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The role of trauma and dissociation in cognitive-behavioral psychotherapy outcome and maintenance for panic disorder with agoraphobia

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Abstract

The relationship between traumatic experiences and dissociation with pretreatment psychopathology and rates of recovery, relapse and maintenance for patients receiving cognitive-behavioral treatments for panic disorder with agoraphobia (PDA) were investigated. One-hundred and forty-seven subjects who met DSM-III criteria for agoraphobia with panic attacks and who completed participation in one of two previously conducted treatment outcome studies were mailed packets containing measures to assess history of trauma, victimization and dissociation. Eighty-nine of these were returned and completed sufficiently to be included in the present study. It was hypothesized that a variety of trauma-related variables (e.g. history of traumatic experience, type of trauma, age at which the trauma first occurred, perceived responsibility, social supports available, self-perceived severity, level of violence, and whether or not the traumatic event was followed by self-injurious or suicidal thoughts and/or behaviors) and dissociative symptomatology would be predictive of (1) greater psychopathology at pretreatment, (2) poorer treatment response and (3) higher relapse rates and poorer maintenance over a 1 year longitudinal follow-up. These hypotheses were supported by the findings and the theoretical, empirical and clinical implications are discussed.

1. Introduction

The purpose of this investigation is to examine the relationships between traumatic experiences and dissociation with pretreatment psychopathology and rates of recovery, relapse and

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maintenance for patients receiving cognitive-behavioral treatments for panic disorder with agoraphobia (PDA).

The cost to those persons suffering from PDA are varied and often quite serious. This disorder has been associated with chronic debilitating anxiety, a self-perception of poor physical or emotional health, increased alcohol abuse, excessive utilization of health care and emergency health care services, unemployment, financial difficulties, stress on important interpersonal relationships, a general sense of demoralization and suicide attempts (Weissman et al., 1978; Chambless, 1982; Weissman et al., 1989; Markowitz et al., 1989; Johnson et al., 1990). Johnson et al. (1990) found that the rates of suicide attempts were significantly increased in subjects with panic disorder if it was comorbid with another disorder (23.6%). However, this rate dropped to 7% if the panic disorder was uncomplicated (not comorbid) and to 1% for subjects who did not receive a psychiatric diagnosis. Given this information, there is additional cause for concern because persons with anxiety disorders tend to underutilize mental health services (Shapiro et al., 1984).

Unfortunately, the lives of those who experience symptoms of PDA are oftentimes further complicated by the difficulties associated with comorbid disorders. For example, major depressive disorder occurs in individuals with panic disorder at a frequency of 50–65% and in two-thirds of these cases the panic disorder preceded the depression (DSM-IV). Other disorders comorbidly associated with panic disorder include substance abuse, social phobia, obsessive–compulsive disorder, specific phobias and generalized anxiety disorder (Barlow, 1988; Stein et al., 1989; Cox et al., 1990; Robins and Regier, 1991; DSM-IV). Barlow (1985) used the anxiety disorders interview schedule (ADIS) to assess comorbidity and found that approximately half of the subjects with agoraphobia received no other DSM-III anxiety disorder diagnosis, but the remaining subjects received one (20%), two (27%) or three or more (10%) secondary diagnoses. Additionally, approximately 50% of patients with PDA also exhibit Axis II comorbidity (Mavissakalian and Hamann, 1986; Friedman et al., 1987; Reich, 1988).

Results from an epidemiologic catchment area (ECA) survey sponsored by NIMH found that 2.8 to 5.7% of the randomly sampled populations met criteria for agoraphobia, with an additional 1% experiencing panic disorder (Myers et al., 1984). However, a more recent ECA study, Robins and Regier (1991), found that only 1.5% of their subjects met DSM-III criteria for panic disorder at some time in their life, with about a third of these persons also meeting criteria for agoraphobia. In the more recent study, rates for panic disorder were cross-sectionally higher in persons ages 35–44 and lowest in older persons ages 65 or older. Peak age of onset was typically in the mid-twenties and no significant differences were found among African-Americans, Mexican-Americans and Caucasians. In a previous study, Robins et al. (1984) found that college graduates across three ECA sites exhibited lower agoraphobia prevalence rates than noncollege graduates (2.9 and 6.1%, respectively). Whether these findings can be attributed to higher levels of education or higher socioeconomic standing is unclear.

In the Agoraphobia Program at the University of Pittsburgh, the average duration of illness for 100 patients with PDA seen in treatment was approximately 11 years (Michelson, 1987). However, there are few studies examining the long-term course of PDA. Findings to date suggest that in most cases it is a chronic disorder that intermittently increases and declines in severity and those who experience PDA tend to have a more severe and complicated course (Wolfe and Maser, 1994).

To date, the majority of literature connecting trauma to panic disorder and agoraphobia has focused on correlational research and the possible etiological role of trauma in the development of PDA. There are no published studies examining the role of trauma as a predictor of pretreatment psychopathology and treatment outcome and maintenance for PDA. The primary aim of the present investigation was to examine the role of trauma and dissociation in pretreatment status, treatment outcome and longitudinal maintenance. This study investigates the relation of prior traumatic experiences and a variety of trauma-related constructs with greater pretreatment psychopathology and poorer treatment outcome in PDA patients receiving cognitive-behavioral treatment protocols across two NIMH-supported studies. In addition, given that prior research has found higher levels of dissociation associated with prior trauma, its potential role in treatment outcome for PDA is also examined.

In the following sections, the role of stress and, more specifically, the role of traumatic stress as an antecedent to the onset of PDA will be discussed. Then, several etiological models of PDA and traumatic stress will be presented to give background and context for possible mechanisms suggesting that trauma could play a role in treatment outcomes for persons with PDA. Additionally, the overlapping symptoms and cognitive schemes for individuals with PDA and individuals who have experienced traumatic stressors will be addressed in light of possible interactions between the two and complications that might arise in treatment for persons dealing with both conditions. Then, dissociation, which is commonly believed in the field of traumatology to be associated with traumatic experiencing, will be discussed, followed by the presentation of several theoretical models of dissociation. As was the case for presenting various models of traumatic stress, the discussion of dissociation models will provide background and context for the role dissociation may play in treatments for PDA. Finally, the hypotheses of the present investigation will be presented.

1.1. The role of stress

It is generally recognized that the occurrence of stressful life events typically precedes the first panic attack in patients who later develop panic disorder and agoraphobia by three to twelve months (Michelson, 1987). Barlow, in a review of studies looking at this topic, found that approximately 80% of patients with panic disorder experienced at least one major negative life event preceding their first panic (Barlow, 1988). As Barlow points out, Roth (1959) found that 96% of their sample of 135 agoraphobics reported some type of stress preceding the onset of the disorder. Some of these stressors were categorized as follows: bereavement or a suddenly developing serious illness in a close relative or friend (37%), illness or acute danger to the patient (31%) and severance of family ties or acute domestic stress (15%). Tearnan et al. (1984) concluded that stress may play a major role in the development of panic. The stress-diathesis model is well-known and posits that an existing proclivity towards a certain disorder may be realized if a given individual is subjected to stress, which presumably weakens the ability of the individual to defend against the onset of the disorder. Next, the role of trauma as an extreme form of stress will be considered.

1.2. Trauma

In the field of traumatology, what constitutes a 'traumatic' event or stressor has been a matter of considerable debate. Peterson et al. (1991) have outlined the essential characteristics of traumatic events: (1) serious life threat, (2) serious threat to one's physical integrity, (3) serious threat or possible harm to one's children, spouse, close relatives, friends, (4) sudden destruction of one's home or community, (5) seeing a serious injury or killing, (6) physical violence and (7) learning about serious harm or threat to a relative or family member. These characteristics are consistent with those delineated in the DSM-IV criteria of an 'extreme traumatic stressor' for posttraumatic stress disorder (PTSD) (APA, 1994) and they will serve as the defining characteristics of a 'traumatic event' for the purposes of the present study. The DSM-IV also requires that the person's response to the event must involve intense fear, helplessness or horror (or disorganized or agitated behavior in children). This requirement was altered from the DSM-III and DSM-III-R criteria which stated that the stressor must be psychologically distressing, markedly distressing to almost anyone and outside the range of usual human experience.

A great deal of the research conducted in the field of traumatology has been directed to the study of PTSD. For the purposes of the present investigation, the specific criteria and diagnosis of this disorder are not of particular import. Rather, the generalizations which can be drawn from this research will be useful in this discussion of traumatic experiencing. And, later in this paper, models more typically utilized in the discussion of PTSD will be discussed as they relate to traumatic experiencing without an emphasis on the specific diagnosis of PTSD.

March (1990) has stated that subjective perception of life threat, perceived potential for physical violence, the experience of extreme fear and attribution of personal helplessness are all factors that affect whether or not an event will induce PTSD. Because the individual's subjective experience of an event is so central to whether it will be experienced as traumatic or not, an inclusive list of traumatic events or stressors cannot possibly be generated. However, some exemplars of commonly discussed traumatic events in the traumatology literature are: military combat, violent assault (sexual assault, physical attack, robbery, mugging), being kidnapped, being taken hostage, terrorist attack, torture, incarceration as a prisoner of war or in a concentration camp, natural or manmade disasters, severe automobile accidents, diagnosis of a life-threatening illness, sexual molestation (with or without violence), observing the serious injury or unnatural death of another person and witnessing a dead body or body parts (DSM-IV).

Several factors contribute to the difficulty in calculating incidence and prevalence rates of traumatic events. The first has been pointed out by Barlow (1988) in a discussion of the incidence and prevalence rates of PTSD. Barlow states that a review of studies looking at PTSD rates among persons who have experienced traumatic events 'exhibits striking inconsistencies'. Approximately equal numbers of studies show either exceptionally high rates of lifetime PTSD (24–70%) or exceptionally low rates (1–6%) (Kluznik et al., 1986; Helzer et al., 1987; Foy et al., 1987; Pynoos et al., 1987; McFarlane, 1989; Shore et al., 1989; Winfield et al., 1990; Breslau et al., 1991; APA, 1994). This inconsistency is apparently present across different types of traumatic events. However, Barlow hypothesizes that if factors such as proximity or level of exposure to the traumatic event were to be looked at, the rates of PTSD

across many of these studies would very likely converge in accordance with the subjects' level of perceived threat. A second difficulty is that PTSD rates may not necessarily be an accurate estimate of the incidence and prevalence of specific traumatic events. Hence, the frequency, duration and severity of traumatic events may need to be examined individually.

In a review of PTSD literature, Davidson and Fairbank (1993) point out that this disorder exhibits a high level of comorbidity. In two ECA studies conducted in St. Louis and North Carolina (Helzer et al., 1987 and Davidson et al., 1991, respectively) lifetime comorbidity rates were found to be 80 and 62% for those persons diagnosed with PTSD. These numbers dropped to 33 and 15% for respondents with no posttraumatic stress symptoms. The highest rates of comorbidity were associated with somatization disorder, panic disorder, obsessive compulsive disorder, social phobia, generalized anxiety disorder, substance abuse, depression and schizophrenia (Shore, 1986; Helzer et al., 1987; Davidson et al., 1991).

1.3. Relation of trauma with panic disorder and agoraphobia

In a large community survey of adult women, Saunders et al. (1992) found that one-third of the women in their study had been victims of rape, molestation or sexual assault not involving physical contact before the age of 18 years. Of these, child rape victims were found to be at significantly greater risk of developing agoraphobia. Another study that has examined the long-term effects of childhood sexual abuse in women found that the risk for lifetime diagnosis of panic disorder was greater in a severely abused group than in women who experienced less severe abuse or no abuse (Walker et al., 1992). These authors also suggested that lifetime diagnosis of panic disorder was actually predictive of former severe childhood abuse. These findings have been supported by an investigation conducted by Murrey et al. (1993) in which the prevalence rate of a childhood history of sexual abuse in women diagnosed with an anxiety disorder was found to be 48.5%. Specifically, high rates of reported sexual abuse were found among women diagnosed with panic disorder (43%) and obsessive compulsive disorder (40%).

Other studies have found similar results. Pribor and Dinwiddie (1992) have suggested that among women who experienced incest, rates of anxiety disorders (especially panic disorder, agoraphobia, social and simple phobia) are significantly higher than base population rates. Additionally, with increasing severity of incestuous abuse, the risk for development of psychiatric disorders increases. Zlotnick et al. (1996) found a relationship between childhood sexual abuse and symptoms of somatization, dissociation, hostility, anxiety, alexithymia, social dysfunction, maladaptive schemes, self-destruction and adult victimization. This cluster of symptoms was seen as representing a newly proposed complex posttraumatic stress disorder called disorder of extreme stress not otherwise specified (DESNOS).

In a Los Angeles Catchment Area study estimating the lifetime and current (defined as the prior six months) prevalence of psychological reactions to child sexual abuse, panic disorder was one of several diagnoses showing an elevated prevalence among those who had been abused, even when controlling for gender, ethnicity, age and educational level (Stein et al., 1989). This study also concluded that the sexual abuse trauma developmentally preceded the onset of panic disorder. In a review of the literature on major life events that precede the onset of agoraphobia, Foa et al. (1984) found that much of the literature pointed to loss of significant others and physical threat or illness as precipitants of agoraphobia. Since that

review, the literature continues to produce evidence supporting loss of a significant relationship as a traumatic stressor associated with panic disorder and agoraphobia. Faravelli (1985) found that patients with a DSM-III diagnosis of panic disorder showed a significant excess of major life events, such as the death (loss) or severe illness (threat of loss) of a cohabiting relative or self, in the two months preceding onset of symptoms. Much of this literature has focused on the loss of a parent by death, divorce or abandonment as being an example of one of the more traumatic 'loss/separation' events experienced by subjects.

In related studies, panic disorder and agoraphobia have been connected to: death of either parent and maternal, but not paternal, separation (Kendler et al., 1992); maternal death and parental separation or divorce (Tweed et al., 1989); and maternal separation for any reason and parental divorce (Faravelli et al., 1985). It appears from this research that loss of, or separation from, either parent may be associated with later development of panic and agoraphobia, but loss of the maternal relationship seems to be more predictive.

In response to the studies which suggest high rates of developmental trauma among adult anxiety disorder patients, David et al. (1995) attempted to replicate these findings in patients with PDA and social phobia. They hypothesized that patients with histories of sexual or physical abuse would exhibit higher rates of social phobia and that patients with histories of traumatic loss or separation would exhibit higher rates of PDA when compared to nonclinical populations. The results of this study supported the hypothesized relationship between sexual/physical abuse and social phobia, but no relationship between separation/loss and PDA was suggested.

To summarize, a significant portion of research conducted to date exploring the relationship between a history of traumatic experiences and PDA has shown that individuals who have experienced childhood physical/sexual abuse or traumas of loss/separation are at significantly higher risk of developing PDA and a variety of other anxiety disorders. Theoretical models addressing the etiological development of PDA will be reviewed next.

1.4. Etiological models of PDA

1.4.1. Cognitive theory of panic disorder

The cognitive theory of panic disorder was developed by several independent researchers during the mid- and late-1980s. Beck and Emery (1985), Clark (1986) and Barlow (1988) were among the first to introduce this theory. Beck has summarized the basic processes at work in the cognitively mediated model of panic disorder (Beck, 1988). Individuals who are panic-prone seem to be particularly sensitive to internal sensations, both bodily and mental, which are perceived as being abnormal. These individuals are hypervigilant to such sensations and tend to focus a great deal of attention on them, especially if they cannot think of a nonpathological explanation. For many, alternative, more serious attributions are made and the sensations are then seen as signs of impending disaster. Beck points out that the imagined disasters tend to fall into three categories: biological (death), mental (insanity or loss of consciousness) or behavioral (loss of control). The hypervigilance toward the unexpected sensations is involuntary and increases the individual's belief that danger is imminent. This, in turn, leads to increased activity of the autonomic nervous system which only worsens the troubling sensations. Thus, a vicious feedback loop is created. An important characteristic of

panickers, according to Beck, is their inability to make realistic appraisals of the threat they are experiencing. It is this quality that exacerbates what could have been severe anxiety and creates a full-blown panic attack.

Similarly, Clark's cognitive theory of panic (Clark, 1986) proposes that individuals who experience panic attacks misinterpret benign bodily sensations as signs of physical or mental catastrophe (e.g. believing palpitations to signal an impending heart attack). "This cognitive abnormality is understood to lead to a positive feedback loop in which misinterpretations of bodily sensations produce increases in anxiety, which in turn strengthen the sensations, thus producing a vicious circle that culminates in a panic attack" (Clark, 1986). According to Clark, if the theory is correct, it should be possible to treat naturally occurring panic attacks by helping patients identify and change their misinterpretations of bodily sensations.

1.4.2. Fear of fear — classically conditioned agoraphobia

Chambless and Goldstein (1982) discuss an etiological theory of the development of agoraphobia in terms of classically conditioned 'fear of fear': they state that panic attacks occur as an unconditioned emotional response to prolonged stress and conflict. As such, they are unpredictable. Therefore, the most consistent stimuli available to the individual are internal ones, such as one's own heartbeat and blood flow and anxiety becomes conditioned to these internal cues. Second-order conditioning, mediated by stimulus generalization, then leads to the development of avoidance behaviors — first towards places where panics have already been experienced and then secondarily towards places that have similar characteristics. Typically, these places possess qualities that increase the agoraphobic individuals belief that escape to safety is going to be difficult or impossible. Situations that are difficult to escape from become discriminative stimuli for extremely aversive consequences, both real and imagined and they subsequently develop the properties of conditioned stimuli and will be avoided.

Next, various models of traumatic stress will be discussed in order to provide background for the etiology, course and treatment. The implications that these models could potentially have for treatment of PDA will then be addressed.

1.5. Etiological models of traumatic stress

1.5.1. Horowitz's information processing model

Horowitz (1979) suggests that catastrophic events contain a great deal of internal and external information — most of which cannot be incorporated within an individual's existing cognitive schemes because it lies outside the realm of normal human experience. The inability to incorporate this information results in 'information overload'. Because the information cannot be integrated with the self, it is kept out of conscious awareness, and subsequently remains in its unprocessed, active form. The defenses of denial and numbing help keep the traumatic information unconscious initially, but gradually it is brought into conscious awareness and 'digested' as a function of what Horowitz refers to as a 'completion tendency'. This form of incremental information processing is experienced as intrusive cognitions such as flashbacks, repetitive nightmares, unwanted thoughts, etc. However, when the ego experiences 'information overload' during these intrusive cognitions, denial and numbing are again utilized. A pattern of oscillation between the experiences of intrusion and the defensive processes of

denial and numbing occurs until all the traumatic information is processed, incorporated into the 'inner schema of the self' and is no longer maintained in its active state (Horowitz and Wilner, 1976; Horowitz, 1979). Horowitz (Horowitz, 1979, 1986) summarizes the stages of reaction to massive stressors as follows: massive stress > avoidance (denial/numbing) > oscillation period > transition > integration.

1.5.2. The psychosocial model

This model, which was proposed by Green et al. (1985), is an extension of Horowitz's information processing model (Peterson et al., 1991). According to this model, a person experiences 'psychic overload' until "the nature, intensity and meaning of a traumatic experience is integrated into his/her existing schemata of reality" (Peterson et al., 1991). During the psychic overload, ego defenses and coping mechanisms fail. If the person is in a healthy or favorable environment, the chances of working through the trauma are increased. Green et al. (1985) argue that factors of the environment which may affect a person's ability to recover from the trauma include: quality and availability of social supports, protectiveness of family and friends, attitudes of the society, intactness of the community and cultural characteristics.

Some characteristics of traumatic experience important to long-term responses have been summarized by Green et al. (1985): (1) severity of the stressor, (2) duration of the trauma, (3) warning speed of onset, (4) degree of bereavement, (5) degree of displacement of the person or community, (6) proportion of the community affected, (7) degree of life threat, (8) exposure to death, (9) dying, destruction or combat stress (10) active or passive participation, (11) degree of moral conflict and (12) potential for and/or control over reoccurrence.

Personal characteristics of the affected individual are also an important factor in coping with traumatic events (Green et al., 1985). Some of these characteristics include the individual's ego strength, coping resources and defenses, preexisting psychopathology, prior history of stressful and traumatic experiences, specific behavioral tendencies, current Eriksonian psychosocial stage of development and various demographic factors (Peterson et al., 1991).

1.5.3. Behavioral and information processing models

Keane et al. (1985) have offered the most comprehensive behavioral model of PTSD (Peterson et al., 1991). It is based on a 'two factor learning theory of psychopathology', which was first proposed by Mowrer. Two factor theory posits that psychopathology is a function of both classical and operant conditioning. First, a fear response is acquired through classical conditioning. Then, as a function of operant conditioning, the individual will avoid those classically-conditioned cues that evoke anxiety (Peterson et al., 1991). These cues can be external events, thoughts, people, specific times, etc., and they can be organized into clusters — a process referred to as 'patterning' (Peterson et al., 1991). An example of this which is relevant to the present discussion could be the troublesome sight of a leather belt similar to one used repeatedly against a boy by his father. The response to this cue may be a sudden wincing facial expression.

'Stimulus generalization' also occurs in PTSD. This concept holds that the more similar a new stimulus is to a conditioned stimulus, the stronger the response will be to that new stimulus (Peterson et al., 1991). In the above example, a razor strap made of leather may elicit a greater response than a leather purse strap because it more closely resembles the

aforementioned leather belt. Keane et al. (1985) suggest that 'higher-order conditioning' is involved in PTSD symptomatology. Once a cue has been conditioned to elicit fear, it too will become a source of fear and any cue that consistently precedes the conditioned cue will be able to elicit the same fear. By its very nature, traumatic experiences are aversive. According to the principles of negative reinforcement, behavior that lessens the likelihood of an aversive situation is reinforced and likely to be repeated. The avoidance behaviors and psychic numbing common in PTSD (and avoidance behaviors in most anxiety disorders, including PDA) are mediated by negative reinforcement (Peterson et al., 1991).

Keane et al. (1985) propose that constant exposure to a conditioned stimulus does not lead to the extinction of the conditioned fear response in PTSD due to the fact that conditioned stimuli account for only a portion of the memory of the traumatic event. PTSD sufferers actively avoid full exposure to stimuli that resemble the original trauma, including thoughts that are associated with the trauma. In addition, these authors suggest that 'state dependent retention' may interfere with memory recall. This concept refers to the differences between the cognitive and physiological state of the person during the traumatic event and the state the person is in at the moment of attempted recall. Without specific cues, incomplete recall results, leading to the continuation of symptoms (Peterson et al., 1991). These learning concepts are reflected in the information processing model discussed below.

An information processing model of PTSD was extended by Foa and Kozak (1986) from Lang's (1977, 1979) discussion of fear structures as a memory network. Such a network would include three kinds of information: "(1) information about the feared stimulus situation (2) information about verbal, physiological and overt behavioral responses and (3) information about the meaning of the stimulus and response elements of the structure" (Foa et al., 1989). The information structure is seen as an internal program of avoidance behavior. PTSD is distinguished from other anxiety disorders in that the initiating traumatic event very powerfully violates formerly held notions about safety by transforming the meaning of certain stimuli and responses from 'safety' to 'danger' (Foa et al., 1989). This is particularly true if a trauma is experienced in what was believed to be a safe environment. The result of this alteration is a loss of predictability and controllability for the individual, who then comes to live in fear (Foa et al., 1989).

This change in an individual's rules of safety leads to an expansion in the number of stimuli that become a part of the fear structure. "The pervasiveness of the stimuli, the intensity of the responses (both physiological and behavioral) and the low threshold for activation of the fear structure renders PTSD more disruptive to daily functioning. Many stimuli activate this structure, resulting in frequent bursts of arousal (e.g. startle) and re-experiencing of the events (e.g. nightmares, flashbacks), alternating with attempts to avoid or escape fear (e.g. numbness, behavioral avoidance, depersonalization)" (Foa et al., 1989). In order for this fear structure to be broken down, Foa and Kozak (1986) suggest that the fear memory must be activated and new cognitive and affective information which contradicts the existing fear structure must be integrated so that change can take place. Fear structures involved in PTSD are typically quite large and are easily matched and activated — but only partially: "situations that match specific elements in the structure may access only that part of it while the rest of the structure remains unactivated and therefore not available for change" (Foa et al., 1989). Additionally, the strong response information in the fear structure may encourage avoidance and result in short,

inadequate activation of the structure and subsequent maintenance of PTSD symptoms (Foa et al., 1989).

According to this model, amelioration of symptomatology occurs when the exaggerated probability of danger is decreased and the valence of stimuli or responses is altered (Foa et al., 1989). Two characteristics of individuals experiencing PTSD that may make these tasks more difficult are pointed out by Foa and her colleagues. The first has to do with the fact that most people assume that a situation is safe in the absence of information signaling danger, but for those experiencing PTSD, the lack of 'safety cues' (Rachman, 1977) may equate with the presence of danger (Foa et al., 1989). Because there are never enough safety signals to ensure that no danger exists, persons with PTSD are always on the alert. The second mediating factor has to do with the fact that some individuals experience anxiety and discomfort at a high negative valence ('anxiety is awful'), so they are more likely to avoid situations that exacerbate their anxiety (Foa et al., 1989). Hence, they are more likely to adopt strong behavioral and cognitive patterns of avoidance. Logically, this factor could have a mediating effect on the cognitive-behavioral treatment of an individual with PDA if that individual is also suffering from traumatic stress symptoms.

1.6. Overlapping diagnostic features of PDA and PTSD

Given the associations reported between traumatic experiences and panic disorder and agoraphobia as previously discussed, there is reason to hypothesize that some of the individuals presenting for treatment of panic disorder and/or agoraphobia may also be suffering from symptoms attributable in part to prior traumatic experiences. The fact that avoidance behaviors and hyperarousal are diagnostic correlates of both PDA and PTSD is potentially further evidence for this proposal. In PDA, avoidance is exemplified in the agoraphobic avoidance central to the diagnosis. In PTSD, efforts to avoid thoughts, feelings, discussions, activities, places and persons that serve as reminders of the traumatic event are symptomatic. It could be important in the treatment of PDA to understand whether certain situations are being avoided due to concerns with panic symptoms or if the avoidance is in response to prior traumatic experiences.

Hyer et al. (1996) in their study of 110 hospitalized Vietnam veterans found that chronic PTSD led to the use of emotion-focused and escape-avoidance strategies to cope with traumatic memories. The use of such strategies could plausibly contribute to increased agoraphobic symptoms. Avoidance in one sphere could easily contribute to avoidance in another as the strategy becomes generalized due to negative reinforcement principles. If so, then agoraphobic symptoms could be exacerbated by traumatic stress symptoms and, central to the purpose of this study, well-established patterns of avoidance-escape coping styles could make the implementation of exposure-based techniques more difficult. Individuals with symptoms of traumatic stress may very well be less likely to effectively use exposure strategies as they may have less tolerance for them. This assertion is supported by the findings of Scott and Stradling (1997) who found that only 57% of individuals with chronic PTSD being treated with exposure-based techniques actually complied with the treatment. Compliance was related to symptom severity and severity of comorbid depression. The authors concluded that

exposure-based treatments were not the 'treatment of choice' for some individuals with chronic PTSD. A similar situation may exist for individuals with PDA who also have trauma histories.

Physiologic hyperarousal is similarly present in both disorders. The criteria for panic attacks include symptoms such as accelerated heart rate, sweating, trembling or shaking, feeling dizzy or lightheaded or faint and chills or hot flushes. These symptoms have also been found to be associated with PTSD. Observations of increased physiological and autonomic arousal have frequently been reported in trauma patients. Studies of PTSD Vietnam Vets have shown tonic levels of increased arousal as measured by heart rate (Malloy et al., 1983; Pallmeyer et al., 1986) and systolic blood pressure (Pallmeyer et al., 1986). Horowitz et al. (1980) found that over 75% of patients with stress response syndromes feel tense or keyed up, nervousness, shakiness inside and fearfulness. Over 50% of these patients reported heart pounding or palpitations, becoming suddenly scared without warning, nausea, muscle soreness, hot/cold spells, faintness or dizziness, numbness or tingling, heavy feelings in the limbs, feeling a lump in the throat and heart or chest pains. Many of these symptoms overlap with the criteria for panic attacks. The experiencing of symptoms related to prior traumatic experience is very likely, in a person also suffering from panic disorder, to initiate or exacerbate a panic attack.

1.7. Dissociation

The APA (1994) recognizes dissociation as the "disruption in the usually integrated functions of consciousness, memory, identity or perception of the environment". Such a disturbance can be sudden or gradual, temporary or chronic. According to Cardena (1994), the term 'dissociation' is used for several purposes: to describe semi-independent mental structures or systems that are not available to conscious awareness, and/or not integrated with conscious memory, identity or volition; to represent a change in consciousness where the self and environment become separated; to describe a defense mechanism resulting in nonorganic amnesia, the splitting off of current physical or emotional pain and other alterations of consciousness. Spiegel et al. (1988) have stated that dissociation includes an intense focus on certain mental contents while simultaneously excluding others. They further explain that dissociation differs from repression in that repressed material excluded from awareness does not contain rules about the admissibility of other materials to consciousness. Cardena (1994) noted that Janet first proposed that dissociation can occur when 'vehement' emotions, including terror, are experienced. These emotions lead to a narrowing of attention and disorganization of the ordinary integrative functions of consciousness.

Dissociation has long been associated with traumatic experience, as exemplified in the DSM-IV criteria sets for PTSD and acute stress disorder which both contain dissociative symptoms. Approximately 44% of female clients presenting to a health center in crisis reported a childhood history of sexual abuse and this prior victimization was associated with dissociation as well as sleep disturbance, sexual difficulties, anger, suicide attempts and revictimization (Briere and Runtz, 1988). With regard to dissociation, similar results were found in a study of psychiatric inpatients. Saxe et al. (1993) reported that 15% of an inpatient population scored above 25 on the dissociative experiences scale (DES) and that 100% of these patients met DSM-III criteria for a dissociative disorder. These inpatients also reported significantly higher rates of childhood trauma. An investigation utilizing an inpatient population of adolescents

noted similar results (Sanders and Giolas, 1991). In a study of 100 multiple personality disorder (MPD) patients, Putnam et al. (1986) noted that, for a majority of these MPD patients, a history of significant childhood trauma was present: 83% reported sexual abuse, 68% reported sexual abuse in the form of incest, 68% reported concurrent physical and sexual abuse and 45% reported having witnessed a violent death, usually of either a parent or sibling. Chu and Dill (1990) also made an important contribution to this literature with their findings that DES scores were highest in patients who reported both physical and sexual abuse in childhood; additionally, childhood abuse by family members was strongly associated with higher levels of dissociative symptoms in adulthood, while abuse perpetrated solely by persons outside of the family was not associated in a clear way with dissociative symptoms. This pattern held true for both physical and sexual abuse.

1.8. Dissociation's interference with PDA treatment

1.8.1. Psychodynamic model

In their chapter on psychodynamic psychotherapy of dissociative identity disorder (DID), Barach and Comstock (1996), present a psychodynamic model of DID which may be extended to other dissociative processes. DID probably represents an extreme position on a continuum of dissociative processes ranging from normality to severe pathology. According to these authors, dissociation occurs in response to trauma, which is defined in terms of the internal experience of the child not external events, with the purpose of maintaining emotional stability by ignoring connections between events. The effects of trauma and dissociation are so pervasive that integrated psychological functioning is hampered and developmental deficits are incurred (Barach and Comstock, 1996).

One of these developmental deficits is the inability to develop a secure base of attachment to important others (Barach and Comstock, 1996). It is suggested that abused, abandoned and/or neglected individuals may detach (dissociate) themselves from awareness of their needs because others have historically not responded to their attachment behaviors during early childhood or these individuals may have developed disorganized patterns of attachment as a result of vacillating between fear of their caregivers and fear of their own needs. Later, perhaps in therapy, repressed and dissociated material may enter awareness triggering danger signals and initiating the panic cycle (Barach and Comstock, 1996).

In their review of this literature, Barach and Comstock (1996) also point out that infants who have been severely traumatized experience any affect as an intolerable reminder of earlier abuse. Later, as adults, these patients may try to avoid affect by utilizing a variety of dissociative defenses in order to keep strongly conflicting feelings and needs from entering awareness together. Important others in the dissociative patient's childhood may have failed to provide predictability and consistent caring (Barach and Comstock, 1996). Additionally, 'splitting' is discussed as a dissociative defense which allows an abused child to interact with the perpetrators of the abuse during those moments when the abuse is not occurring.

How might this psychodynamic model of dissociation aid in understanding the mediating effects of traumatic experience on cognitive-behavioral treatment for PDA? For those individuals who have experienced abuse, abandonment and/or neglect in their childhood, it would seem that the often-used graduated exposure techniques could lead to intolerable levels

of affect. As a result, these patients may become dissociative in a defensive attempt to escape their discomfort. If such dissociative processes were to occur, habituation may be interrupted and other therapeutic instruction may not be attended to or consolidated sufficiently.

1.8.2. Information processing model

Foa and Hearst-Ikeda (1996) have proposed an information-processing perspective of emotional dissociation in response to trauma. According to these authors, emotional experiences are oftentimes experienced long after the original traumatic events have taken place. This re-experiencing typically diminishes in both frequency and intensity over time; when it does not, psychopathology emerges. The symptoms exhibited are similar to PTSD and the authors suggest that the presence of such symptomatology may reflect an impairment in the emotional processing of traumatic experiences.

As discussed previously, Foa and Kozak (1986) extended Lang's (1977, 1979) bioinformational model of pathological fear in order to explain the mechanism by which cognitive-behavioral therapy is able to reduce pathological anxiety. As explained by Foa and Hearst-Ikeda (1996), a trauma memory can be viewed as a fear structure which includes information about stimuli and responses associated with the trauma in addition to information about their meaning. Trauma structures for most people undergo modification over time due to emotional processing of the information related to the trauma. Dissociation may be seen as a strategy used to avoid trauma-related aversive emotions and memories. Therefore, it is logical that dissociation could impede the information-processing necessary to modify the trauma structure (Foa and Hearst-Ikeda, 1996). ''Indirect evidence supporting the hypothesis that dissociation impairs emotional processing and hence impedes recovery comes from the repeated finding that dissociation during or immediately after the traumatic experience is associated with later psychopathology'' (Foa and Hearst-Ikeda, 1996). Thus, successful treatment of trauma-related distress would necessitate that dissociative tendencies be addressed so that repeated emotional processing of the traumatic memory is possible. The authors report that clinical observations, however, show that some traumatized individuals continue to dissociate during the reliving of the trauma which renders exposure therapy ineffective.

In the cognitive-behavioral treatment of individuals with PDA who also have a history of traumatic experiences, it is quite possible that the presence of elevated dissociation levels may interfere with the modification of fear structures based on misattributions of danger associated with the symptoms of anxiety. Another possibility is that exposure directed at the PDA fear structure may access portions of the PTSD trauma structure which may then lead to a subsequent re-experiencing of the trauma. This re-experiencing in turn may mobilize the individual's tendency to avoid emotional and physiological distress by dissociating thereby rendering the exposure treatment less effective.

1.9. Hypotheses

As stated in the preceding sections, trauma has been associated with both PDA and dissociative symptoms. It is hypothesized that the presence of a trauma history, a variety of trauma-related conditions and associated elevations in dissociation levels will be predictive of: (1) greater psychopathology at pretreatment, (2) poorer treatment outcome and (3) higher

relapse rates and poorer longitudinal maintenance. Two measures of traumatic experiencing will be used in this investigation. One will focus on sexual victimization, the other on more broad-based traumata. The sexual victimization trauma variables that are hypothesized to predict increased levels of psychopathology are: younger age, greater levels of violence, perceived responsibility for the traumatic event, perceived severity of the trauma, and lower levels of social support. Additionally, response to the traumatic sexual event with self-injurious or suicidal ideations or behaviors are also hypothesized to predict increased levels of psychopathology at each assessment phase. From the more broad-based measure of trauma, the number of PTSD symptoms reported following a traumatic event will be examined with the expectation that higher numbers of symptoms will be related to increased psychopathology. Three general types of trauma, (1) real or threatened loss of home, family member or close other, (2) interpersonal violence and (3) accidents, will be examined in see if any of these categories of trauma is a superior predictor of greater psychopathology.

2. Methods

2.1. Subjects

The following has been adapted from Michelson et al. (1988, 1996) with the authors' permission.

One-hundred and forty-seven subjects who met DSM-III criteria for agoraphobia with panic attacks and who completed participation in one of two previously conducted treatment outcome studies were invited to participate in the current retrospective study. Three to five years after the completion of treatment, subjects who had participated in one of the two NIMH-funded studies, Michelson et al. (1988) (study-I) and Michelson et al. (1996) (study-II), were mailed packets containing assessment measures to investigate history of trauma, victimization and dissociation. Of the 147 packets mailed out, 89 were returned and completed sufficiently to be included in the present investigation. Thus, all cells in the present study were made up of a sample of convenience.

Participation in the protocols of the original studies and the present retrospective study was voluntary, free of charge and in accordance with human use guidelines. Subjects were required to discontinue use of psychotropic medications at least two weeks prior to treatment in the original studies and routine compliance checks were conducted and revealed abstinence throughout the protocols.

Diagnostic and screening procedures were consistent across the original studies. Subjects underwent an initial psychiatric diagnostic evaluation and a detailed clinical-research interview with a licensed clinical psychologist and received a concurrent diagnosis of agoraphobia with panic attacks across both interviews. Additional inclusion criteria were onset prior to 40 years, duration of illness of at least 1 year, age between 18 and 65 years and a score >3 (moderate to severe) on the *global assessment of severity scale* (see Section 2.6). Exclusion criteria differed slightly between study I and study II.

In study I, persons with social phobia or anxiety neuroses without the typical fears of leaving home, entering public places or using public transportation were excluded. Also

excluded in study I were subjects experiencing a severe depressive episode or those with any history of affective disorder preceding the onset of agoraphobia or a diagnosis of organic brain syndrome, schizophrenia, obsessive–compulsive neurosis, antisocial personality, Briquet's syndrome, current substance abuse or alcoholism. Exclusion criteria for study II included diagnosis of organic brain syndrome, schizophrenia, severe personality disorder, mental retardation or current substance abuse.

Subjects who participated in study I were randomly assigned to either graduated exposure (GE), paradoxical intention (PI) or progressive deep muscle relaxation training (RT) treatment conditions. Of the 88 subjects who entered the clinical-research protocol, 73 completed the program. The distribution of dropouts across treatments was not statistically significant ($\chi^2 = 0.61$; $p = 0.73$) with attrition rates of PI = 5 (16%), GE = 6 (21%) and RT = 4 (14%). Final sample size per treatment condition were PI = 26, GE = 22 and RT = 25. These 73 subjects had a mean age of 37 years (range = 22 to 63; S.D. = 9.4) with 57 (78%) women and 16 (22%) men. Seventy-two subjects were Caucasian, one was black; 60 were married and 13 were single, divorced or separated. The average duration of illness was 10 years (range = 1 to 34); S.D. = 8.0) with a mean onset age of 26.8 years (range = 16 to 39; S.D. = 6.5). Overall, 89% of the subjects had received some form of previous outpatient treatment for an average duration of 32.4 months with no benefit. The mean number of protocol sessions attended was 11, which did not significantly differ across treatments.

Subjects in the study II were randomly assigned to either cognitive therapy plus graduated exposure (CT + GE), relaxation therapy plus graduated exposure (RT + GE) or graduated exposure (GE). Of the 92 subjects who entered the clinical-research program for study II, 74 completed the treatment protocol. The distribution of dropouts across treatments was not statistically significant ($\chi^2 = 3.19$; $df = 2$; $p = 0.20$) with attrition rates of CT + GE = 5 (17%), RT + GE = 10 (29%) and GE = 3 (11%). The attrition rates include all individuals who either discontinued treatment or were removed from the protocol for medical, psychiatric or noncompliance reasons. Drop-outs were replaced to ensure equivalent sample sizes across conditions. Final sample sizes per treatment were CT + GE = 25, RT + GE = 25 and GE = 24. These 74 subjects had a mean age of 37 years (range = 21 to 63; S.D. = 9.9) with 61 (82%) women and 13 (18%) men. Seventy-two subjects were Caucasian, two were black; 50 (67.6%) were married and 24 (20.3%) were single, (5.4% divorced, 5.4% separated and 1.3% widowed). The average duration of illness was 10.5 years (range = 1 to 37; S.D. = 9.0) with a mean onset age of 26 years (range = 11 to 39; S.D. = 6.2). Overall, 80% of the subjects had received some form of prior outpatient treatment for an average duration of 24 months, with limited or no benefit. The average number of protocol sessions attended was 13, which did not significantly differ across treatments.

As stated previously, 89 subjects completed and returned the questionnaires sent to them by mail and were included in the present investigation. The means for age at time of treatment, age of onset and duration of illness were computed and the results are presented in Table 1. The average age for this sample at the time of treatment was 37.27 (S.D. = 8.10). The average age at the time of PDA onset was 27.06 (S.D. = 6.97) and the mean duration of illness in years was 9.65 (S.D. = 7.55). Also presented in Table 1 are frequencies and percentages for gender, marital status and race. Approximately 85% of the sample consisted of females. Seventy-five percent of the sample was married and the categories of single and divorced/

Table 1
Demographics

	Mean	S.D.	Range
Age at time of treatment	37.27	8.10	34.00 (23–57)
Age of PDA onset	27.06	6.97	40.00 (13–53)
Duration of illness (years)	9.65	7.55	33.08 (~1–34)
	Frequency	Percentage	
Sex			
Females	76	85.4	
Males	13	14.6	
Marital Status			
Single	9	10.1	
Married	67	75.3	
Divorced/separated	9	10.1	
Widowed	2	2.2	
Cohabiting	2	2.2	
Race*			
Caucasian	85	95.5	
African-American	3	3.4	

*1 subject did not respond.

separated each made up about 10% of the sample population. Approximately 2% of the sample had lost their spouse to death and another 2% was cohabiting with a partner. Almost 96% of the sample was Caucasian, with the remaining 3–4% being made up of African-American individuals.

2.2. Treatment conditions and procedures for study I and study II

The treatment conditions and procedures for studies I and II are discussed in detail in Michelson et al. (1988, 1996).

2.2.1. Therapists

In both studies, three licensed clinical psychologists experienced in cognitive and behavioral treatments served as therapists and they were counterbalanced across treatment conditions. To ensure the treatment procedures were administered consistently, weekly meetings were held to discuss and review all treatment sessions. Excellent treatment integrity ratings ($>99\%$) indicate that the treatments were faithfully implemented.

2.2.2. Treatment and therapist credibility ratings

To examine possible differential levels of treatment and therapist credibility and expectancy of positive therapeutic outcome across the conditions, subjects completed ratings at each

assessment. No differences were found between therapists or treatments across any of the assessment phases.

2.3. Assessment

2.3.1. Studies I and II

For both of the original studies, a comprehensive assessment battery consisting of clinical ratings of severity, phobia, anxiety, panic, depression and generalization measures, as well as direct measures of agoraphobia in behavioral and self-report response systems, were administered at pretreatment, posttreatment and 1 year follow-up by independent research associates. See Michelson et al. (1988, 1985) for complete details of the assessment battery. The clinical assessment battery for studies I and II included an operationalized measure of endstate functioning (see Section 2.11), the global assessment of severity, the self-rating of severity, the phobic anxiety and avoidance scale, the fear survey schedule, the Taylor manifest anxiety scale, the panic attack form, the Beck depression inventory, the Hopkins symptom checklist, the subjective symptom checklist, the standardized behavioral avoidance course and the subjective units of discomfort scale.

2.3.2. Retrospective investigation of trauma and dissociation

Trauma and dissociation were measured in the present investigation 3–5 years posttreatment via packets mailed to subjects who had participated in studies I and II. Whereas, clinical ratings of global severity, anxiety, panic, depression, generalized psychopathology and direct measures of agoraphobia in behavioral and self-report response systems were completed at pretreatment, posttreatment and 1 year follow-up. The measures used in the present investigation are described below.

2.4. Trauma and victimization history

Trauma was assessed using the intergender relationships inventory (IGR) (Koss et al., 1987) and the traumatic experiences questionnaire (TEQ) (Davidson and Smith, 1990). The IGR was developed from an NIMH epidemiologic study on the incidence and prevalence of trauma in the general population. This instrument, which has been widely used in the traumatology field, has been found to be reliable and valid. It provides a detailed assessment of sexual and physical abuse and victimization experiences across developmental phases. Additionally, data are provided regarding the age of the victim, severity and chronicity of the abuse and whether or not the abuse occurred in an intrafamilial or extrafamilial context. Characteristics of the perpetrator(s) are also assessed as well as important information related to posttrauma experiences. An abbreviated version of this measure was utilized and a variety of important subfactors regarding the nature of subjects' experiences of sexual trauma were looked at separately. These subfactors include: the presence and severity of sexual trauma experienced before age 14, the presence and severity of sexual trauma experienced after age 14, level of violence experienced during a sexually traumatic event, support received following the event, the subject's perceived responsibility for the traumatic event, the subject's perception of traumatic severity, self-injurious and suicidal ideation and behavior following sexual traumata

experienced before age 14, self-injurious and suicidal ideation and behavior following sexual traumata experienced after age 14 and the age at which the subject was first sexually abused. 'The presence and severity of sexual trauma before and after age 14' variables were calculated on a continuous scale by assigning points incrementally for each affirmative report of having experienced graduated sexually traumatic events. For example, if a subject responded yes to the question, ''Since the age of 14, have you ever given in to sex play due to the arguments or pressure of another?'' then 1 point would be assigned. If that person also had experienced sex that was forced on them physically — the most severe form of sexual victimization inquired about — then a score of 9 points would be assigned. Then, these 2 scores would be added together for a total of 10 points. The 'age at the time of the first reported sexual victimization' was used in the statistical analyses without alteration. 'Physical violence' was assigned a score from 1 to 5 with greater severity of violence being assigned a higher score. The 'social support' variable scores ranged from 0 to 5, with higher scores denoting less support, which was conceptually expected to contribute to a more severe traumatic experience. 'Perceived responsibility', which was only inquired about for traumatic events occurring after age 14, was based on the sum of two 5 point (range 0 to 4) Likert scale items from the IGR which asked about perceived levels of responsibility of the subject and of their perpetrator. Therefore the final score range was from 0 to 8. The 'self-injury and/or suicidal ideations and/or behaviors following a sexually traumatic (before and after age 14)' variables were dummy coded 1 for yes and 0 for no. 'Self-perception of trauma severity' was coded on a 4-point Likert scale ranging from 0 for 'not at all traumatic' to 4 for 'severely traumatic'.

The TEQ is a version of Davidson and Smith's trauma questionnaire (TQ). The TEQ is used to assess an individual's life history for traumatic events. This questionnaire asks whether an individual has ever experienced one or more events which are outside the range of usual human experience and which would be markedly distressing to almost anyone. A list of potentially traumatic events is provided and the individual is asked to circle 'yes' or 'no' to determine whether or not the event has ever been experienced. If the individual responds 'yes', then their age at the time of the event is requested as is duration of the event. For those individuals who have experienced one or more of the traumatic events listed, they are asked to consider the most upsetting event as they answer additional yes/no questions directed at perceptions of the event such as: whether they felt prepared for the event, found it to be frightening or disgusting, were physically injured, thought that their life was in danger, were alone or accompanied, perceived that they were powerless to stop the event and whether they ever talked about the event to others. Additionally, PTSD symptoms experienced after traumatic events are listed and the individual is asked again to circle 'yes' or 'no' to determine which, if any, symptoms have been experienced. This measure has demonstrated good reliability and validity as reported by its authors. For the purposes of the present investigation, the traumatic events listed on the TEQ were categorized into one of the three following groups: (1) actual or threatened loss of home, family member or close other, (2) interpersonal violence and (3) accidents. The number of traumatic events falling into these 3 categories were summed for each subject, creating 3 of 4 TEQ variables. The fourth TEQ variable used in this investigation was the total number of PTSD symptoms reported by each subject.

2.5. Dissociation

Dissociation was assessed using the dissociative experiences scale (DES) (Bernstein and Putnam, 1986) and the questionnaire of experiences of dissociation (QED) (Riley, 1988). It is viewed as an excellent research and clinical measure exhibiting very good split-half reliability with coefficients ranging from 0.71 to 0.96. This measure also demonstrates very good stability with a test–retest reliability coefficient of 0.84. Additionally, the DES possesses fairly good construct validity; it does not correlate with some theoretically unrelated variables such as social class and sex while it does show a high degree of agreement among item scores in differentiating diagnostic groups (Bernstein and Putnam, 1986). Factor analytic studies conducted by Ross et al. (1990) and Ray and Faith (1995) have found that the DES is comprised of 3 major factors: (1) absorption–derealization, (2) amnesia and (3) depersonalization. In order to gain more specific understanding about the hypothesized relationship between dissociation and psychopathology, these 3 subfactors were utilized in the present investigation.

The QED is a comparable measure made up of twenty-six true/false items. The items of this measure were drawn from a review of the clinical literature detailing the experiences of 'classical' hysterics, persons with multiple personality disorder and other dissociative disorders and persons with temporal lobe epilepsy. The reliability of the measure (using Cronbach's alpha, the mean of all possible split-half intercorrelations) has a coefficient of 0.77. In a normal population, the QED has a mean of 9.92 and a standard deviation of 4.28. Within clinical populations of persons believed to be at risk for dissociative experiences, elevated scores occur. Briquet hysterics show a mean score of 13.9 and patients with dissociative identity disorder demonstrate an average score of 24.6. Because QED items and DES items do not overlap significantly, the QED, with its different response format, provides an alternate-form assessment technique (Riley, 1988). In the present investigation the QED total score was utilized.

2.6. Agoraphobic severity

Agoraphobic symptomatology severity was assessed using the global assessment of Severity (GAS; Michelson, 1987) and the self-rating of severity (SRS; Marks and Mathews, 1979). The GAS is a 5 point scale in which a score of 1 represents no clinical complaints and remission of symptomatology and a score of 5 indicates severe and incapacitating symptomatology due to agoraphobia. Final determination of all clinical ratings were conducted with the coinvestigators who were blind to treatment status and who were responsible for assigning final clinical ratings of subjects using a consensus strategy. In cases of uncertainty, the most conservative (i.e. severe rating) was assigned.

The SRS scale is a 9 point analogue measure rated by the subject in answer to the following question: "How would you rate the present state of your phobic symptoms on the scale below?" The scale ranged from no phobias present (0) to very severely disturbing, disabling agoraphobic symptoms (8). The SRS provides a client-rated measure of overall severity.

2.7. Anxiety

Clinical anxiety was assessed using an abbreviated version of the Taylor manifest anxiety scale (TMAS; Taylor, 1953).

2.8. Panic

The frequency of panic attacks was assessed using the panic attack form (Michelson, 1987). This form employs a self-rating scale for panic disorder for which subjects were requested to rate overall frequency and severity of their panic attacks and panic-related symptoms over the past 3 weeks. For the present investigation, only the panic frequency score from this measure was utilized.

2.9. Depression

Depression and dysphoria were assessed using the Beck depression inventory (BDI; Beck et al., 1961).

2.10. General symptomatology

General symptomatology was assessed using the Hopkins symptom checklist (HSCL; Derogatis et al., 1974) and the subjective symptom scale (SST; cf. Michelson and Mavissakalian, 1983). The SST consists of several subscales including agoraphobia, panic, depression, obsessions and depersonalization rated on a scale ranging from 0, indicating low symptomatology, to 8, indicating severe symptomatology. Good internal reliability and validity have been demonstrated by this scale.

2.11. Operationalized measure of endstate functioning

An operationalized composite measure was used to classify subjects' overall level of endstate functioning. This is a revised version of endstate functioning which has been found to more accurately reflect a high level of clinical functioning, since the panic dimension of PDA has now been incorporated into the composite. Further, to ensure that only nondiagnostic individuals (i.e. GAS < 2) were eligible for high endstate classification, any subjects who met high endstate criteria but had a GAS > 3 were reassigned to the medium endstate category. Five a priori criteria were used to classify subjects' level of endstate (low–medium–high) functioning. Subjects were assigned 1 point for each of the following scores: (a) panic frequency = 0 on the PAF-frequency per 3 weeks; (b) global assessment of severity = 1; (c) ≤ 2 on the self-rating of severity; (d) ≤ 3 on the phobic anxiety and avoidance scale; (e) 20 steps on the behavioral avoidance course with ≤ 3 for the SUDS (in vivo anxiety) measure, indicating completion of the entire 1 mile course with minimal or no anxiety. This operationalized definition of revised endstate functioning has the merit of combining severity (as judged by both clinician and subject) and clinical ratings of phobic anxiety and avoidance. It also encompasses a rating of panic frequency and a direct behavioral measure of agoraphobia.

Subjects with 0–1 points were classified as low endstate functioning (LEF); 2–3 points were assigned to medium endstate functioning (MEF); and those with 4–5 points were classified as high endstate functioning (HEF). Further, to ensure that the endstate classification provided the most accurate reflection of subjects' outcome, a last known endstate replacement procedure was used in those cases in which relevant data were not available at a given assessment period. This is consistent with the use of endpoint analyses in the fields of psychotherapy and pharmacotherapy research and is believed to provide a more conservative and veridical assessment of outcome status. A pretreatment functioning equivalent of the endstate functioning score was computed for subjects in the present investigation using the above definition.

3. Results

To ensure data reliability, 100% of all data were entered and double-checked for accuracy. Statistical analyses were performed on an IBM compatible system using the statistical package for the social sciences for windows (SPSS for Windows version 6.1.3; SPSS, 1995). Prior to testing the primary hypotheses, a 3×3 MANOVA was conducted to examine potentially confounding main and interaction effects between trauma history and type of treatment. Trauma history, as defined by the number of self-reported traumatic experiences on the TEQ, was divided into three levels — 'none' (0 traumatic experiences), 'moderate' (1–2 traumatic experiences) and 'high' (3 or more traumatic experiences). Cutoff points for the number of self-reported traumatic experiences were set by the investigators on both a conceptual basis and with attention to number of subjects in each level. The 'moderate' level of trauma history could have been defined as 1–2 or 1–3 self-reported traumatic experiences with essentially similar cell sizes, but the definition of 1–2 traumatic experiences was utilized in an effort to be more conservative.

The original six treatment groups were collapsed into three categories to enhance statistical power for the MANOVA. First, a cognitive treatment category was created by combining the PI and CT + GE treatment groups. Second, the RT and R + GE treatment groups were combined to create a deep muscle relaxation treatment category. Third, a behavioral-exposure treatment category was created by merging the two GE treatment groups from both studies. Main and interaction effects were examined using the following major outcome measures at posttreatment and 1 year follow-up: BDI, HSCL, LKE, SST and TMAS. Once assured of no main or interaction effects, this permitted aggregation of all treatment cells for the subsequent analyses and no further treatment-specific hypotheses were generated.

To empirically examine the three major hypotheses of the present study, a series of multiple, linear stepwise regressions was performed using mean substitution to replace missing values. For each series of regressions (pretreatment, posttreatment and 1 year follow-up), three predictor domains were utilized. The first predictor domain focused on sexual trauma and consisted of nine subcategories of traumatic experiencing as measured by the IGR. These subcategories were as follows: (1) history of sexual trauma before age 14, (2) history of sexual trauma after age 14, (3) violence experienced during the traumatic event, (4) support received following the traumatic event, (5) perceived responsibility for the event, (6) perceived severity

of the traumatic event, (7) self-injurious and suicidal ideations or behaviors following a traumatic event experienced before age 14, (8) self-injurious and suicidal ideations or behaviors following a traumatic event experienced after age 14 and (9) age at the time of the first traumatic event. Originally, a tenth subcategory for the subjects' immediate emotional reaction to the traumatic event was included. However, because this subcategory was correlated highly ($r = 0.86$) with the perceived severity of trauma subcategory, it was dropped from all further analyses.

The second predictor domain focused on more broad-based traumas, covering the full range of traumata. This domain consisted of four subcategories from the TEQ: (1) actual or threatened loss of home, family member or important other, (2) interpersonal violence (including sexual abuse, incest and rape), (3) accidents and (4) number of PTSD symptoms experienced following the traumatic event. The last predictor domain consisted of four measures of dissociation: (1) DES depersonalization subfactor, (2) DES amnesia subfactor, (3) DES absorption subfactor and (4) QED total score. In no case was the DES total score used in conjunction with the DES subfactors to preclude redundancy of the data analyses.

3.1. Hypothesis 1: pretreatment

It was hypothesized that a history of traumatic experiences and associated elevations in dissociation levels would be predictive of greater psychopathology at pretreatment. The descriptive statistics for the major trauma, dissociation and psychopathology variables used in the present investigation are summarized in Table 2. It is important to note that the means for the IGR trauma variables may seem unexpectedly high. Many of the subjects in this study who

Table 2
Descriptive statistics on major trauma, dissociation and psychopathology variables

	Mean	S.D.	Range
Predictor variables			
IGR-sexual trauma before age 14	10.70	25.62	164.00
IGR-sexual trauma after age 14	26.80	64.37	360.00
IGR-violence	1.28	1.46	5.00
IGR-support	5.68	2.94	10.00
IGR-perceived responsibility	2.72	1.69	6.00
IGR-perceived severity	2.02	1.82	6.00
IGR-self-injury/suicide before 14	0.11	0.32	1.00
IGR-self-Injury/suicide after 14	0.15	0.36	1.00
IGR-age at time of earliest trauma	13.40	7.93	41.00
TEQ-loss of home, family, friends	1.54	1.42	8.00
TEQ-interpersonal violence	0.55	1.15	6.00
TEQ-accidents	0.82	0.92	5.00
TEQ-total number of traumata	3.01	2.48	16.00
TEQ-number of PTSD symptoms	6.62	5.91	19.00
DES absorption subfactor	13.54	11.11	57.78
DES amnesia subfactor	3.38	5.12	23.75

DES depersonalization subfactor	5.00	7.80	46.67
DES total score	8.90	7.19	33.20
QED total score (0–26)	8.27	4.89	21.00
Outcome variables			
BDI-pre	16.91	9.78	38.00
BDI-post	6.17	6.62	27.00
BDI-1YrFu	7.23	8.97	45.00
GAS-pre	4.54	0.59	2.00
GAS-post	2.30	0.95	4.00
GAS-1YrFu	2.21	1.26	4.00
HSCL-pre	119.75	27.60	116.00
HSCL-post	86.51	21.42	103.00
HSCL-1YrFu	86.46	26.47	131.00
Pretreatment functioning (1–3)[a]	1.07	0.25	1.00
Last known endstate-post (1–3)[a]	1.97	0.70	2.00
Last known endstate-1YrFu (1–3)[a]	2.20	0.73	2.00
PFREQ-pre	5.74	7.82	42.00
PFREQ-post	2.46	2.93	18.00
PFREQ-1YrFu	2.18	3.76	25.00
SRS-pre	5.76	1.76	8.00
SRS-post	2.32	1.08	7.00
SRS-1YrFu	2.33	1.70	8.00
SST-pre	25.57	13.21	54.00
SST-post	10.30	10.08	44.00
SST-1YrFu	10.37	13.24	56.00
TMAS-pre	30.16	10.23	44.00
TMAS-post	19.37	10.55	46.00
TMAS-1YrFu	19.16	12.40	49.00

[a] Categorical variable with 1 for low functioning, 2 for moderate functioning and 3 for high functioning.

experienced sexual traumas as reported on the IGR were repeatedly traumatized over a period of months or years. Therefore, while approximately half of the sample did not report any sexual trauma on the IGR, the mean was still inflated due to the repetitive nature of the sexual abuse experienced by others in the sample.

The traumatic experiences endorsed by the subjects in this investigation on the TEQ is presented in Table 3. These traumatic experiences were categorized into three groupings as discussed previously: (1) actual or threatened loss of home, family members or close others, (2) interpersonal violence and (3) accidents. Additionally, the frequency and percentages of individuals who reported specific traumas in another study, Escalona et al. (1997), were provided for a comparison. In Escalona et al. (1997), 343 psychiatric inpatients admitted to an

Table 3
Trauma endorsement on the TEQ

Type of trauma	Frequencies and Percentages	
	present study	Escalona et al. (1997)
Actual/threatened loss of home, family, close other		
Loss of home by fire	6	26 (8%)
Loss of home or family member by flood	0 (0.0%)	5 (1%)
Loss of home or family member by tornado/hurricane	1 (1.1%)	8 (2%)
Loss of home or family member by earthquake	0 (0.0%)	0 (0%)
Serious threat or harm to family member or close friend	23 (25.8%)	66 (19%)
Unexpected death of family member or close friend	60 (67.4%)	202 (59%)
Seeing another person seriously injured or dying due to accident/violence	24 (27.0%)	90 (26%)
Interpersonal violence		
Physical abuse	13 (14.6%)	73 (21%)
Assault	9 (10.1%)	32 (9%)
Rape	9 (10.1%)	33 (10%)
Incest	8 (9.0%)	20 (6%)
Being kidnapped	3 (3.4%)	3 (1%)
Being captive	3 (3.4%)	14 (4%)
Combat	2 (2.2%)	17 (5%)
Accidents		
Car, train, or airplane accident	46 (51.7%)	148 (43%)
Industrial or work-related accident	8 (9.0%)	49 (14%)
Near drowning	11 (12.4%)	64 (19%)
Being seriously burned	1 (1.1%)	26 (8%)
Other	10 (11.2%)	56 (16%)

affective-disorders unit were administered the TEQ. Frequencies are similar for the two samples.

As shown in Table 4, two of the IGR subcategories — presence and severity sexual trauma experienced after age 14 and the level of violence during the event — were retained as significant predictors of higher psychopathology at pretreatment. The severity of sexual trauma experienced after age 14 was associated with higher scores on the HSCL and SST and level of violence was associated with greater psychopathology as measured by the SRS. The number of PTSD symptoms reported on the TEQ significantly predicted higher scores on the BDI, HSCL, SST and TMAS. The DES depersonalization subfactor exhibited a significant positive association with the GAS. Lastly, the QED total score significantly predicted higher scores on the BDI, HSCL, SST and TMAS, accounting for 12–22% of the variance on these measures at pretreatment. In all cases discussed above, higher levels of trauma and dissociation were associated with greater pretreatment psychopathology. All relationships were in the expected direction (as was the case for hypotheses 2 and 3).

Table 4
Significant stepwise regressions of pretreatment psychopathology

Criterion	Predictor measures	Significant predictors retained	Adjusted		
			R^2	*df*	*F*-ratio
BDI	IGR	–	–	–	–
	TEQ	[c]	0.06	1,87	6.52**
	DES/QED	[e]	0.12	1,87	13.39***
GAS	IGR	–	–	–	–
	TEQ	–	–	–	–
	DES/QED	[d]	0.06	1,87	5.20*
HSCL	IGR	[a]	0.05	1,87	5.21*
	TEQ	[c]	0.09	1,87	9.22***
	DES/QED	[e]	0.19	1,87	21.43***
LKE	IGR	–	–	–	–
	TEQ	[c]	0.04	1,87	4.71*
	DES/QED	–	–	–	–
PFRQ	IGR	–	–	–	–
	TEQ	–	–	–	–
	DES/QED	–	–	–	–
SRS	IGR	[b]	0.05	1,87	5.22*
	TEQ	–	–	–	–
	DES/QED	–	–	–	–
SST	IGR	[a]	0.04	1,87	4.32*
	TEQ	[c]	0.09	1,87	10.14***
	DES/QED	[e]	0.22	1,87	25.17***
TMAS	IGR	–	–	–	–
	TEQ	[c]	0.06	1,87	7.09**
	DES/QED	[e]	0.13	1,87	13.91***

*$p < 0.05$; **$p < 0.01$; ***$p < 0.005$.
[a] Presence and severity of sexual trauma after age 14 (from the IGR).
[b] Level of violence during the event (from the IGR).
[c] Number of PTSD symptoms reported on the TEQ.
[d] Depersonalization subfactor of the DES.
[e] QED total score.

3.2. Hypothesis 2: posttreatment

The second hypothesis proposed that the presence of past traumatic experience and associated levels of dissociation would be predictive of greater psychopathology at posttreatment, denoting poorer treatment outcome. In order to examine this hypothesis, the scores for the outcome variables were individually adjusted to covary out pretreatment levels of psychopathology. The adjustment for each subject's score, $(Y-Y')$, was obtained by subtracting from the deviation of that score from the grand mean a value that was based on the deviation of the corresponding covariate from the grand mean on the covariate, weighted by the

regression coefficient for predicting the dependent variable from the covariate (Tabachnik and Fidell, 1996)

$$(Y - Y') = (Y - \mathrm{GM}_y) - B_{y \cdot x}(X - \mathrm{GM}_x).$$

As shown in Table 5, the level of violence during a past sexual trauma along with the subcategory of age at the time of one's earliest traumatic event were found collectively to predict BDI scores. That is, greater violence and younger ages at the time of initial traumatization were associated with higher scores on the BDI. Reports on the TEQ provided consistent findings with regard to interpersonal violence. Subjects who reported experiences of interpersonal violence on the TEQ also reported significantly higher BDI scores at

Table 5
Significant stepwise regressions of treatment outcome measures at posttreatment

Criterion	Predictor measures	Significant predictors retained	Adjusted		
			R^2	*df*	*F*-ratio
BDI	IGR	[a,b]	0.09	2,86	5.30**
	TEQ	[c]	0.04	1,87	4.46*
	DES/QED	–	–	–	–
GAS	IGR	–	–	–	–
	TEQ	–	–	–	–
	DES/QED	–	–	–	–
HSCL	IGR	–	–	–	–
	TEQ	[d]	0.07	1,87	8.04**
	DES/QED	–	–	–	–
LKE	IGR	–	–	–	–
	TEQ	–	–	–	–
	DES/QED	–	–	–	–
PFRQ	IGR	–	–	–	–
	TEQ	–	–	–	–
	DES/QED	–	–	–	–
SRS	IGR	–	–	–	–
	TEQ	–	–	–	–
	DES/QED	[e]	0.07	1,87	8.10***
SST	IGR	–	–	–	–
	TEQ	–	–	–	–
	DES/QED	[f]	0.04	1,87	4.23*
TMAS	IGR	–	–	–	–
	TEQ	[d]	0.05	1,87	5.17*
	DES/QED	–	–	–	–

Note. *$p < .05$; **$p < .01$; ***$p < .005$.
[a] Level of violence (from IGR).
[b] Age at time of trauma (from IGR).
[c] [d]
[e] Amnesia subfactor of DES.
[f] QED total score.

posttreatment. Additionally, subjects who reported experiences of actual or threatened loss of home, family members or close others on the TEQ scored significantly higher on the HSCL and the TMAS. Dissociation, as measured by the QED total score, was found to be associated significantly with the scores on the SST and SRS scores were significantly predicted by the DES amnesia subfactor. The amount of variance accounted for in the significant regressions discussed above ranged from 4 to 9%. For all the relationships discussed here, higher levels of trauma and dissociation predicted higher levels of psychopathology and poorer treatment outcome.

3.3. Hypothesis 3: 1 year follow-up

The final hypothesis proposed in the present investigation stated that a history of traumatic experiencing and associated levels of dissociation would be associated with greater psychopathology at 1 year follow-up, signifying higher relapse rates and poorer longitudinal maintenance of treatment gains. As was the case for posttreatment analyses, scores for the outcome variables were individually adjusted to covary out pretreatment levels of psychopathology in order to adequately assess this hypothesis.

As displayed in Table 6, 'presence and severity of sexual trauma after age 14' and 'perceived responsibility for the sexual trauma', combined in a single regression model, were significantly associated with elevated scores on the BDI, HSCL and SRS. The amount of variance accounted for when these two predictors were combined into one regression model ranged from 9% to 12%. 'Presence and severity of sexual trauma after age 14' alone predicted PFRQ scores at 1 year follow-up.

As was the case at pretreatment, 'presence and severity of sexual trauma experienced after age 14' significantly predicted several of the psychopathology variables, whereas the 'presence and severity of sexual trauma experienced before age 14' did not. This was not expected and counters much of the traumatology research which has suggested that sexual trauma experienced at earlier ages is associated with increased symptomatology. In this investigation, 'presence and severity of sexual trauma experienced before age 14' was not found to be a strong predictor of psychopathology at pretreatment, posttreatment or 1 year follow-up.

The number of PTSD symptoms reported on the TEQ significantly predicted scores on the GAS, HSCL, SRS and TMAS at the 1 year follow-up, accounting for 4–8% of the variance for these outcome variables. Significant associations between the DES depersonalization subfactor and GAS and TMAS scores were obtained, accounting for 15 and 5% of the variance, respectively. The QED total score was found to significantly predict scores on the SRS. Once again, higher levels of traumatic experiences and dissociation were shown to relate significantly with higher scores of psychopathology at 1 year follow-up, denoting greater relapse rates and poorer longitudinal maintenance of treatment gains.

Three 2×2 MANOVAS were conducted, one for each assessment phase, as an alternative method of analyzing the effects of trauma and dissociation on psychopathology levels and treatment outcome and maintenance. The two factors used in the analysis were the QED total score and the total number of traumatic experiences reported across all three TEQ subcategories. Median splits were utilized in order to create high and low categories for both variables. The same set of dependent variables which were analyzed by multiple regression was used for these analyses.

Table 6
Significant stepwise regressions of treatment outcome measures at 1 year follow-up

Criterion	Predictor measures	Significant predictors retained	Adjusted		
			R^2	*df*	*F*-ratio
BDI	IGR	[a,b]	0.10	2,86	6.10***
	TEQ	–	–	–	–
	DES/QED	–	–	–	–
GAS	IGR	–	–	–	–
	TEQ	[c]	0.05	1,87	5.97*
	DES/QED	[d]	0.15	1,87	16.30***
HSCL	IGR	[a,b]	0.09	2,86	5.13**
	TEQ	[c]	0.06	1,87	6.43**
	DES/QED	–	–	–	–
LKE	IGR	–	–	–	–
	TEQ	–	–	–	–
	DES/QED	–	–	–	–
PFRQ	IGR	[a]	0.05	1,87	5.62*
	TEQ	–	–	–	–
	DES/QED	–	–	–	–
SRS	IGR	[a,b]	0.12	2,86	6.77***
	TEQ	[c]	0.04	1,87	4.91*
	DES/QED	[e]	0.05	1,87	5.59*
SST	IGR	–	–	–	–
	TEQ	–	–	–	–
	DES/QED	–	–	–	–
TMAS	IGR	–	–	–	–
	TEQ	[c]	0.08	1,87	8.89***
	DES/QED	[d]	0.05	1,87	5.51*

Note. *$p < 0.05$; **$p < 0.01$; ***$p < 0.005$.
[a] Presence and severity of sexual trauma after age 14 (from IGR).
[b] Perceived responsibility (from IGR).
[c] Number of PTSD symptoms from the TEQ.
[d] Depersonalization subcategory of DES.
[e] QED total score.

