery from trauma, it is plausible that targeting risk factors (when possible) may help both in the treatment of PTSD and in preventing recurrences of PTSD over time. This hypothesis is in need of clinical and empirical study.

Studies that have looked at risk factors for PTSD vary in terms of instrumentation, sampling methods, types of traumas studied, and other research design factors. These variations have led to different studies reporting some conflicting results. Moreover, most studies use retrospective or cross-sectional designs rather than prospective designs. As such, knowledge of etiological factors, interactions, and pathways is not conclusive at this point in time. In my view, it has not been ruled out that particularly severe traumas, *per se*, can result in PTSD in the absence of risk factors.

One of the limitations of this treatment conceptualization is that it presumes that treatment should target etiological factors; however, there is no *a priori* reason to believe that treatment effectiveness requires targeting of etiological factors. It may be that exposure, desensitization, and processing are effective components for the treatment of PTSD, regardless of the exact causal nature of the symptoms.

Another limitation is that this view does not take into account the synergistic effects of etiological/risk factors, nor does it take into account the synergistic effects of various treatment ingredients. It is possible that various combinations of treatment components (*e.g.*, therapist warmth, empathy, safety, structure, exposure, relaxation, cognitive restructuring, inclusion of body-focused techniques, and mastery experiences) combine in a manner such that the synergistic effects are particularly efficacious in terms of treatment outcome.

Ideas for Clinical and Empirical Study

What follows are suggestions for those who wish to subject their experimental treatment approaches to systematic clinical (case) study, followed by a few brief suggestions for empirical study in relation to the roles of various etiological pathways and variables.

Clinical Study. As noted by van der Kolk, McFarlane, and van der Hart (1996) results from the Koach project (Solomon *et al.*, 1992) show that subjective impressions of treatment success (*i.e.*, anecdotal reports) are not always borne out by objective data. As such, when clients appear to improve, objective ratings of their symptoms may fail to substantiate those impressions. As such, some clinicians may wish to conduct case study analyses of their treatment approaches. When case studies meet specific criteria, they offer information that can approximate the information that is obtained by experimental research (Kazdin, 1998).

Kazdin places case study criteria into two broad categories. The first refers to characteristics of the case, and the second refers to threats to internal validity. Documenting characteristics of the case functions to provide information that is important for ruling out threats to internal validity (internal validity refers to the evidence that observed treatment effects are actually due to the treatment, and not to other factors).

Characteristics of the case include (1) *the type of data that is obtained* (*e.g.*, self-report indices, physiological measurements, *etc.*, as opposed to anecdotal reports). According to Kazdin (1998), "...anecdotal reports are usually not sufficient to conclude that changes really occurred in client behavior" (p. 406) and "scientific inferences are difficult if not impossible to draw from anecdotal information" (p. 406); (2) *the frequency with which measurements are taken*. Taking several measurements over the course of treatment and follow-up assists in controlling for statistical regression to the mean (which can lead to inaccurate findings of treatment effectiveness) and other testing artifacts that can confound results; (3) *stability and course of the disorder*. Stability and course of the disorder will influence the interpretation of the observed treatment effects. For example, if a given treatment procedure results in changes during the acute phases of PTSD, it is not clear whether the changes are due to the treatment, or simply to natural healing (*e.g.*, Briere, *in press*). However, with chronic PTSD, changes in symptoms that occur immediately following the treatment and are maintained over follow-up periods, suggests that the treatment is what led to the changes; (4) *whether observed effects are immediate and marked*. When effects are immediate (occur in close proximity to the interventions) and marked (result in considerable changes in the severity of symptoms), it is more likely that the changes are due to the treatment intervention. As such, third variables can be more easily ruled out as causal factors; and (5) *the use of multiple, diverse cases*. The more cases that show change following treatment, the less likely it is that extraneous factors are that which is leading to the observed changes.

All of the above information is useful in terms of providing evidence for internal validity. Threats to internal validity include historical and maturation effects, testing effects, instrumentation (*i.e.*, using different assessment measures at different times can distort results), and statistical regression to the mean, which cannot be easily ruled out with simple pre-post measurements. Thus, for given clients, the clinician might wish to obtain informed consent to include the client in case study analyses and administer the same measures of PTSD and associated features to each client before treatment begins, immediately after treatment ends, and at several pre-determined follow-up times. In addition, the clinician should keep detailed records that document the nature and type of the trauma(s), the stability and course of PTSD and associated features for each client, whether observed changes are immediate and marked, and demographic data for each client (*e.g.*, age, sex, and so forth). It is also recommended that clinicians document the risk factors present. (See Appendix B for a sample Case Study form).

Empirical Study. It is recommended that further study on etiological factors in PTSD take into consideration the following: (1) when assessing for PTSD, take steps to consider and rule out differential diagnoses; (2) utilize large sample sizes to allow comparisons of effects of different types of traumatic stressor events and their interactions with risk factors; (3) consider creative ways in which to measure the stimulus properties of the traumatic stressor event (*e.g.*, severity of injury) when doing retrospective studies; (4) utilize research design and statistical analyses to assess the effects of proposed mediating and moderating variables; (5) identify physiological markers (*e.g.*, cortisol) (Yehuda, 1999b) that might differentiate PTSD from other disorders. For those interested in the topic of empirical studies, see Yehuda (1999a) for further issues and ideas in studying risk factors in PTSD.

In closing, these volumes provide the reader with several diverse and interesting treatment applications for use with traumatized clients. Some of these approaches (*e.g.*, cognitive-behavioral therapy and EMDR) have strong evidence for their treatment efficacy with PTSD. The evidence for other of the approaches consists largely of case study data. It is my view that the professional community would be at an advantage if it were more fully exposed to the findings observed with case studies (*e.g.*, Ogden & Minton, 2001). To this end, systematic analysis of clinical case material (*e.g.*, Kazdin, 1998), written up and published in professional journals, would assist in this endeavor.

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Appendix A

Client Identification: _____

Risk Factors Checklist

- Extended duration of trauma(s) _____
- Significant life threat _____
- Severe injury _____
- Loss of life and/or property _____
- Close proximity to the event _____
- Prior history of other DSM disorders _____
- Gender female
- Family members who have PTSD _____
- Family members who have a Mood disorder _____
- Family members who have an Anxiety disorder _____
- Prior traumas _____
- Peritraumatic dissociation _____
- Physiological stress reactions (if known) _____
- Neurocognitive sequelae (e.g., memory impairments) _____
- Personality factors _____
- History of Child maltreatment _____

***Total Number of Risk Factors:** ____/16____

Appendix B

Case Study Form

Date: _____
Client Identification: _____
Age at treatment start date: _____
Sex: _____
SES: _____
Race: _____
Marital Status: _____

Type and duration of treatment(s) given:

Type and Nature of Trauma(s):

Presence and Nature of Risk Factors:

Diagnosis:

Length of time with PTSD at intake: _____

Course of PTSD over time: _____

Associated

Features:

