Psychotherapy Processes Underlying Sudden Gains in Treatment of PTSD

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Abstract

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To date, there is a dearth of empirical evidence within the "black box" between pre- and post-treatment to fully understand how and why our interventions work. It is essential to take a process-oriented approach with research to elucidate trajectories and mechanisms of individual change in PTSD treatment. Sudden gains, defined as rapid, large symptom improvements during between-session intervals, have been investigated across different samples and interventions and have consistently been associated with better treatment outcome. Utilizing sudden gains as markers of critical points of transition, the in-session therapist-patient interactions prior to the gains were examined using a process-oriented, detailed coding system for potential processes of change associated with sudden gains. This was the first study to systematically examine in-session therapy content for potential mechanisms triggering sudden gains in PTSD treatment. Pre-treatment trait-like factors of distress tolerance and neuroticism and more proximal factors of

fear activation, between-session, and within-session distress reductions were also examined as potential predictors of sudden gains. When examining the pre-gain sessions, patients who experienced sudden gains expressed more positive hope and had more cognitive-emotional processing than those who did not exhibit sudden gains, suggesting these are key elements for discontinuous change. Sudden gains occurred similarly in both PE only and PE combined with sertraline treatment. However, when examining pre-gain in-session content, patients receiving PE only had more cognitive-emotional processing than patients receiving PE combined with sertraline, highlighting potential different mechanisms between the two treatments. Finally, distress tolerance-related absorption predicted the occurrence of sudden gains, suggesting that individuals who pay more attention to negative emotional states and thoughts are more likely to experience a sudden gain. However, no other predictors of neuroticism, fear activation, between-session distress reduction, and within-session distress reduction were associated with sudden gains. All together, the findings of this study bring a better understanding of the sudden gain phenomenon and elucidate what are the key elements of change in PTSD treatment. By enhancing these key elements of change, the efficacy and efficiency of the interventions can be maximized and will allow the tailoring of interventions to specific patients based on his or her needs and strengths of achieving change.

Table of Contents

List of Figures

List of Tables

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Psychotherapy Processes Underlying Sudden Gains in Treatment of PTSD

Prevalence and Impact of PTSD

In the United States, 75% of the population is projected to experience a traumatic event in their lifetime (Sledjeski, Speisman, & Dierker, 2008), and 6.8% will meet criteria for posttraumatic stress disorder (PTSD; Kessler et al., 2005). PTSD is one of the most costly anxiety disorders to treat (Greenberg et al., 1999; Jaycox & Foa, 1999). Although effective psychological and pharmacological treatments are available, there is still great individual variation in treatment response (Ballenger et al., 2004; Davidson, 2001). Thus, there is a new focus on tailoring existing interventions to make them more efficacious for each unique individual. A barrier to significant progress in intervention research is the heavy focus traditional research methods give to outcome data, only comparing pre- to post-treatment (e.g., Brady et al., 2000; Davidson et al., 2001a; Davidson, Rothbaum, van der Kolk, Sikes, & Farfel, 2001b; Foa, Rothbaum, Riggs, & Murdock, 1991; Foa et al., 1999a, 2005; Resick, Nishith, Weaver, Astin, & Feuer, 2002). This approach allows us to identify effective interventions but treats what is occurring during treatment as a "black box." This disregards the fundamental understanding of how and why interventions work and for whom and under what conditions they work (Laurenceau, Hayes, & Feldman, 2007). It is imperative to revise research methods to examine psychotherapy processes within this "black box" to identify mechanisms of change to be personalized for specific individuals.

Process of Change and Sudden Gains

Dynamic systems theory. A key to investigating the "black box" of treatments is to shift the focus to process-oriented approaches in research (Kazdin, 2008). Examining individual trajectories of symptom change can show us the shape and rate of change, which can highlight

critical points of an intervention most likely to reveal potential factors causally related to processes of change (Hayes, Laurenceau, Feldman, Strauss, & Cardaciotto, 2007b). Change in psychotherapy can be gradual and linear; and, in many instances, the change can be discontinuous (Hayes et al., 2007b). Dynamic systems theory (Thelen, 1995; Thelen & Smith, 1994) captures both types of change, and emphasizes the importance of further examining the discontinuous, nonlinear patterns of change. A dynamic system consists of elements that interact and continually evolve over time (Vallacher, Read, & Nowak, 2002). When new elements are introduced into the system and can be easily assimilated, the system reorganizes and the change is gradual and linear. If the elements are challenging the system and are too great to assimilate, then the system becomes perturbed and, instead, a critical fluctuation of discontinuous change occurs (Kelso, Ding, & Schöner, 1993; Salvatore & Tschacher, 2012; van Geert & van Dijk, 2002). These fluctuation patterns are important predictors of transitions (Kelso, 1997; Schiepek, Eckert, & Weihrauch, 2003; van der Maas & Molenaar, 1992). During this period, the system is destabilized and open to new information and exploration of more adaptive associations and configurations. The system then continues to move between its old and new patterns, until it eventually settles into a new dynamically stable state and variability decreases (Kelso, 1997; Thelen & Smith, 1994; van Geert & van Dijk, 2002). Several theorists have suggested that this principle of destabilization before change, mainly examined in other sciences, may also apply to the process of change in psychotherapy (Hager, 1992; Hayes & Strauss, 1998; Mahoney, 1991; Schiepek, Fricke, & Kaimer, 1992). Thus, change in psychotherapy can be gradual and linear, but also can be discontinuous and nonlinear when new interventions and elements are introduced to challenge and destabilize old system patterns in individuals (Hayes et al., 2007b).

Discontinuous change patterns. A variety of discontinuous, change markers have been identified in process research: sudden gains referring to significant reductions in symptoms between any two sessions (Tang & DeRubeis, 1999), rapid early responses referring to substantial decreases in symptoms early in treatment (Ilardi & Craighead, 1999), and spike patterns, the opposite of sudden gains, referring to temporary symptom exacerbation in treatment that are associated with later symptom reduction (Hayes et al., 2007a). Sudden gains clearly mark large symptom improvements in individuals (Tang & DeRubeis, 1999). Rapid early responses typically analyze group means of time courses that obscure change patterns otherwise revealed when plotting individual courses (Tang & DeRubeis, 1999). Spike patterns, though yielding information on symptom worsening, most likely will not yield information on crucial mechanisms underlying improvement (Hayes et al., 2007a). Thus, of these common patterns, sudden gains are most likely to reflect key points of discontinuity underlying rapid symptom improvement.

Empirical Accounts and Significance of Sudden Gains

Sudden gains in treatments for depression. Sudden gains are rapid, large symptom improvements during between-session intervals, and they are operationally defined and identified by specific quantitative criteria devised by Tang and DeRubeis (1999). Sudden gains occur at varying rates in psychotherapy and have largely been examined in treatments for depression. In cognitive therapy for major depression, 39% to 46% of patients exhibited sudden gains (Busch, Kanter, Landes, & Kohlenberg, 2006; Hardy et al., 2005; Tang & DeRubeis, 1999; Tang, DeRubeis, Hollon, Amsterdam, & Shelton, 2007; Vittengl, Clark, & Jarrett, 2005). In individual (Tang, DeRubeis, Beberman, & Pham, 2005) and group (Kelly, Roberts, & Ciesla, 2005) cognitive-behavioral therapy (CBT) for major depression, between 42% and 44% of

individuals experienced sudden gains. A wider range of frequencies of 17% to 50% of sudden gains have been reported in other forms of psychotherapy for depression, such as supportive-expressive therapy (Tang, Luborsky, & Andrusyna, 2002) interpersonal psychotherapy (Kelly, Cyranowski, & Frank, 2007), behavioral activation (Hopko, Robertson, & Carvalho, 2009), non-manualized psychotherapies in clinic settings (Stiles et al., 2003), and non-directive supportive therapy (Gaynor et al., 2003). The majority of these studies have found that individuals who experienced sudden gains have better treatment outcomes and maintenance of improvements through follow-up than those who do not show sudden gains, suggesting they are not random fluctuations of symptoms but rather significant, stable improvements (e.g., Gaynor et al., 2003; Hardy et al., 2005; Stiles et al., 2003; Tang & DeRubeis, 1999).

Only one study to date has looked at the phenomenon of sudden gains in pharmacotherapy for depression (Vittengl et al., 2005). Vittengl and colleagues examined the presence of sudden gains in pharmacotherapy for atypical depression. They investigated sudden gains in cognitive therapy, pharmacotherapy (i.e., phenelzine sulfate) with clinical management, and pill placebo with clinical management. Patients experienced sudden gains at rates of 25% to 47%, and the gains predicted better psychosocial functioning at the end of the acute phase treatment. There were no differences across treatment conditions in frequency or magnitude of the sudden gains. However, patients receiving pill placebo with clinical management experienced higher rates of reversals of sudden gains compared to those receiving cognitive therapy or pharmacotherapy. Thus, the phenomenon of sudden gains is significant and has repeatedly been found across various treatments, including pharmacotherapy, for depression, yet very little is known about sudden gains in pharmacotherapy.

Sudden gains in treatments for PTSD. To date, only four studies have investigated sudden gains in clinical samples of PTSD (Aderka, Appelbaum-Namdar, Shafran, & Gilboa-Schechtman, 2011; Doane, Feeny, & Zoellner, 2010; Jun, Zoellner, & Feeny, 2013; Kelly, Rizvi, Monson, & Resick, 2009). These studies report similar rates of sudden gains as those in the treatment of depression. Kelly and colleagues (2009) assessed for rapid symptom improvements in a sample of women receiving cognitive behavioral therapy, specifically cognitive processing therapy (CPT; Resick et al., 2008), CPT with cognitive therapy only and no written account, or written account only, for PTSD from a sexual or physical assault. Patients experienced sudden gains at rates of 28% to 38%, and they were associated with greater PTSD symptom reduction at post-treatment. These changes were largely associated with reductions in the avoidance/numbing and hyperarousal symptoms. This was the first study to examine sudden gains across three treatment conditions for PTSD, allowing us to see if certain treatment components were associated to the gains. They found no differences across the treatment conditions; however, this may have been due to small sample sizes per group in the analyses. For two of the three conditions, sessions were held twice a week and PTSD self-report scores were only available from every other session. Thus, they modified the calculation criteria of sudden gains and used weekly mean scores instead, which may have inflated the number of people meeting the sudden gains criteria. The study sample was also restricted to adult women who had PTSD from a sexual or physical assault, limiting the generalizability of the findings.

In the second study examining sudden gains in PTSD, Doane and colleagues (2010) examined sudden gains in a small sample of female assault survivors with PTSD undergoing prolonged exposure (PE; Foa, Hembree, & Rothbaum, 2007) therapy. They found that sudden gains occurred in 52% of the sample, and the gains accounted for 61% of overall PTSD symptom

reduction. Again, individuals who exhibited sudden gains had greater reductions in PTSD symptoms than those who did not have sudden gains. This study also examined depression and social functioning measures in relation to the sudden gains and found similar improvement for both in those who exhibited sudden gains and did not in PTSD symptoms. This was the first study to investigate the phenomenon of sudden gains in PE treatment for adults with PTSD with weekly reported PTSD severity at every session. However, the study sample was restricted to exclusively adult women with sexual assault trauma, limiting the generalizability of the findings.

In the third study, Aderka and colleagues (2011) examined sudden gains among children and adolescents (ages 8-17) receiving developmentally adjusted prolonged exposure (Foa, Chrestman, & Gilboa-Schechtman, 2009) therapy for PTSD. Sudden gains were exhibited by 49% of the patients, which constituted 48.6% of the total reduction in PTSD symptoms. Individuals who exhibited sudden gains had lower PTSD symptoms post-treatment and at follow-up periods. This was the first study to extend the examination of sudden gains to children and adolescents with PTSD and showed they were predictive of better long-term treatment outcome. However, there were some limitations in the reliability of child self-report measures utilized to identify the sudden gains, and they did not include self-reports from parents to see if they matched with the children's data.

Finally, in the fourth study, Jun and colleagues (2013) investigated the occurrence of sudden gains in psychotherapy, specifically PE, and pharmacotherapy, specifically sertraline, in a large, randomized trial for PTSD. This is the only study to date that has looked at sudden gains in pharmacotherapy for PTSD. Individuals in both PE (42%) and sertraline (31%) exhibited sudden gains, and these gains were strongly associated with better treatment outcome. The number of patients experiencing sudden gains was similar in PE and sertraline, even after

accounting for higher rates of sudden gain reversals in sertraline. The pattern of gains was different between the two groups. Patients in PE made more multiple gains throughout treatment. Patients in sertraline made larger early gains, while those in PE made more gains toward the end of treatment. This was the first study to examine sudden gains in a large clinical trial for PTSD directly comparing pharmacotherapy and psychotherapy. The different patterns of frequency and magnitude of sudden gains in PE and sertraline may suggest divergent therapeutic mechanisms; however, more proximal potential mediators of sudden gains were not examined. Thus, the four studies confirm that sudden gains in PTSD are in fact significant reductions that are generally stable and maintained throughout treatment.

Pre-Treatment Predictors of Sudden Gains

Pre-treatment predictors in treatments for depression. In order to identify what could be triggering the sudden gains and whether patients are predisposed to the experience of sudden gains, possible pre-treatment predictors have been examined. In the depression literature, several studies have examined pre-treatment symptom severity, cognitive content, social interpersonal functioning, positive and negative life events, and early therapeutic alliance as predictors of experiencing sudden gains, but found no consistent predictors (e.g., Gaynor et al., 2003; Hardy et al., 2005; Kelly et al., 2005). However, one study (Hardy et al., 2005) found that higher self-reported dysfunctional attitudes at pre-treatment were associated with an increased likelihood to exhibit sudden gains. It may be that patients with greater negative attitudes had more opportunities for cognitive shifts, following Tang and DeRubeis' (1999) theory that sudden gains involve cognitive change. In addition, Vittengl and colleagues (2005) found that those with higher pre-treatment depressive symptoms reported more sudden gains. They suggested that patients who begin treatment with greater symptom severity are more likely to experience a

sudden gain, perhaps because they have more to gain and therefore more opportunities for large symptom improvements. However, at the present time, it is unclear what are strong, consistent predictors of sudden gains in depression treatment.

Pre-treatment predictors in treatments for PTSD. Similarly, in the PTSD literature, there are few findings of significant pre-treatment predictors of sudden gains. Studies have examined demographic measures, pre-treatment clinical measures (e.g., PTSD, depression, trauma-related beliefs, anxiety sensitivity), and treatment adherence as possible predictors of sudden gains, but they found no consistent predictors (Aderka et al., 2011; Doane et al., 2010; Jun et al., 2013; Kelly et al., 2009). However, Jun and colleagues (2013) did find that patients of younger age were more likely to experience sudden gains than older patients. One potential explanation may be that a decline of cognitive flexibility with age decreases the ability to quickly gain insights and rapidly modify existing beliefs. Overall, no strong pre-treatment predictors of sudden gains have been found to date; and considering that sudden gains have been observed in diverse samples, this suggests that people are not necessarily predisposed to experiencing a sudden gain. Instead, it may be the processes occurring within treatment are eliciting the gains. This emphasizes the importance of a finer degree of examination within treatment sessions to find potential predictors, mediators, and mechanisms of change that are more proximal to the sudden gains that traditional pre-post designs would not be able to reveal.

Understanding Elements of Sudden Gains

Sudden gains can help elucidate the "black box" problem by identifying critical sessions and transition points for detailed analyses of what is occurring in therapy to trigger significant improvements in symptoms (Hayes et al., 2007b; Tang & DeRubeis, 1999). In the depression literature, two studies (Tang & DeRubeis, 1999; Tang et al., 2005) examined in-session cognitive

change, defined as identifying an error in a belief and arriving at a new belief on a specific issue, in cognitive therapy and CBT by utilizing a coding scale to rate session audio-tape recordings and the corresponding transcripts of the pre-gain and control sessions (pre-pre-gain sessions). They reported more within-session cognitive change prior to the occurrence of sudden gains, consistent with a cognitive mediation hypothesis (e.g., Whisman, 1993), suggesting that cognitive change may underlie sudden gains. However, two studies were not consistent with these findings (Andrusyna, Luborsky, Pham, & Tang, 2006; Kelly et al., 2005). Kelly and colleagues (2005) found no difference in cognitive changes in self-esteem between the pre-gain and control sessions in CBT for depression. It should be noted that, in this study, cognitive changes were assessed from weekly self-report measures rather than actual in-session content. In supportive-expressive therapy for depression, Andrusyna and colleagues (2006) utilized coding scales to rate audio-tape recordings and the corresponding transcripts of the pre-gain and control sessions for cognitive changes, therapeutic alliance, and therapist interpretation accuracy, defined as the congruence between the therapist's interpretation and the patient's core conflictual relationship themes (Luborsky, 1984). They found that sudden gains were preceded by greater therapist interpretation accuracy and slightly better therapeutic alliance. However, the amount of cognitive changes were the same for the pre-gain and control sessions. Thus, depending on the type of treatment, the precipitants of sudden gains may vary.

To date, no study has examined possible in-session precipitants of sudden gains in PTSD nor has any study compared the in-session content between those who exhibited sudden gains and control individuals who did not experience sudden gains. In PTSD treatment, cognitive change is likely to play a similar role, because cognitive shifts and disruptions that occur after a traumatic experience are thought to underlie the development and amelioration of PTSD

symptoms (e.g., Ehlers & Clark, 2000; Foa & Kozak, 1985; 1986; Horowitz, 1976, 1986). Indeed, studies have shown that cognitive changes do occur in psychotherapy for PTSD (e.g., Foa & Rauch, 2004; Resick et al., 2002) and that these cognitive shifts precede PTSD symptom change (Zalta et al., 2014).

Theoretical Processes Underlying Sudden Gains in PTSD

Theoretical link between extinction and cognitive-emotional processing in PTSD.

Exposure-based therapy for PTSD is thought to utilize extinction, where conditioned stimuli (CS) are repeatedly presented in the absence of the unconditioned stimuli (US) and anxiety reduction occurs. Specifically, it is thought that extinction is a form of inhibitory learning in regard to expectancies and contingency beliefs about neutral stimuli (CS) and the fear-eliciting stimulus (US; Bouton, Mineka, & Barlow, 2001; Hofmann, 2008). Extinction plays a large role in Foa and Kozak's (1985, 1986) emotional processing theory. This theory suggests successful treatment of PTSD requires activation of the pathological fear structure and integration of corrective information that is incompatible with this structure. Pathological fear structures contain associations among stimuli, response, and meaning representations that distort reality. A pathological PTSD fear structure is thought to consist of maladaptive associations among the trauma, its reminders, which are essentially safe situations or images, and their meaning of danger or a sense of incompetence (Foa, Huppert, & Cahill, 2006; Foa & Riggs, 1993; Foa & Rothbaum, 1998). Pathological fear structures are thought to be resistant to modification due to behavioral and cognitive avoidance (e.g., avoiding situations that are actually safe) and cognitive biases in processing information (during encoding, interpretation, and retrieval). This prevents acquisition of relevant, corrective information that is inconsistent with the fear structure (Foa et

al., 2006). The integration of new information with the existing fear structure is hypothesized to underlie recovery or successful cognitive and emotional processing of the trauma.

Extinction learning and cognitive-emotional processing are strongly theoretically linked, focusing on the meaning of the CS-US relationship. Inhibitory learning in extinction involves making the *meaning of the CS ambiguous,* such as by altering CS-US expectancy beliefs (Bouton et al., 2001; Hofmann, 2008). Cognitive-emotional processing (Hayes, Feldman, & Goldfried, 2006) derived from emotional processing theory (Foa & Kozak, 1985, 1986) postulates the role of an underlying pathological fear structure, where integration of corrective information similarly alters the *meaning of the CS-US relationship*. Thus, extinction is thought to change the meaning of the CS and its relationship to the US, and cognitive-emotional processing captures this change in meaning. Cognitive-emotional processing captures the combination of affective engagement and arousal, cognitive analysis, and meaning making, which may in turn be the process of change underlying the occurrence of sudden gains in PTSD treatment.

Theoretical link between cognitive theory and cognitive-emotional processing in PTSD. Many trauma theories note the importance of changes in the survivor's cognitions and beliefs and their role in the development of PTSD (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000; Epstein, 1991; Foa & Kozak, 1985, 1986; Horowitz, 1986; Janoff-Bulman, 1992). In particular, there are several key psychosocial theories that specifically highlight the role of negative trauma-related beliefs in PTSD (e.g., Ehlers & Clark, 2000; Foa & Kozak, 1985, 1986; Horowitz, 1986).

Stress response model. Horowitz' (1986) stress response model proposes that trauma-exposed individuals initially experience an emotional response, followed by attempts to process and assimilate the experience with their pre-existing belief schemas about the self and world.

The reconciliation of old and new trauma-related information is characterized by alternating phases of intrusions and denial. Gradual incorporation of new information leads to resolution of the discrepancies between prior and new beliefs, whereas, failure to process and integrate the trauma-related beliefs leads to persistent psychological reactions.

Emotional processing theory. Foa and Kozak's (1985, 1986) emotional processing theory, as described above, attributes post-trauma symptoms to pathological fear structures containing maladaptive associations among trauma-related stimuli, response, and meaning representations that distort reality. Often the erroneous perceptions are about the world as utterly dangerous and oneself as totally incompetent (Foa & Riggs, 1993; Foa & Rothbaum, 1998). Updates on emotional processing theory place more emphasis on the modification of these erroneous associations for successful treatment of PTSD (Foa et al., 2006). Thus, successful recovery after trauma requires activation of the pathological fear structure and integration of corrective information that is inconsistent with the fear structure particularly containing the negative trauma-related beliefs.

Cognitive model of PTSD. Ehlers and Clark's (2000) cognitive model of PTSD proposes that processing the trauma in a way that leads to a belief that there is serious, ongoing threat in the world underlies the persistence of PTSD. This is thought to be a consequence of negative and overgeneralized appraisals of the trauma, one's reactions during the trauma, and its sequelae, where individuals are unable to perceive the trauma as a discrete, time-limited event that does not necessarily have negative implications for the future.

Across these cognitive theories, although they propose varying psychological processes as key to the development and maintenance of PTSD, they all highlight the role of distorted trauma-related thoughts and beliefs contributing to PTSD. Negative trauma-related beliefs tend

to focus on a few core themes in the area of self, world, and blame. Negative self beliefs include negative views of self or self-worth, incompetence, permanent change, alienation, hopelessness, lack of self-trust, and negative interpretations of symptoms (e.g., Epstein, 1991; Foa, Ehlers, Clark, Tolin, & Orsillo, 1999; Janoff-Bulman, 1996). Negative world beliefs include thoughts of an unsafe world and mistrust of people (e.g., Epstein, 1991; Foa et al., 1999b; Janoff-Bulman, 1992; McCann & Pearlman, 1990). Negative self-blame beliefs include blaming self or guilt for the trauma and the maintenance and intolerance of post-trauma symptoms (e.g., Foa et al., 1999b; Kubany et al., 1996). The empirical literature consistently reports trauma survivors with PTSD exhibiting greater distorted and maladaptive appraisals of the trauma compared to trauma-exposed individuals without PTSD (e.g., Ali, Dunmore, Clark, & Ehlers, 2002; Foa et al., 1999b; Nasby & Russell, 1997). Studies have also found that negative trauma-related beliefs predicted the development of PTSD or higher PTSD severity (e.g., Ali et al., 2002; Ehring, Ehlers, & Glucksman, 2006; Moser, Hajcak, Simons, & Foa, 2007; Owens, Chard, & Cox, 2008). The increasing evidence of the centrality of trauma-related beliefs to PTSD has led to the incorporation of a separate symptom criterion of negative alterations in cognitions for PTSD diagnosis in the DSM-5 (Friedman, Resick, Bryant, & Brewin, 2011). These beliefs change with successful treatment, including exposure-based treatment (e.g., Ehlers, Clark, Hackmann, McManus, & Fennell, 2005; Foa & Rauch, 2004, Resick et al., 2002), and changes in cognitions precede changes in PTSD symptoms in treatment (Zalta et al., 2014). Specifically, in exposure-based treatment, patients are provided the opportunity to directly approach and counter maladaptive cognitions, incorporating corrective information into the fear structure (Foa & Kozak, 1985, 1986). Although cognitive distortions or maladaptive beliefs are not targeted in a

direct sense, cognitive and emotional processing of the trauma is thought to occur and may underlie sudden gains.

Processes of Change Assessed with the CHANGE Coding System

Thus, a potential factor underlying sudden gains in PTSD may be cognitive-emotional processing of negative trauma-related beliefs. Hayes and colleagues (2006) developed the Change and Growth Experiences Scale (CHANGE) in order to operationalize cognitive-emotional processing and other therapeutic processes hypothesized to be important predictors of treatment outcome in CBT treatments. CHANGE is an observational measure that can be used to code narratives or therapy sessions. It has been utilized to assess the process of change in CBT treatments for depression and personality disorders (Hayes, Beevers, Feldman, Laurenceau, & Perlman, 2005; Hayes, Yasinski, Ready, & Laurenceau, 2014; Hayes et al., 2006; 2007a) and developmental interventions for children (Dozier, Peloso, Lewis, Laurenceau, & Levine, 2008). The coding system includes a range of variables for patients and therapists that assess cognitive, affective, behavioral, somatic, and interpersonal aspects of functioning. The CHANGE categories that were coded for this study were cognitive-emotional processing, positive hope, negative hope, unproductive processing, avoidance, positive self, negative self, and therapist support stabilization. The categories particularly relevant for therapeutic change are cognitive-emotional processing, positive hope, unproductive processing, and avoidance.

Cognitive-emotional processing. The CHANGE category of cognitive-emotional processing is defined as the exploration and questioning of difficult experiences and emotions with some change in meaning or shift in perspective. Thus, cognitive-emotional processing represents aspects of both cognitive and affective change. This category captures concepts labeled as emotional processing, meaning-making, benefit-finding, cognitive change, and

schema change. Cognitive-emotional processing is a dynamic process, ranging from fleeting, superficial realizations to more in-depth, substantial understandings, and is often accompanied by high levels of emotion. Affective arousal without some insight or perspective shift is not considered cognitive-emotional processing. In several studies, peak levels of cognitive-emotional processing have consistently been found to be associated with better treatment outcome in CBT for depression (Hayes et al., 2005; 2007a, 2014), thus, highlighting its role as a potential key process of change that facilitates symptom reduction.

Positive hope. Positive hope is another CHANGE category that can facilitate improvement. Positive hope is the extent the person describes an expectation that the future will be better and progress can be made on problem areas. It also captures noticing positive change or making a commitment to change. Hope has been proposed to be a crucial process of change across different treatments (e.g., Howard, Moras, Brill, Martinovich, & Lutz, 1996; Snyder, Ilardi, Michael, & Cheavens, 2000). In fact, decreases in hopelessness predicted better treatment outcomes in depression (e.g., Kuyken, 2004). Utilizing CHANGE, Hayes and colleagues (2007a) found that positive hope preceded rapid early symptom reductions and was associated with better depression treatment outcome.

Unproductive processing and avoidance. When examining processes of change, it is helpful to also assess factors that may inhibit symptom reduction. Unproductive processing is conceptually the opposite of cognitive-emotional processing. This category captures concepts labeled as rumination, worry, or venting, which are all hallmark symptoms of depression and anxiety (Borkovec, 2002; Siegle, 2008). Unproductive processing is characterized by repetitive questioning and analyzing without any insight. Thus, unproductive processing can also function as an avoidance technique that prevents deep experiencing (Hayes et al., 2006). The CHANGE

category of avoidance is defined as difficulty facing disturbing emotions, thoughts, or circumstances and attempts to block this distress. Avoidance often has behavioral manifestations where an individual may attempt to block disturbing experiences or emotions by avoiding therapeutic tasks, isolating oneself from certain situations, or numbing oneself from emotional experiences. Hayes and colleagues (2005) found that in depression treatment, peak levels of avoidance were associated with less improvement.

CHANGE and sudden gains. The CHANGE categories are not mutually exclusive and can co-occur. Often times the different categories exhibit similar time courses, such as positive self and positive hope increasing together (Hayes et al., 2005). Or they can exhibit different time courses, such as positive hope preceding cognitive-emotional processing (Hayes et al., 2007a). Thus, in order to get a picture of the processes of change in an individual in treatment, it is important to examine these different factors simultaneously. Given that cognitive-emotional processing has consistently been associated with better treatment outcome, it is possible that this process of change underlies sudden gains. Thus, it would be meaningful to examine the time course of cognitive-emotional processing to see if high levels of this category in the pre-sudden gain sessions predict the experience of sudden gains.

Treatment Modality and Sudden Gains

One method of further understanding the mechanisms of change and for whom and under what conditions change occurs, is to examine different treatment modalities. PE, a cognitive-behavioral therapy, is one of the most common psychotherapies for PTSD. PE involves psychoeducation about common reactions to a trauma, breathing retraining, *in vivo* exposure to situations that are feared or avoided due to the traumatic event, imaginal exposure to the trauma memory, and processing of the imaginal exposure. Its efficacy has been replicated across many

trials (Foa et al., 1991, 1999a, 2005; Nacasch et al., 2011; Schnurr et al., 2007) and has been established in comparison to other treatments (Foa et al., 1999a; 2005; Resick et al., 2002; Taylor et al., 2003). Another common treatment for PTSD is pharmacotherapy with selective serotonergic reuptake inhibitors (SSRIs). Sertraline is one of the FDA-approved SSRIs for the treatment of PTSD, and it has shown better efficacy than placebo (Brady et al., 2000; Davidson et al., 2001a, 2001b) and comparable efficacy compared to other serotonergic agents (Davidson et al., 2006; McRae et al., 2004).

In the anxiety and stressor-related disorders, it is not clear that combined treatment of psychotherapy and psychotropic medication improves treatment outcome (e.g., Otto, McHugh, & Kantak, 2010; Pontoski & Heimberg, 2010). In PTSD, there are no large-scale trials that have examined combination treatment. Only two small clinical studies have examined stepped-care treatment (not fully combined) for PTSD (Rothbaum et al., 2006; Simon et al., 2008), with findings being mixed as to whether stepped care provided greater efficacy. Rothbaum and colleagues (2006) found additive benefits in individuals who received 10 weeks of open label sertraline and then 10 sessions of twice-weekly PE. The stepped-care treatment led to further reductions in PTSD symptom severity. However, this augmentation effect was only observed in patients who exhibited a partial response to the medication initially. Simon and colleagues (2008) did not find additive benefit of augmenting continued PE with paroxetine-CR compared to pill placebo in PTSD treatment.

Thus, further understanding of the mechanisms of change for the different treatment modalities is needed to elucidate the effects of combined treatment. In PE, patients are provided the opportunity to approach their trauma-related fears through imaginal exposure, followed by processing of the exposure where patients' reactions and any related thoughts are discussed (Foa

et al., 2007). The bulk of cognitive-emotional processing should occur during this processing component of imaginal exposure, where extinction learning is thought to be consolidated; however, this critical component of PE has not been systematically studied. The most widely accepted theory of mechanism of SSRI's is that they may reverse neuronal deficits by increasing neurogenesis, specifically increasing the hippocampus volume in PTSD patients, which is associated with declarative memory (Thomaes et al., 2013). This may help patients more skillfully process and consolidate cognitive material during exposure learning. Thus, combining the effects of PE and sertraline in augmented treatment may lead to more occurrences of cognitive-emotional processing and sudden gains compared to monotherapy.

Psychosocial Pre-Treatment Markers Predicting Sudden Gains

Although the findings of pre-treatment predictors of sudden gains have been sparse, there are several key concepts of trait-like factors that are consistently linked to psychopathology that have not yet been examined, such as distress tolerance and neuroticism. To date, no study has investigated the relationship between these factors and sudden gains.

Distress tolerance. Distress tolerance is the perceived or actual capacity to withstand aversive experiential states (Simons & Gaher, 2005), such as negative emotion, uncertainty, and physical discomfort (Zvolensky et al., 2010). Several studies found that lower levels of distress tolerance were associated with greater PTSD symptom severity (Marshall-Berenz, Vujanovic, Bonn-Miller, Bernstein, & Zvolensky, 2010; Vujanovic, Bonn-Miller, Potter, Marshall, & Zvolensky, 2011). Individuals with low distress tolerance may perceive PTSD symptoms as more threatening and intense and may engage more in strategic efforts to avoid trauma related stimuli (Vujanovic et al., 2011). In fact, low levels of distress tolerance had a mediating effect between PTSD symptom severity and drug or alcohol use as coping mechanisms (e.g., Marshall-

Berenz, Vujanovic, & MacPherson, 2011; Potter, Vujanovic, Marshall-Berenz, Bernsein, & Bonn-Miller, 2011). To date, no study has examined the association between distress tolerance and PTSD treatment outcomes. Thus, high levels of distress tolerance may facilitate the individual to stay engaged with the treatment and treatment tasks, promoting the occurrence of cognitive-emotional processing and sudden gains.

Neuroticism. Neuroticism measures the general tendency to experience negative mood states, such as worry, anxiety, depression, irritability, and vulnerability (Eysenck & Eysenck, 1964). It has also been defined as the disposition to perceive threat easily and to become quickly aroused (Craske, 1999). Neuroticism is thought to be a risk and maintenance factor of various psychological and physical disorders (for a review see Lahey, 2009). In the PTSD literature, neuroticism has consistently been reported as a risk factor to the development of PTSD (e.g., Breslau & Schultz, 2013; Hyer et al., 2003; McFarlane, 1988, 1989; Talbert, Braswell, Albrecht, Hyer, & Boudewyns, 1993). There is only one study, to date, that has looked at the associations between trait neuroticism and treatment response in PTSD (van Emmerik, Kamphuis, Noordhof, & Emmelkamp, 2011). They found that trait neuroticism was neither related to treatment dropout or poorer treatment response. However, in the depression literature, higher neuroticism generally, consistently predicted worse treatment outcome (for a review see Mulder, 2002). Thus, high levels of neuroticism may be a factor that could inhibit the occurrence of cognitive-emotional processing and, therefore, the occurrence of sudden gains.

Behavioral In-Session Markers Predicting Sudden Gains

The lack of pre-treatment predictors of sudden gains brings reason to examine predictors that are more proximal to the occurrence of the gains. According to emotional processing theory (Foa & Kozak, 1985; 1986), fear activation, between-, and within-session decreased fear

responding are thought to be critical for the integration of incompatible information into the pathological fear structure. Thus, fear activation and distress reduction during imaginal exposure and across treatment sessions may affect the experience of sudden gains. Typically, between-session distress reduction, rather than fear activation and within-session distress reduction, has been found to be more strongly associated with better treatment outcome in the general anxiety literature (e.g., Baker et al., 2010; Craske et al., 2008). However, fear activation, between- and within-session distress reduction are not necessary for better treatment outcome (Craske et al., 2008), possibly because distress tolerance and the ability to withstand experiential discomfort can still promote corrective learning incompatible to the fear structure.

To date, only four studies have looked at the role of between-session distress reduction in exposure therapy for PTSD treatment. Jaycox and colleagues (Jaycox, Foa, & Morral, 1998) found that individuals who exhibited a pattern of higher fear activation and gradual distress reduction during imaginal exposure across treatment had better post-treatment end-state functioning and lower PTSD symptoms compared to those who did not exhibit between-session distress reduction. Similarly, Rauch and colleagues (Rauch, Foa, Furr, & Filip, 2004) found that individuals who exhibited greater between-session distress reduction of peak fear during the imaginal exposure over the course of treatment exhibited greater reductions in PTSD symptoms post-treatment. van Minnen and Hagenaars (2002) found that early between-session distress reduction during imaginal exposure was associated with better treatment outcome. Finally, Bluett and colleagues (Bluett, Zoellner, & Feeny, 2014) found only a minority of patients exhibited reliable change in distress (35%), while the majority of patients did not exhibit reliable change (65%) over the course of imaginal exposure sessions. There were no differences in overall post-treatment PTSD diagnostic status between those who exhibited a reliable distress

change and those who did not; however, those who had reliable change did report lower PTSD symptom severity and better functioning post-treatment than those without reliable distress change. Thus, while not necessary to improve, there is preliminary evidence that between-session distress reduction during imaginal exposure may be associated with better treatment outcome. To date, no research has been conducted examining the association between distress reduction and the presence of sudden gains. It is possible that greater between-session distress reduction during imaginal exposure may facilitate the occurrence of more positive hope and cognitive-emotional processing, thus, promoting the experience of sudden gains.

Current Study

To date, there is a dearth of empirical evidence within the "black box" between pre- and post-treatment to fully understand how and why our interventions work. It is essential to take a process-oriented approach with research to elucidate trajectories and mechanisms of individual change in PTSD treatment. The current study examined individual trajectories of symptom change by investigating the occurrence of sudden gains in individuals receiving PE only or PE combined with sertraline treatment for chronic PTSD. Utilizing sudden gains as markers of critical points of transition, the in-session content of the processing of imaginal exposure prior to the sudden gains were examined in-depth with the CHANGE coding system for potential processes of change associated with sudden gains. The pre-gain sessions of patients who experienced sudden gains and randomly matched sessions of patients who did not exhibit sudden gains were coded. This was the first study to systematically examine in-session therapy content for potential mechanisms triggering sudden gains in PTSD treatment. Finally, various trait-like factors of distress tolerance and neuroticism and behavioral markers of distress reduction were looked at to see if they predicted the occurrence of sudden gains.

Following the consistent findings of sudden gains being associated with better treatment outcome, it was predicted that sudden gains would be associated with better treatment outcome measured in PTSD symptom severity. Since cognitive-emotional processing, positive hope, unproductive processing, and avoidance have been found to be key processes of change in depression treatment and may encompass critical changes in PTSD, it was predicted that individuals who exhibit sudden gains will show more cognitive-emotional processing and positive hope and less unproductive processing and avoidance within the pre-sudden gain session than those who do not experience sudden gains. Due to the potential combined effects of differential mechanisms of PE and sertraline, it was predicted that augmented treatment would be more effective than monotherapy, and individuals receiving PE combined with sertraline will be more likely to exhibit sudden gains and more cognitive emotional processing and positive hope and less unproductive processing and avoidance than those who receive PE alone. Following the findings that low levels of distress tolerance and high levels of neuroticism were associated with greater symptom severity, it was predicted that lower neuroticism and higher distress tolerance would be associated with an increased likelihood of sudden gains. Finally, given greater between-session distress reduction is associated with better treatment outcome, it was predicted that greater between-session distress reduction during imaginal exposure between the pre-pre-gain and pre-gain sessions would be associated with an increased likelihood of sudden gains.

Method

Participants

Sixty treatment-seeking women (78%; $n = 47$) and men (22%; $n = 13$) with chronic PTSD were selected consecutively from an ongoing, larger randomized NIMH-funded clinical trial conducted at the University of Washington (2R01MH066347) and Case Western Reserve

University (2R01MH066348). Inclusion criteria were purposely broad and exclusion criteria limited to recruit a clinically representative sample. Inclusion criteria included: having a current DSM-IV primary chronic PTSD diagnosis (American Psychiatric Association, 1994) and being between the ages of 18 and 65. Exclusion criteria included: having a current diagnosis of schizophrenia or delusional disorder; medically unstable bipolar disorder, depression with psychotic features, or depression severe enough to require immediate psychiatric treatment (e.g., actively suicidal with intent and plan); severe self-injurious behavior or suicide attempt within the past three months; no clear trauma memory or trauma before age of three; current diagnosis of alcohol or substance dependence within the previous three months; ongoing intimate relationship with the perpetrator (in assault cases); unwilling or medically not advisable to stop current trauma focused behavioral psychotherapy or antidepressant medication, based on condition assignment; previous non-response to an adequate trial of either PE (8 sessions or more) or sertraline (150 mg/d; 8 wks); or medical contraindication for the initiation of sertraline (e.g., pregnancy/likely to become pregnant).

Patients' mean age was 36.77 years (*SD* = 12.43). The majority of patients were Caucasian (63%), not college educated (55%), and many had an annual household income of $20,000 or less (47%). Target trauma experiences were distributed as follows: 32% reported an adult sexual assault, 28% adult non-sexual assault, 15% childhood sexual assault, 7% childhood non-sexual assault, 7% motor vehicle accident, and 11% other traumas (i.e., death or violence to a loved one, medical complications). Mean time since the target trauma was 12.93 years (*SD* = 13.52).

Interview Measures

Structured Clinical Interview for DSM-IV (SCID-IV; First, Spitzer, Gibbon, &

Williams, 1995). The SCID-IV is a semi-structured diagnostic interview used to assess a current primary diagnosis of PTSD and co-occurring disorders at pre-treatment as well as past and exclusion DSM-IV diagnoses. It has an acceptable inter-rater reliability (κ = .70 - .94; Skre, Onstad, Torgersen, & Kringlen, 1991).

Posttraumatic Symptom Scale – Interview (PSS-I; Foa, Riggs, Dancu, & Rothbaum, 1993). This is a 17-item interview measure used to assess current DSM-IV PTSD diagnosis and severity by independent evaluators. Items are rated on a 0 (*not at all*) to 3 (*5 or more times a week/very much*) scale to measure frequency and severity of symptoms within the past two weeks in reference to their target trauma. The measure has an inter-rater reliability of κ = .97 for diagnosis and *r* = .97 for symptom severity scores (Foa et al., 1993). A high correlation of *r* = .87 was found between the Clinician-Administered PTSD Scale (Blake et al., 1995) and the PSS-I total scores (Foa & Tolin, 2000). This measure was given at the pre-treatment and post-treatment evaluations as a primary measure of outcome.

Quick Inventory of Depressive Symptomatology – Clinician Rating (QIDS-C; Rush et al., 2003). This is a 16-item interview measure used to screen for depression severity. Responses are based on the past two weeks, ranging from 0 (*little impairment*) to 3 (*extreme impairment*) points. This measure has good internal consistency, with α = .75, and good convergent validity, with a correlation of *r* = .86 with the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). This measure was given at the pre-treatment and post-treatment evaluations as a measure of secondary outcome.

Self-Report Measures

Posttraumatic Symptom Scale – Self-Report (PSS-SR; Foa et al., 1993). This is a 17-item self-report measure of PTSD symptoms within the past week that corresponds with the

DSM-IV of PTSD. Items are rated on a 0 (*not at all*) to 3 (*5 or more times a week/very much*) scale to measure frequency and severity of symptoms in reference to their target trauma. The items are clustered into re-experiencing (e.g., feeling very emotionally upset when reminded of the trauma), avoidance (e.g., trying to avoid activities, people, or places that remind of the trauma), and arousal (e.g., being overly alert) symptoms. The PSS-SR has good convergent validity with the SCID, correctly identifying 86% of individuals with a DSM-III PTSD diagnosis, and a test-retest reliability of .74 (Foa et al., 1993). This measure was completed at pre-treatment, prior to every treatment session, and at post-treatment. The PSS-SR was used to calculate sudden gains.

Eysenck Personality Questionnaire Revised-Neuroticism Scale (EPQ-R; Eysenck, Eysenck, & Barrett, 1985). The neuroticism subscale consists of 24-items, each assessed on a dichotomous scale (0 = *no*, 1 = *yes*), questionnaire that measures the predisposition to experience negative emotions, such as worry, anxiety, depression, irritability, and vulnerability. The measure has good test-retest reliability for the neuroticism subscale of .76 for males and .81 for females (Eysenck et al., 1985). This measure was given at the pre-treatment evaluation as a trait predictor of sudden gains.

Distress Tolerance Scale (DTS; Simons & Gaher, 2005). This is a 15-item self-report measure on a 5-point Likert-type scale (1 = *strongly agree* to 5 = *strongly disagree*) of the extent individuals believe they can experience and withstand distressing emotional states. The measure consists of four subscales: tolerance, appraisal, absorption, and regulation. These subscales reflect the perceived ability to tolerate emotional distress (e.g., “I can’t handle feeling distressed or upset”), subjective appraisal of distress (e.g., “My feelings of distress or being upset are not acceptable”), attention being absorbed by negative emotions (e.g., “When I feel distressed or

upset, I cannot help but concentrate on how bad the distress actually feels"), and regulation efforts to alleviate distress (e.g., "I'll do anything to avoid feeling distressed or upset") respectively. Higher scores represent higher general distress tolerance. The DTS has good convergent validity with other measures of affective functioning and a test-retest reliability of .63 (Simons et al., 2005). This measure was given at the pre-treatment evaluation as a potential trait predictor of sudden gains.

Post-Traumatic Cognitions Inventory (PTCI; Foa, Ehlers, Clark, Tolin, & Orsillo, 1999b). This is a 33-item self-report measure that assesses negative trauma-related thoughts and beliefs. This measure yields three subscales: negative cognitions of self, negative cognitions of world, and self-blame. Higher scores represent greater negative beliefs. The PTCI has a test-retest reliability of .89 and good internal consistency for each factor (α = .86-.97; Foa et al., 1999b). This measure was completed at pre-treatment and prior to every treatment session to assess for negative trauma-related cognitions.

Subject Units of Distress (SUDs; Wolpe & Lazarus, 1966). SUDs are self-ratings of distress ranging from 0 (*complete relaxation*) to 100 (*maximum distress*). SUDs ratings are commonly utilized as a measure of distress in the treatment of anxiety disorders (e.g., van den Hout, van der Molen, Griez, Lousberg, & Nansen, 1987). SUDs of 0 is anchored to a situation when the patient feels no discomfort at all or complete relaxation. SUDs of 100 is anchored to a situation when the patient felt extreme fear or anxiety, the most he or she has ever been in their life. During imaginal exposure (30-60 min), SUDs ratings were recorded every 5 min. SUDs ratings correspond well to other measures of fear expression and physiological indicators (Foa, Riggs, Massie, & Yarczower, 1995; Griez, Lousberg, van den Hout, & van der Molen, 1987; van den Hout et al., 1987).

For this study SUDs ratings were utilized to calculate fear activation, between-session, and within-session distress reduction. The SUDs ratings taken every 5 min during imaginal exposure were used as the primary measure of fear activation, between-session, and within-session distress reduction following the formula described by Kozak, Foa, and Steketee (1988). Fear activation was measured as the peak response of an imaginal exposure trial of session N (pre-gain session). Two different types of between-session distress reduction were computed. The first one was measured as the difference in peak responses from session N-1 (pre-pre-gain session) to session N+1 (gain session). A second more proximal to the sudden gain between-session distress reduction was calculated as the difference in peak responses from session N (pre-gain session) to session N+1 (gain session). Within-session distress reduction was measured as the difference between peak response and the end response of an imaginal exposure trial of session N (pre-gain session).

Treatments

Prolonged exposure alone (Foa, Hembree, & Dancu, 2002). PE consisted of 10 weekly sessions that were 90-120 min. PE involved psychoeducation about common reactions to a trauma, breathing retraining, *in vivo* exposure to situations that are feared or avoided due to the traumatic event, repeated prolonged imaginal exposure to the trauma memory (30-40 min), and processing of the imaginal exposure (15-20 min). The first session consisted of orienting the patient to the treatment. During the second session, psychoeducation and *in vivo* exposure were introduced. During session three, imaginal exposure began (45-60 min). Sessions 3-10 consisted of reviewing *in vivo* and imaginal exposure homework completion, conducting of imaginal exposure (30-45 min), processing of imaginal exposure (15-20 min), and assignment of next week's homework. PE therapists were masters or PhD level clinicians with some CBT

experience, who were trained in the delivery of PE prior to treating patients on the trial. Therapists received standardized clinical training via training workshops. Treatment sessions were digitally recorded. PE supervision occurred weekly at each site including case discussion and tape review.

In PE, patients were provided the opportunity to approach their trauma-related fears through imaginal exposure, followed by processing where patients were encouraged to discuss their thoughts and reactions to the imaginal exposure (Foa et al., 2002). Therapists are instructed to encourage patients to talk about their reactions to revisiting the trauma memory and discussing feelings and thoughts they may have about the trauma or its meaning in their lives. It is common for patients to emerge from imaginal exposure with new awareness or insights, and therapists are instructed to encourage patients to describe and expand on these insights and make them more explicit. The bulk of cognitive-emotional processing should occur during this processing component of imaginal exposure, where extinction learning is thought to be consolidated. Accordingly, this processing section of the pre-gain sessions was selected for CHANGE coding.

Prolonged Exposure and sertraline. PE + SER consisted of the same psychotherapy structure as PE alone but with the additional component of meeting with a psychiatrist who prescribed and monitored side effects and responses to sertraline. Patients met with the psychiatrist for six 15-30 min (session 1, 2, 4, 6, 8, and 10) medication visits in conjunction with weekly PE sessions. Sertraline was prescribed using a standardized, yet flexible titration schedule with a maximum dosage goal of 200 mg/day by week six (Brady et al., 2000). Psychiatrists provided general encouragement and support. All study psychiatrists were board certified and experienced in the treatment of anxiety disorders. Medication sessions were also digitally recorded. PE therapists and psychiatrists were allowed to discuss the progress and any

clinical issues.

Sudden Gains

The weekly PSS-SR ratings were used as the primary measure of PTSD to identify the individuals who experienced and did not experience sudden gains. We utilized Tang and DeRubeis' (1999) three primary criteria for identifying sudden gains. A sudden gain was identified when all the three criteria were met. We consecutively selected patients who have been randomized and completed at least five treatment sessions. This criterion was based on Tang and DeRubeis (1999), where individuals were excluded if an adequate definition of sudden gains could not be made. For missing data, the previous observation was used, conservatively limiting sudden gains and in line with previous studies (Aderka et al., 2011; Doane et al., 2010; Drymalski & Washburn, 2011).

Gain is large in absolute terms (Criterion 1). Sudden gains are large in absolute terms between any session N (pre-gain session) and session N+1 (post-gain session). See Figure 1. Sudden gains of PTSD symptoms were identified when there was a reduction of at least seven points on the PSS-SR. This cutoff value was derived using the guidelines of Jacobson and Truax (1991) for computing reliable clinically significant change. The standard error difference in PTSD symptoms on the PSS-SR between two administrations of the measure was 6.15 (Devilly & Foa, 2001). This change in PTSD symptom severity was based on the PSS-SR measure's two to three week test-retest reliability reliability (.83) and standard deviation (10.54; Foa et al., 1993). Tang and DeRubeis (1999) chose a BDI (Beck et al., 1961) cutoff of one standard deviation from the clinical sample mean, constituting 11% of the range. Although this is illustrative of one possible way to define and identify sudden gains, one standard deviation change on the PSS-SR may not be clinically comparable to one standard deviation change on the

BDI. Thus, the cut-off value for this study was based on the measures reliable clinically significant change value. Comparably, the cut-off value for this study of seven on the PSS-SR constitutes 13.7% of the range.

Gain represents at least 25% reduction from pre-gain symptom level (Criterion 2). The magnitude of the gain is large compared to the pre-gain symptom severity, with at least a 25% reduction of the pre-gain session PSS-SR score. See Figure 2.

Mean symptom level in three pre-gain sessions significantly higher than mean level in three post-gain sessions (Criterion 3). The magnitude of the gain is large relative to symptom fluctuations before and after the sudden gain. This was determined by comparing the mean PSS-SR score of the three pre-gain sessions (sessions N - 2, N - 1, and N) to the mean score of the three post-gain sessions (N + 1, N + 2, and N + 3) using a two-sample t test. See Figure 3. We utilized critical values of $t(4) = 2.78$, $p = .05$ or $t(3) = 3.19$, $p = .05$. when only 2 pre- or post-gain sessions are available to check for significant differences in means.

Reversals of sudden gain. In order to ensure the sudden gains were sustained, reversals of sudden gains when PTSD symptoms increased were calculated. Following Tang and DeRubeis (1999), reversal of sudden gains was defined when there was an increase in reported symptoms of at least 50% of the original improvement of the sudden gain at any point later in treatment.

In-Session Process Coding Using CHANGE

Selection of therapy sessions. After identification of individuals who exhibited sudden gains and those who did not, the pre-gain sessions for individuals who experienced sudden gains were coded. These pre-gain sessions were compared to randomly matched sessions of individuals who did not exhibit sudden gains, which were selected by generating random patient

ID numbers through SPSS. The control sessions were matched in order to control for phase and nature of the therapy content. Thus, the processing of imaginal exposure sections of pre-gain (N) sessions of individuals who experienced sudden gains and did not were coded. Only pre-gain sessions of sessions 3-10 of PE were coded, since processing of imaginal exposure begins at session 3. If an individual had more than one sudden gain, only the pre-gain session of the first sudden gain was coded, in order for each observation to be independent and representative of one unique patient.

Change and Growth Experiences Scale (CHANGE; Hayes et al., 2006). CHANGE is an observational measure, capturing therapeutic processes thought to be crucial in cognitive-behavioral therapies. It can be used to code narratives or therapy sessions. This coding system was used to code content of digital video recordings of therapy sessions. For the current study, the coding system was adapted for use in PTSD treatment trials, specifically PE. Specific coding guidelines were created for the homework review, imaginal exposure, and processing sections of therapy respectively, providing coders various content examples for each of the different sections of PE. Timing guidelines (e.g., code content that reflects the past week up until the current session) were added for all the variables in order to effectively assess change with time. CHANGE assesses a range of variables that include views of self, hope, cognitive-emotional processing, unproductive processing, avoidance, and therapist support. Ratings are on a four-point scale: 0 (*not present or very low*), 1 (*low*), 2 (*medium*), and 3 (*high*). Coding variables are not mutually exclusive and can co-occur. Inter-rater agreements on these categories are good to excellent (*ICC* = .73 - .89; Hayes et al., 2005, 2007a, 2014). For this study, estimates of inter-rater agreement were based on coding the processing of imaginal exposure sections from 60 sessions from 60 patients. According to Cicchetti (1994), *ICCs* below .40 reflect “poor”

agreement, *ICCs* from .40 to .59 reflect "fair" agreement, *ICCs* from .60 to .74 reflect "good" agreement, and *ICCs* .75 and higher reflect "excellent" agreement. Overall, agreement was in the fair to excellent range on all coding categories (*ICCs* = .56 - .83).

Cognitive-emotional processing. Cognitive-emotional processing is the exploration and questioning of difficult experiences and emotions with new connections and meaning leading to insight and a shift in perspective. This variable consists of both cognitive and affective change, since significant insight is usually associated with emotional and behavioral manifestations. This variable would capture concepts of emotional processing, meaning making, benefit finding, cognitive change, and schema change. An example of a coded 3 *cognitive-emotional processing* was, "Then it hit me, I must stop running. I run and run so that I won't get hurt, but then I can't feel at all, and I am alone. I want to feel again, connect with other people." Cognitive-emotional processing is hypothesized to be a key mechanism of change in exposure treatments and has been found to be associated with greater improvements in depression treatments (Hayes et al., 2005; 2007a). The inter-rater agreement for cognitive-emotional processing was *ICC* = .56.

Hope. Hope captures the individual's capacity to see the possibility of change in the future, to recognize recent positive changes, recognition of feeling better, and/or an expression of commitment or determination to make changes. An example of a coded 3 *positive hope* was, "I am beginning to see a way out of this black hole. I believe I will make it." An example of a coded 3 *negative hope* was, "I hate my life, I feel stuck. I can't see a way out." Hope includes both the motivation for change and the belief that one can effect change and has been found to be associated with early rapid reductions in depression symptoms (Hayes et al., 2007a). The inter-rater agreement for positive hope was *ICC* = .75 and *ICC* = .64 for negative hope.

Unproductive processing. Unproductive processing captures the extent someone

approaches a problem and tries to explore and understand it but gets stuck in repetitive thoughts, analyzing without significant insight. Affective arousal without insight or a perspective shift, with rumination, worry, or perseveration would fall under this category. An example of a coded 3 *unproductive processing* was, "I spent most of the week worrying. I can't stop thinking about how I could get hurt again." The inter-rater agreement for unproductive processing was *ICC* = .57.

Avoidance. Avoidance captures the difficulty facing disturbing emotions, thoughts, or circumstances. It may involve attempts to block or move away from the uncomfortable thoughts or situations, such as discontinuing therapeutic tasks. An example of a coded 3 *avoidance* was, "I was feeling anxious, so I didn't leave the house all week." The inter-rater agreement for avoidance was *ICC* = .83.

Self. Self captures the individual's self-concept and sense of worth, desirability, competence, and identity. An example of a coded 3 *positive self* was, "I feel so proud of myself. For the first time in years I was able to go to the mall on my own. I feel like a completely new woman!" An example of a coded 3 *negative self* was, "I didn't do any of my homework. It just confirmed for me that I am weak and damaged forever as a result of my rape." The inter-rater agreement for positive self was *ICC* = .60 and *ICC* = .71 for negative self.

Therapist support. Therapist support captures interventions designed to enhance the patient's readiness for change or to stabilize new patterns after change occurs. It can include providing a sense of safety, trust, respect, and security; augmenting the patient's strengths, self-esteem, coping resources, and social support; and providing a sense of hope. Low scores are given if the therapist provides some support, but the therapeutic environment is somewhat problematic. Medium scores are given if the therapist provides adequate support, following the

treatment protocol. High scores are given if the therapist goes over and beyond the minimum requirement of the treatment protocol, and it may occur when there is a difficulty in the therapeutic environment. The inter-rater agreement for avoidance was *ICC* = .66.

Coders. Coding was conducted at both the University of Washington and Case Western Reserve University. Across sites, the coders were volunteers, undergraduate, and doctoral-level coders. Coders were trained to criterion with practice coding tapes for approximately 10 hours. Utilizing the adapted CHANGE manual and didactic methods, coders were trained to criterion (*ICC* ≥ .80 and within one point on a 4-point rating scale on all categories) prior to coding actual therapy recordings. Two coders rated each session. Coders were paired with each other an equal number of times in order to reduce rater drift or idiosyncratic ratings of pairs. In order to reduce rater bias of knowing the therapists are or treatment modality, coders only rated sessions conducted at the other site. Weekly meetings with the coding team and biweekly cross-site meetings were held to discuss coding discrepancies and prevent rater drift. When rating scores between two coders were off by 2 or more, the coding team reviewed the tape and came to an agreement on a consensus score. For this study, ratings were averaged, and the averaged ratings or consensus scores when derived were used in the analyses. Coders were blind to the presence or absence of sudden gains and blind to the treatment modality for all target sessions. Once a year, tapes were submitted to Adele Hayes, Ph.D. (creator of CHANGE; Hayes et al., 2006) for criterion checks and coding feedback.

Procedure

Individuals were sequentially selected from the ongoing NIMH-funded clinical trial conducted at the University of Washington and Case Western Reserve University. Potential study patients were recruited through: referrals from practices; advertisements in newspapers;

advertisements on buses; flyers in community centers, churches, grocery stores, libraries; campus message board; and advertisements on the tv and the radio. Potential study patients were initially screened for eligibility through a semi-structured telephone interview and then were invited for an intake evaluation. Potential patients were provided informed consent and were assessed on the SCID-IV, PSS-I, and QIDS-C by a trained independent evaluator (IE), who was blind eventually to treatment condition and therapist, which was reviewed by the clinical team. If eligible for the study, individuals were randomized to treatment condition and pre-treatment self-report measures were completed: PSS-SR, EPQ-R, DTS, and PTCI. Potential patients randomized to the PE + sertraline treatment condition received a physical exam and completed a laboratory panel. Patients began 10 weeks of active treatment of PE or PE + SER. Therapy sessions were digitally recorded for later coding. Throughout treatment, patients completed weekly self-report measures of PTSD symptoms (PSS-SR, PTCI). Once treatment was completed, patients were again assessed post-treatment on PTSD (PSS-I) and depression (QIDS-C) symptoms. Individuals who exhibited sudden gains and those who did not were identified. Then, the pre-gain session for individuals who reported sudden gains and randomly matched control sessions were coded. A total of 60 treatment sessions were coded.

General Data Reduction

Measures were screened for missing data prior to data analyses. Missing data was tested for randomness by utilizing dummy variable coding. A means test was conducted looking for differences between cases missing data and cases without on main demographic and pre-treatment measures. There were no differences in missing data. There were a total of six missing cases on the pre-treatment EPQ-R measure, and they were excluded from analyses. Measures were examined for outliers using the Mahalanobis distance procedure. Measures were

screened for normality, linearity, and homoscedasticity. No data transformations were necessary.

PE and PE + sertraline were then compared to assess for differences in demographics and pre-treatment symptom severity. As shown in Table 1, those in the PE + sertraline group were of older age compared to those in the PE only. The mean time since the target trauma was also greater in the PE + sertraline compared to those in the PE only. There were no differences in the other demographic variables, pre-treatment PTSD (PSS-I scores, and pre-treatment depression (QIDS-C) scores between the two treatment conditions. Due to the differences, treatment group, age, and years since target trauma were examined as possible covariates in the analyses involving treatment modality.

Specific Hypotheses and Analyses Plan

Sudden gains' relationship to treatment outcome (Hypothesis 1). In order to examine whether sudden gains are positively associated with better treatment outcome, a hierarchical regression was conducted with the dichotomous variable of sudden gains (yes/no) as a predictor and post-treatment PTSD symptom severity (PSS-I) as the dependent variable, while controlling for pre-treatment symptom severity (PSS-I).

CHANGE coding variables in sudden gains versus no sudden gains (Hypothesis 2). Based on Hayes et al.'s (2005, 2007) studies, it was predicted that more cognitive-emotional processing and positive hope and less unproductive processing and avoidance would be associated with sudden gains. To test this, one-tailed independent samples *t*-tests were conducted. Individuals' means scores of cognitive-emotional processing, positive hope, unproductive processing, and avoidance for pre-sudden gain sessions and matched control sessions for those who did not experience a sudden gain were compared. Secondary CHANGE

coding variables of positive and negative self, negative hope, and therapist support were also examined. It was predicted that more positive self and therapist support would be associated with sudden gains, while less negative self and negative hope would be associated with sudden gains.

Sudden gains and CHANGE coding variables in PE versus PE + sertraline (Hypothesis 3). In order to examine whether individuals who received PE + sertraline are more likely to experience sudden gains than those only receiving PE, a chi-square test comparing the frequencies of individuals who experienced sudden gains was conducted. In order to examine whether individuals who received PE + sertraline are more likely to experience cognitive-emotional processing than those only receiving PE, a one-tailed independent samples t-tests, comparing individuals' mean scores of cognitive-emotional processing, positive hope, unproductive processing, and avoidance for pre-sudden gain sessions in PE and PE + SER were conducted. Secondary CHANGE coding variables of positive and negative self, negative hope, and therapist support were also examined. It was predicted that more positive self and therapist support would be associated with PE + sertraline, while less negative self and negative hope would be associated with PE + sertraline.

Distress tolerance and neuroticism as predictors of sudden gains (Hypothesis 4). In order to examine whether lower neuroticism and higher distress tolerance is associated with an increased likelihood of sudden gains, a logistic regression was conducted with pre-treatment distress tolerance and neuroticism as predictors and the dichotomous variable of exhibiting sudden gains (yes/no) as the dependent variable.

Fear activation, between-session distress reduction, and within-session distress reduction as predictors of sudden gains (Hypothesis 5). Based on the literature (Craske et al.,

2008), between-session distress reduction was examined to see if it predicted the occurrence of sudden gains. Fear activation and within-session distress reduction were also included based on the emotional processing theory (Foa & Kozak, 1985; 1986). Thus, in order to examine whether greater between-session distress reduction are associated with increased likelihood of sudden gains, a logistic regression was conducted with pre-sudden gain fear activation, between-session distress reduction, and within-session distress reduction scores as predictors and the dichotomous variable of exhibiting sudden gains (yes/no) as the dependent variable, while controlling for treatment condition if necessary.

Power Analyses

Power analyses were conducted using G*power 3.1.1 (Erdfelder et al., 1996). Based on Doane et al. (2010) and Kelly et al. (2009), we expected 39-52% of our sample to experience sudden gains. Across hypotheses, given the focus on large, clinically meaningful facilitators of change, analyses are powered to find medium to large effects. For sudden gains' relation to treatment outcome (Hypothesis 1), examining whether sudden gains are positively associated with better treatment outcome using two predictors (pre-treatment PSS-I, sudden gain) in a regression, we had power of .80 to detect a medium to large effect (f^2 = .35). For CHANGE coding variables in sudden gains versus no sudden gains (Hypothesis 2), examining whether individuals who exhibit sudden gains will show more cognitive-emotional processing in the pre-sudden gain session (i.e., one-tailed test) than those who do not using an independent *t*-test, we had power of .80 to detect a medium to large effect (d = .65). For sudden gains and CHANGE coding variables in PE versus PE + sertraline (Hypothesis 3), examining whether individuals who receive PE + SER will experience more sudden gains and cognitive-emotional processing (i.e., one-tailed test) than those who receive PE only using a chi-square test, we had power of .80

to detect a medium to large effect (w = .36). For the independent t-test again, we had power of .80 to detect a medium to large effect (d = .65). For distress tolerance and neuroticism as predictors of sudden gains (Hypothesis 4) and fear activation, between-session distress reduction, and within-session distress reduction as predictors of sudden gains (Hypothesis 5), examining whether lower neuroticism, high distress tolerance, and greater fear activation and distress reduction will be associated with greater sudden gains using a maximum of three predictors in a logistic regressions, we had power of .80 to detect a medium to large effect (odds ratio = 2.40).

Results

Frequency of Sudden Gains

Initially, 107 patients entered the clinical trial. Patients who had fewer than five treatment sessions were excluded. This yielded 74 patients to be assessed for sudden gains following the three criteria. First, the gains had to be large in absolute terms (Criterion 1). Reductions of at least seven points on the PSS-SR were observed in 133 of 666 (20.0%) between session intervals. Second, the gains had to represent at least a 25% reduction (Criterion 2). After evaluating the magnitude of symptom reductions of the pre-gain PSS-SR severity score, 109 of 133 (82.0%) sudden gains remained. Third, the mean symptom level in the three pre-gain sessions had to significantly be higher than the mean level in the three post-gain sessions (Criterion 3). Two-sample independent t tests were conducted to examine whether the sudden gains were large relative to symptom fluctuations of pre- and post-gain sessions, and 59 of 109 (54.1%) sudden gains remained. Finally, after omitting reversals of sudden gains when patients reported symptoms of at least 50% of the original sudden gain symptom improvement, 48 of 59 (81.4%) sudden gains remained. Nine patients were excluded because all of their sudden gains were reversed. A total of 48 sudden gains were exhibited by 36 of 74 (48.6%) patients. Eleven

patients experienced multiple sudden gains. Of these, 10 patients exhibited two gains, and one patient exhibited three gains during treatment. Five patients were excluded because they exhibited sudden gains only at session 2 or session 3, because they did not have any pre-gain sessions with processing of the imaginal exposure available for coding. One patient was excluded because the pre-gain session was not digitally recorded. This yielded a total of 30 patients who exhibited a total of 40 sudden gains. If an individual had more than one sudden gain, only the pre-gain session of the first sudden gain was coded. These pre-gain sessions were compared to randomly matched sessions of 30 patients who did not exhibit sudden gains. A diagram of sudden gain and no sudden gain patient selection can be seen in Figure 3.

For those who experienced a sudden gain, the modal session of sudden gain occurrence was session 7, and the average magnitude of the gains was $M = 10.80$, $SD = 4.29$. Table 2 displays comparisons on demographic variables and pre-treatment symptom severity between patients who experienced sudden gains and those who did not exhibit sudden gains. As can be seen, there were no differences on demographic variables and pre-treatment symptom severity. Table 3 displays correlations among sudden gains and the demographic variables. There were no strong associations between sudden gains and demographic variables.

Sudden Gains' Relationship to Treatment Outcome

Zero order correlations between sudden gains and treatment outcome measures in PTSD can be seen in Table 4. As can be seen, the experience of sudden gains was associated with less post-treatment PTSD symptom severity. Hierarchical regression analysis tested whether the experience of sudden gains was associated with better treatment outcome in PTSD and depression symptoms. Pre-treatment PTSD symptom severity (PSS-I) was entered in step 1, and the experience of sudden gains (0 = *no sudden gains*, 1 = *yes sudden gains*) was entered in step

2. After step 1, with pre-treatment PSS-I in the equation, $R^2 = .18$, $F_{inc}(1, 52) = 11.74$, $p = .001$. After step 2, with the experience of sudden gains added to the prediction of pre-treatment symptom severity, $R^2 = .48$, $F(2, 51) = 23.76$, $p < .001$. Specifically, the occurrence of sudden gains was associated with lower post-treatment PTSD severity ($\beta = -.55$, $t = -5.42$, $p < .001$) over and above pre-treatment symptom severity. The adjusted R^2 value of .48 indicated that about 48.2% of the variability of post-treatment PTSD symptom severity was predicted by the experience of sudden gains.