At pretreatment, no significant multivariate interaction effects were found, but one significant univariate interaction effect emerged for the HSCL ($p = 0.05$). No multivariate main effects for the TEQ were found either, but again one univariate main effect surfaced for the SRS ($p = 0.05$). For the QED, significant multivariate and univariate main effects were obtained. For the entire set of treatment outcome variables the significance level of the QED main effect was $p < 0.001$. The univariate main effects were as follows: BDI ($p < 0.005$), HSCL ($p < 0.001$), SST ($p < 0.001$) and TMAS ($p < 0.005$).

At posttreatment, no significant multivariate or univariate interaction effects were obtained. Three univariate main effects for the TEQ were found: BDI ($p < 0.05$), HSCL ($p < 0.05$) and TMAS ($p < 0.05$). However, no TEQ main effect resulted when the entire set of dependent

variables was included, which was also the case for the QED, but one univariate main effect for the QED emerged — BDI ($p < 0.05$). A multivariate interaction effect was not found at one-year follow-up, but one univariate interaction effect, GAS ($p < 0.01$), was obtained. No multivariate or univariate main effects emerged for the TEQ or the QED.

4. Discussion

4.1. Hypothesis 1: pretreatment

A history of traumatic experiences and associated elevations in dissociation levels were hypothesized to be predictive of greater psychopathology at pretreatment. In this study, the presence and severity of sexual trauma after age 14 was associated with higher levels of general symptomatology at pretreatment as measured by the HSCL and SST. Additionally, higher levels of violence during sexually traumatic events were correlated with more severe agoraphobic symptoms (SRS). For both researchers and clinicians interested in the treatment of PDA, this finding suggests that careful gathering of information regarding prior sexual abuse and the extent of violence involved would be very important, especially for more severely agoraphobic subjects/clients. Contrary to other studies that have found positive correlations between earlier sexual trauma and later panic and agoraphobic symptomatology (Stein et al., 1989; Saunders et al., 1992; Walker et al., 1992; Pribor and Dinwiddie, 1992; Murrey et al., 1993), earlier sexual trauma was not associated with greater PDA pathology in this sample. Most of these studies (Saunders et al., 1992; Walker et al., 1992; Murrey et al., 1993) specifically identified childhood sexual abuse as an important predictor. So, it is surprising that neither of the variables, 'presence and severity of sexual trauma experienced before age 14' and 'earliest age of sexual abuse', proved to be significantly associated with greater pretreatment psychopathology in the present investigation. These counterintuitive results may be indicative of generally lower levels of severity of sexual abuse experienced before age 14 relative to sexual abuse experienced after age 14 (e.g. sexual intercourse vs. fondling). Other trauma-related variables, such as 'support received' following the sexually traumatic event, 'perceived responsibility' for the event, 'perceived severity' and 'self-injurious and suicidal ideations and behaviors', which have often been cited as important predictors of PTSD and general symptomatology were not found to be predictive of greater levels of psychopathology in this PDA treatment sample.

When looking at more broad-based trauma (not limited to sexual trauma), subjects who reported experiencing higher numbers of PTSD symptoms following a traumatic event also reported higher levels of panic (LKE), agoraphobia (LKE), anxiety (TMAS), depression (BDI) and other general symptomatology (HSCL). None of the three categories of trauma from the TEQ, (1) actual or threatened loss of home, family member or important other, (2) interpersonal violence and (3) accidents, were found to be correlated with greater psychopathology at pretreatment. This result diverges from the literature that has pointed to the loss of significant others and physical threat or illness as precipitants of panic disorder and agoraphobia (Foa et al., 1984; Faravelli, 1985; Faravelli et al., 1985; Tweed et al., 1989; Kendler et al., 1992).

Higher levels of dissociation as measured by the QED total score, were related to increased anxiety (TMAS), depression (BDI) and general symptomatology (HSCL and SST) at pretreatment as predicted. Of the DES subfactors of amnesia, absorption and depersonalization, only the depersonalization subfactor was found to be a significant correlate of any pretreatment psychopathology. In this sample, greater depersonalization was associated with higher levels of agoraphobia as measured on the GAS. Possibly, the overlap in symptoms between dissociation and PDA was reflected in this relationship. Many persons suffering from PDA have reported experiencing depersonalization during panic attacks and periods of heightened anxiety. Depersonalization was included among the symptoms of panic attacks in the DSM-III-R and the DSM-IV (APA, 1980; APA, 1994).

The 2×2 MANOVA looking at high/low distinctions on the QED and total number of traumatic experiences reported on the TEQ suggested that an interaction between high trauma and high dissociation predicted greater general psychopathology on the HSCL. Also, higher numbers of traumatic experiences were found to be associated with greater agoraphobia as measured by the SRS. However, the most strongly significant findings resulted when looking at the main effects of dissociation on pretreatment psychopathology. Higher dissociation levels were predictive of greater depression (BDI), anxiety (TMAS) and general psychopathology (HSCL and SST). These results were very consistent with those found using multiple regression modeling.

The ability to detect significant associations between traumatic experiences and associated elevations in dissociation with greater pretreatment psychopathology was attenuated in this study by floor effects imposed by the selection criteria of the two original studies. To be included in either study, subjects had to exhibit moderate to severe levels of panic and agoraphobia symptomatology. Despite this limitation, both traumatic experiencing and dissociation were significantly related to increased levels of pretreatment general psychopathology and symptomatology more specifically related to PDA. General symptomatology at pretreatment was consistently associated with each category of interest — sexual trauma, broad-based trauma and dissociation. Agoraphobic symptoms were predicted by violence during sexually traumatic events, number of PTSD symptoms endorsed and depersonalization.

These results suggest that researchers and clinicians should pay close attention to issues of comorbidity and differential diagnosis. The findings also suggest that clients who report more severe PDA symptoms may also be experiencing sequelae from prior trauma. This is important information to discern as the treatment focus may need to be broadened in the clinician's office.

4.2. Hypothesis 2: posttreatment

The second hypothesis of this study proposed that the presence of past traumatic experiences and associated elevations in dissociation would be predictive of greater psychopathology at posttreatment, denoting poorer treatment outcome. This hypothesis was supported by the results. At posttreatment, having experienced a sexually traumatic event after age 14 ceased to be a significant predictor of psychopathology. Instead, 'age at the time of the first sexual trauma' and 'level of violence' from the IGR emerged as correlates of depression (BDI). That

is, the younger subjects were when first sexually traumatized and the more violence that is experienced during a sexually traumatic event, the greater the reports of depression at posttreatment. This finding was consistent with other studies that have found heightened risk for psychopathology when the traumatic event is accompanied by violence (Frederick, 1987). These results are curious given that 'presence and severity of sexual trauma before age 14' never correlated significantly with any of the measures of psychopathology at pretreatment or posttreatment. As discussed in the previous section, sexual trauma experienced at younger ages has been found to be at least as traumatic and predictive of later psychopathology, or even more so, than sexual trauma experienced later in life. Again, this result may be reflective of the fact that sexual abuse experienced after age 14 tended to be more severe than sexual abuse experienced before age 14.

When more broad-based trauma was evaluated using the TEQ, higher numbers of PTSD symptoms reported by subjects were not found to be correlated with any of the outcome variables. This presented a notable difference from the many relationships found at pretreatment between number of PTSD symptoms and depression (BDI), anxiety (TMAS), agoraphobic avoidance (LKE) and general symptomatology (HSCL and SST). At least two possible theoretical explanations for this result exist. First, the fear structures of subjects who had experienced a traumatic event might be accessed during exposure treatments for PDA to some degree, but not fully. The principles of exposure-based treatments for PDA and PTSD (and other anxiety disorders for that matter) are essentially quite similar. The subjects in this study may very well have generalized the PDA-focused exposure techniques to more PTSD-related avoidance. However, as will be discussed in the next section, their PTSD fear structures may not have been adequately accessed and targeted to achieve long-term treatment gains. Second, immediately following treatment for PDA, these subjects were likely to be very focused on fears and avoidance related to PDA. PTSD-focused anxiety and avoidance may have taken a temporary 'back-seat'. That is, the focus of their anxiety and avoidance may have been redirected from their traumatic experience to PDA concerns during and immediately following treatment. Just as likely, they may have attributed many of their anxiety-related symptoms to PDA rather than traumatic stress.

Although the regression analyses did not find significant associations between any of the three TEQ subcategories of trauma, the 2×2 MANOVA looking at high/low median splits of the total number of traumatic experiences reported on the TEQ suggested that greater traumatic experiencing was predictive of higher depression (BDI), anxiety (TMAS) and general psychopathology (HSCL) at posttreatment. It is possible that dividing the total number of traumatic experiences into the three conceptual categories of trauma that were used in the regression analyses diminished statistical power to detect relationships that exist between number of traumatic experiences and increased posttreatment psychopathology. These results lend further support to the proposed hypothesis that increased traumatic experiencing is predictive of poorer treatment outcome.

Greater dissociation, as measured by the QED total score, was related to general symptomatology (SST) at posttreatment, using multiple regression analyses and depression (BDI), when analyzed via MANOVA. This finding is consistent with literature on dissociation's high degree of correlation with general psychopathology (Norton et al., 1990; Sandberg and Lynn, 1992; Nash et al., 1993; Tillman et al., 1994). The DES subfactor of amnesia was

positively associated with agoraphobia as measured by the SRS. One logical explanation for a link between amnesia and poorer treatment outcome has to do with a subject's ability to attend to and remember what happened during the treatment session. For example, if a subject was unable to remember explanations regarding the importance of gradual exposure in treating PDA, instructions for homework assignments, cognitive restructuring techniques, instructions for relaxation techniques, etc., then that subject would be much less likely to achieve significant treatment gains. PDA subjects who are also experiencing dissociation are very possibly 'checking out' during treatment sessions or simply not remembering what occurred during session, so that they are unable to take full advantage of the treatment. This explanation would seem even more likely for dissociative subjects who are experiencing the treatment as being stressful, which in turn might lead to increased dissociation (Brown, 1994). Certainly, it is likely that subjects in a treatment program utilizing exposure techniques are going to feel stress at some point, but those who are also experiencing dissociative symptoms may be less able to tolerate the stress and less able to make effective use of the treatment.

The explanation proposed above, if accurate, would have important implications for both clinicians and researchers. It could then be argued that the administration of the QED or DES may provide useful data about clients entering treatment for PDA or any other treatment that will expose them to significant levels of stress. For those clients who report greater dissociative symptoms, techniques may need to be added to the PDA treatment protocol to assure that they are attending to the treatment. For example, a dissociative client might be instructed to periodically restate or write down what has just been covered by the therapist or perhaps some sort of grounding exercise could be utilized.

More relevant for PDA researchers, these findings suggest that a significant proportion of those subjects who are not achieving successful outcomes in treatment may have high levels of dissociative symptoms in addition to PDA. Therefore, part of the variance for clients who have not improved significantly in PDA treatments potentially might be explained by the presence of comorbid dissociative symptoms and the inability to fully attend to treatment.

4.3. Hypothesis 3: 1 year follow-up

At the 1 year follow-up, a history of traumatic experiencing together with associated elevations in dissociation levels was predicted to be related to greater psychopathology, signifying higher relapse rates and poorer longitudinal maintenance of treatment gains. This hypothesis was also supported by the results of the present investigation. At follow-up, the regression model combining 'presence and severity of a sexually traumatic event after age 14' and 'perceived responsibility for the traumatic event' re-emerged as an important positive correlate of depression (BDI), agoraphobia (SRS) and general symptomatology (HSCL). Additionally, 'presence and severity of a sexually traumatic event after age 14' by itself predicted panic frequency (PFRQ); more evidence suggesting that long-term maintenance of PDA treatment gains is poorer for subjects who experienced sexual trauma after age 14, especially if they feel responsible for the traumatic event. One possible explanation for these relationships is that subjects who experienced sexual trauma after age 14 also felt more responsible for the event, perhaps believing that they were old enough that they should have been able to prevent it somehow. These feelings of responsibility may have led to associated

feelings of shame or guilt. As a result, the stress of the sexual trauma itself coupled with the stresses of feeling responsible, shameful and guilty, could have been collectively internalized. Consistent with a stress-diathesis model, the internalized stress may have then contributed to a heightened predisposition to psychopathology in general and, more specifically for this sample, PDA. Consequently, this heightened predisposition may have placed these individuals at increased risk for relapse.

When considering more broad-based traumata, the number of reported PTSD symptoms alone was associated with greater psychopathology at the 1 year follow-up, as suggested by the regression analyses. For the most part, number of traumatic experiences was not predictive of greater psychopathology in either the regression analysis or the 2×2 MANOVA conducted at 1 year follow-up. The only significant finding resulting from either analysis was one significant univariate interaction effect suggesting that higher levels of trauma in combination with higher levels of dissociation were predictive of greater agoraphobia (GAS). Subjects who reported more PTSD symptoms following a prior traumatic experience were significantly more likely to report anxiety (TMAS), agoraphobia (SRS and GAS) and general symptomatology (HSCL). Therefore, those subjects who had greater numbers of PTSD symptoms following prior traumatic experiencing also exhibited poorer maintenance of treatment gains and higher relapse rates. Interestingly, the relationships between number of PTSD symptoms reported and these various psychopathology constructs re-emerged one year following treatment when all of these relationships had disappeared at posttreatment. An explanation for this outcome would be that many subjects experienced comorbid symptoms of PDA and PTSD. Given that all treatments in studies I and II were found to be significantly efficacious with treating PDA, perhaps the subjects' PTSD symptoms were no longer masked by or confused with their PDA symptoms immediately following treatment. Perhaps the PTSD fear structures had only been partially accessed during exposure treatments, enough to experience partial and temporary remission of PTSD symptoms, but not adequately accessed for long-term maintenance of treatment gains. A second related explanation is that the symptoms related to trauma were not specifically addressed in treatment and a resurgence of these PTSD symptoms, many of which overlap with PDA symptoms, may have been experienced, increasing the likelihood of relapse. A third explanation, which could be consistent with either of those posited above, is suggested by the findings of Hyer et al. (1996), discussed previously. For persons who have suffered PTSD symptoms chronically, increases in the use of emotion-focused and escape-avoidance strategies were detected. Long-term reliance on such strategies could tend to make effective use of graduated exposure techniques less likely. This explanation is supported by the findings of Scott and Stradling (1997) in which only 57% of subjects being treated for PTSD with an exposure-based technique actually complied with the treatment. Compliance was related to initial symptom severity and to comorbid depression. Therefore, for some individuals with histories of trauma, exposure techniques were not the treatment of choice.

At 1 year follow-up, higher levels of dissociation as measured by the total QED score were positively associated with greater agoraphobic symptomatology (SRS per the regression analyses and GAS per the MANOVA in an interaction with total number of traumatic experiences reported on the TEQ) and the DES subfactor of depersonalization was found to be positively correlated with both anxiety (TMAS) and agoraphobia (GAS). As was discussed previously, symptoms of dissociation, specifically depersonalization, overlap with symptoms

experienced by many sufferers of PDA. Individuals with PDA are particularly sensitive to internal sensations, both bodily and mental, which are perceived as being abnormal (Beck and Emery, 1985; Clark, 1986). Dissociative symptoms and depersonalization would fall into this category for many persons with PDA. Therefore, these individuals might then become hypersensitive to experiences of dissociation and depersonalization and catastrophize about their meaning, leading to hyperarousal and eventually panic. This mechanism may be responsible for the relationship of dissociation and depersonalization with agoraphobia found at pretreatment and 1 year follow-up.

Once again, these findings would suggest the utility of knowing prior to treatment whether or not an individual presenting for PDA treatment has a history of trauma and symptoms of PTSD following the trauma. Additionally, the administration of the QED or DES would provide valuable information about the presence and level of dissociative symptoms. If trauma and/or dissociation are found at clinically significant levels, the treatment would need to target these areas in addition to PDA symptoms to increase outcome and maintenance effects. In future treatment outcome studies of PDA, clients' histories of trauma, PTSD symptomatology and levels of dissociation should be assessed to enable more fine-grained delineations of their relation to short- and long-term efficacy.

4.4. Methodological issues

4.4.1. Retrospective self-reporting

In this investigation, the dissociation measures were completed 3–5 years following completion of treatment. The scores on these measures were then used in the analyses with pretreatment, posttreatment and 1 year follow-up psychopathology levels. It is possible that dissociation levels at the time of measurement were not accurate estimates of dissociation at pretreatment, posttreatment and follow-up. However, the consistent relationships between dissociation and psychopathology that were exhibited at each of these assessment phases might suggest that dissociation levels were, in fact, fairly stable across time for this sample. Conceptually, this limitation would not seem to threaten the validity of measurement of trauma history in the same way. The subjects were asked for their retrospective account of any traumatic experiences and their reactions to that experience. Whereas dissociation scores reflected actual dissociation levels 3–5 years posttreatment, trauma measurement taken 3–5 years posttreatment reflected the subjects' retrospective accounts of traumatic experiencing. For all subjects who reported a trauma history except one, the first traumatic experience occurred eight or more years prior to entering treatment for PDA.

Another potentially problematic issue exists related to the collection of data on dissociation 3–5 years following treatment. The symptoms of PDA are quite aversive and typically cause the individual suffering from PDA a great deal of distress. Dissociation, as discussed in the introduction, is understood by many in the field to be a strategy for avoiding strong affect. Therefore, it is possible that PDA symptomatology 3–5 years following treatment may have led to an increase in dissociative symptoms for some subjects who were not able to tolerate the uncomfortable affect resulting from their PDA symptoms. If this were the case, then higher levels of dissociation measured 3–5 years posttreatment should not be viewed as a potential factor contributing to poorer treatment response and/or maintenance.

The limitations of retrospective self-reporting are well-documented as both an internal and external threat to validity. In general, it is plausible to assume that some subjects may have forgotten the details surrounding a traumatic event or the extent of their reactions to a trauma, or even that a traumatic event occurred at all. If one excepts the notion that trauma and dissociation are often linked to one another, then it is reasonable to assume that some subjects who may have experienced a traumatic history did not remember the event(s) due to ongoing dissociation of the event(s). Additionally, some subjects may have consciously failed to report trauma or dissociation because of associated shame, embarrassment or issues of social desirability (Widom and Shepard, 1996).

Research has been conducted on the specific nature of retrospective self-report among subjects who report trauma and/or dissociation. Varying results have been reported. Some researchers contend that retrospective self-reports of trauma and dissociation are overestimated (Conte, 1986; Loftus, 1993; Tillman et al., 1994). While others contend that traumatic experiences, specifically childhood physical and sexual abuse, tend to be underestimated. Widom and Shepard (1996) and Widom and Morris (1997) make very convincing arguments to support this latter position. In these studies, children who were either physically or sexually abused were included in a 20 year prospective-cohorts design. Almost 1200 of the original 1575 participants were interviewed as adults. Approximately 40% of those who were physically abused as children failed to report the abuse. With regard to childhood sexual abuse, about 37% of those who were abused failed to report the abuse. The percentages varied significantly, however, as a function of the relationship the victim had to the perpetrator: 76% reported when the offender was a stranger, 57% when the offender was known and 22% when the offender was a relative. Very similar findings were found by Williams (1994) when 129 women with previously documented histories of sexual victimization in childhood were interviewed 17 years later and asked detailed questions about their abuse histories. Thirty-eight percent of these women did not recall the abuse. Women who were younger at the time of the abuse and those who were abused by someone they knew were more likely to have no recollection for the abuse. To date, however, there is no general consensus in the literature concerning the validity of retrospective self-reports of trauma and dissociation.

4.4.2. Sample selection

The sample included in the present investigation was highly selected and the results may not be generalizable to other clinical populations. In order to be included in study I or study II, subjects had to have a primary diagnosis of PDA, with moderate to severe levels of panic and agoraphobic avoidance and most tended to fall in the severe range. Additionally, as is problematic in much of psychological research, this sample was overwhelmingly Caucasian (97%) and a majority of the subjects were married. It is possible that the relationships between trauma, dissociation and the dependent measures of psychopathology might be different for other ethnic and/or demographic populations. Further, the associations could be very different even across other anxiety-disorder populations. Hence, further research is needed to address these questions.

As stated previously, floor effects were present at pretreatment due to the highly selected nature of this sample. The subjects included in studies I and II were experiencing moderate to severe symptoms of PDA. For this reason the range was restricted and the ability to discover

relationships between pretreatment pathology and trauma and dissociation was therefore limited. In a similar way, but in the opposite direction and to a lesser extent, ceiling effects may have limited detection of these relationships at posttreatment and 1 year follow-up as a significant number of subjects had experienced an amelioration of PDA symptomatology.

4.5. Future directions

One of the unexpected findings was the relationship between sexual trauma experienced after age 14 and various measures of psychopathology, in light of the absence of any significant relationships between sexual trauma experienced before age 14 and psychopathology at any of the assessment periods. This finding was especially interesting given that younger age at the time of the initial sexual abuse was related to increased anxiety and depression at posttreatment. The traumatology literature would suggest that earlier childhood sexual abuse is at least as predictive, or more predictive, of psychopathology as sexual abuse occurring later. It is possible that this unique finding is restricted to highly selected PDA samples or some other unique characteristic of the subject pool. This finding needs to be replicated and further explored.

Given the interesting pattern of relationships between the number of PTSD symptoms and anxious, agoraphobic and generalized symptomatology at pretreatment and 1 year follow-up, but not posttreatment, it would be useful to try to replicate these findings not only with PDA samples but other anxiety disorder populations as well. The more well-established treatments for the various anxiety disorders are quite similar and tend to utilize cognitive and behavioral (especially exposure-based) techniques. It seems possible that this study provides some evidence for the efficaciousness of exposure-based treatments for anxiety disorders in general, as seen at posttreatment. Yet, the cognitive restructuring must target the anxiety disorder specifically to achieve long-term gains. Those subjects who had greater numbers of PTSD symptoms following prior traumatic experiencing also exhibited poorer maintenance of treatment gains and higher relapse rates. Because symptoms related to trauma were not specifically addressed in treatment, a resurgence of these symptoms, many of which overlap with PDA symptoms, may have been experienced thus increasing the likelihood of relapse. It would be interesting to explore whether a similar relationship between PTSD and other anxiety disorders would be detected.

In the present investigation, the QED was significantly related to several measures of depression, agoraphobia, anxiety and general psychopathology. It would be interesting and useful to understand whether these relationships could be replicated using a prospective, rather than retrospective, design and whether the relationships would be found in a variety of clinical samples. Given the QED's ability to predict psychopathology at pretreatment, posttreatment and follow-up, is this evidence that dissociation is causally related to depression, agoraphobia, anxiety and other general psychopathology? Or, rather, is it the trauma which may have preceded the dissociation? Those who are traumatized and also experience dissociation may be at greater risk for psychopathology.

In conclusion, the three hypotheses proposed in this study were supported by the findings. Histories of traumatic experiences and associated elevations in dissociation levels were related to higher levels of psychopathology at pretreatment, posttreatment and 1 year follow-up in a

sample of subjects who had undergone treatment for PDA. Therefore, it appears that severity of pathology at pretreatment is influenced by prior traumatic experiences and dissociation. At posttreatment, trauma and dissociation continued to predict the presence of greater psychopathology and poorer treatment outcome. Finally, at 1 year follow-up, this relationship continued suggesting that subjects who had been traumatized and who experienced dissociative symptoms were less likely to maintain treatment gains and more likely to suffer relapse. These findings would suggest the importance of systematic assessment of traumatic experiences and dissociative symptomatology by clinicians and researchers working with PDA clients specifically, and more generally, with all anxiety disorder clients.

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