Zero order correlations between sudden gains and treatment outcome measures in depression can be seen in Table 4, which shows that the experience of sudden gains was associated with less post-treatment depression symptom severity. A hierarchical regression analysis was conducted to examine whether the experience of sudden gains in PTSD symptoms was associated with better secondary treatment outcome in depression symptoms (QIDS-C). In step 1, pre-treatment depression symptom severity (QIDS-C) was entered, and the experience of sudden gains (0 = *no sudden gains*, 1 = *yes sudden gains*) was entered in step 2. After step 1, with pre-treatment depression (QIDS-C) in the equation, $R^2 = .11$, $F_{inc}(1, 52) = 6.22$, $p = .016$. After step 2, with the experience of sudden gains added to the prediction of pre-treatment symptom severity, $R^2 = .27$, $F(2, 51) = 9.60$, $p < .001$. The adjusted R^2 value of .25 indicated that the occurrence of sudden gains accounted for 24.5% of the total reduction in depression symptoms. Sudden gains in PTSD symptoms were associated with lower post-treatment depression severity as well ($\beta = -.42$, $t = -3.42$, $p = .001$) over and above pre-treatment symptom severity, suggesting that the experience of sudden gains in PTSD symptoms accounted for significant portions of improvement in other areas as well.

CHANGE Coding Variables Associated with Sudden Gains

CHANGE coding variables in sudden gains versus no sudden gains. The pre-gain sessions immediately before the sudden gains and matched control sessions were compared for differences in cognitive, affective, and behavioral changes. Table 5 presents correlations among demographic variables, pre-treatment symptom severity, and the CHANGE coding variables. None of the coded CHANGE variables were significantly associated with demographic variables or pre-treatment symptom severity. Independent *t*-tests were conducted to see whether the CHANGE coding variables of cognitive-emotional processing, hope, unproductive processing, avoidance, self, and therapist support varied between patients who experienced sudden gains and those who did not (0 = *no sudden gains*, 1 = *yes sudden gains*). It was expected that those who exhibited sudden gains would have higher cognitive-emotional process, positive hope, positive self, and therapist support and lower unproductive process, avoidance, negative hope, and negative self than those who did not have sudden gains. As seen in Table 6, as predicted, patients who experienced sudden gains had more cognitive-emotional processing (d = .42) than those who did not have sudden gains in the matched pre-gain sessions. Also, patients who experienced sudden gains expressed more positive hope (d = .46) than those who did not have sudden gains in the matched pre-gain sessions. For all other CHANGE coding variables, there were no differences between patients who exhibited sudden gains and those who did not. As predicted, the occurrence of sudden gains was associated with more cognitive-emotional processing and greater positive hope in the processing section of imaginal exposure in the session before the sudden gain occurred.

Negative trauma-related beliefs in sudden gains versus no sudden gains. Conceptually cognitive-emotional processing and positive hope would be capturing changes in trauma-related beliefs. Thus, as an exploratory analysis, to see whether these changes were also

being reflected in self-report measures, independent *t*-tests were conducted to see whether self-reported negative trauma-related beliefs in self, world, and blame assessed at the beginning of the pre-gain sessions measured by the PTCI varied between patients who experienced sudden gains and those who did not (0 = *no sudden gains*, 1 = *yes sudden gains*). As seen in Table 7, there were no differences in pre-gain negative trauma-related beliefs between patients who had sudden gains and those who did not. This finding suggests that the sudden gains are not necessarily associated with just general changes in beliefs prior to the therapy session, but rather the changes that are localized and occurring in the processing section of therapy.

Correlations between the CHANGE coding variables and the pre-gain trauma-related beliefs were examined using a Bonferroni-Holm step down test, and there were no significant associations. Table 8 presents correlations among the CHANGE coding variables and the pre-gain trauma-related beliefs.

Sudden Gains in PE Versus PE + Sertraline

Thirty-five patients received PE only and 25 patients received PE + sertraline treatment. It was expected that more patients in the combined treatment of PE + sertraline would experience sudden gains than those in PE only. As seen in Table 9, contrary to the prediction, similar frequencies of patients experiencing sudden gains occurred in PE only (42.9%) and PE + sertraline (60.0%). The number of patients who experienced multiple sudden gains in PE only was similar to that in PE + sertraline. There was also no difference in the number of total sudden gains that occurred by treatment condition. Patients receiving PE only experienced a total of 21 out of 315 (6.7%) possible sudden gains. Patients receiving PE + sertraline experienced a total of 19 out of 225 (8.4%) possible sudden gains. The average magnitude of the sudden gains as well the magnitude of the gains as a percentage of total improvement were similar for patients in

PE only and those in PE + sertraline. The frequency of sudden gains that were reversed was similar for both treatment conditions. Overall, the occurrence, frequency, and magnitude of sudden gains in PE only and PE + sertraline did not differ.

CHANGE Coding Variables Associated with Treatment Modality

Independent *t*-tests were conducted to see whether the CHANGE coding variables of self, hope, avoidance, cognitive-emotional processing, unproductive processing, and therapist support varied between patients who received PE only and those who received PE + sertraline (0 = *PE only*, 1 = *PE + sertraline*). It was expected that patients in the combined treatment of PE + sertraline would have cognitive-emotional processing, positive hope, positive self, and therapist support and less unproductive processing, avoidance, negative hope, and negative self than those in PE only. As seen in Table 10, in contrast to the hypothesis, patients who received PE only had more cognitive-emotional processing (d = .49) than those who received PE + sertraline across matched pre-gain sessions. There were no differences in the other coding variables between patients who received PE only and those who received PE + sertraline. Comparing the CHANGE variables between PE only and PE + sertraline in only those patients who experienced sudden gains yielded similar results. Patients in PE only had more cognitive-emotional processing than those in PE + sertraline, and there were no differences in the other coding variables. When controlling for age and years since trauma, the findings held.

Distress Tolerance and Neuroticism as Predictors of Sudden Gains

Zero order correlations among sudden gains, distress tolerance (DTS) and its subscales (tolerance, absorption, appraisal, and regulation) and neuroticism (EPQ-R) can be seen in Table 11. As can be seen, to address multicollinearity issues among the predictors, only one DTS subscale was selected. Absorption was selected because it had the strongest association with

sudden gains and one of the lower associations with neuroticism. It was expected that patients who scored higher on the absorption subscale and lower on neuroticism would be more likely to experience a sudden gain.

A logistic regression analysis was conducted on the occurrence of sudden gains for the patients as the outcome (0 = *no sudden* gains, 1 = *yes sudden gains*) and two predictors: pre-treatment absorption and pre-treatment neuroticism. A test with the full model with the two predictors against a constant-only model was at a trend level, $\chi^2(2, N = 57) = 5.04$, $p = .08$, indicating that the predictors, as a set, somewhat distinguished between patients who experienced sudden gains and those who did not exhibit sudden gains. The addition of the treatment group as a covariate did not change results.

According to the Wald criterion, only absorption reliably predicted whether a patient experienced sudden gains or not, ($B = -.71$, $Wald = 4.31$, $p = .04$, $OR = .49$), suggesting that patients who scored higher on absorption (less attention being absorbed by negative emotions) are less likely to experience sudden gains. Table 12 shows regression coefficients, Wald statistics, odds ratios, and 95% confidence intervals for odds ratios for the three predictors. Figure 5 shows the observed relationship between absorption and the probability of experiencing a sudden gain, showing that as scores on the absorption subscale increases (less attention being absorbed by negative emotions), the probability of experiencing a sudden gain decreases.

Behavioral In-Session Markers Predicting Sudden Gains

Fear activation, between-session, and within-session distress reduction as predictors of sudden gains. Table 13 displays the correlations among sudden gains, fear activation, between-session distress reduction, and within-session distress reduction. As can be seen, there were no strong associations with the behavioral in-session markers and the occurrence of sudden

gains. It was expected that patients who experienced greater fear activation, between-session and within-session distress reduction would be more likely to experience a sudden gain. A logistic regression analysis was conducted on the occurrence of sudden gains for the patients as the outcome (0 = *no sudden gains*, 1 = *yes sudden gains*) and three predictors: fear activation, between-session distress reduction (N-1, N), and within-session distress reduction. A test with the full model with the two predictors against a constant-only model was not significant, $\chi^2(3, N = 60) = 3.76$, $p = .29$, indicating that the predictors, as a set, did not distinguish between patients who experienced sudden gains and those who did not exhibit sudden gains. Table 14 shows regression coefficients, Wald statistics, odds ratios, and 95% confidence intervals for odds ratios for the three predictors. No individual predictors were significant. The addition of the treatment condition as a covariate did not change results.

Fear activation, proximal between-session, and within-session distress reduction as predictors of sudden gains. Direct logistic regression analysis was conducted with fear activation, between-session distress reduction more proximal to the sudden gains, and within-session distress reduction as the predictor variables and the presence of sudden gains as the outcome variable. A test of the full model with the three predictors against a constant-only model was also not significant, $\chi^2(3, N = 60) = 4.50$, $p = .21$, indicating that the predictors, as a set, did not distinguish between patients who experienced sudden gains and those who did not exhibit sudden gains. Table 15 shows regression coefficients, Wald statistics, odds ratios, and 95% confidence intervals for odds ratios for the three predictors. Controlling for treatment modality did not change results. Overall, behavioral in-session markers of fear activation and distress reduction during the imaginal exposure did not predict who would or would not experience sudden gains.

Associations between fear activation, between-session, and within-session distress reduction and CHANGE. Table 16 presents the correlations between the CHANGE coding variables and fear activation, between-session distress reduction, and within-session distress reduction. As can be seen, greater within-session distress reduction was associated with more cognitive-emotional processing. Thus, the more distress reduction a patient experienced during the imaginal exposure, more cognitive-emotional processing occurred in the processing section that followed.

Discussion

Sudden gains, defined as rapid, large symptom improvements during between-session intervals, have been investigated across different samples and interventions and have consistently been associated with better treatment outcomes (for a review see Aderka, Nickerson, Bøe, & Hofmann, 2012). Indeed, these discontinuous, non-linear patterns of change can mark critical points in treatment where it is most likely to reveal potential factors causally related to the processes of change (Hayes et al., 2007b). This was the first study to take a process-oriented approach to systematically examine in-session therapy content for potential mechanisms underlying the occurrence of sudden gains in PTSD treatment. Specifically, in-session therapist-patient interactions prior to the gains were examined using a process-oriented, detailed coding system. When examining the pre-gain sessions, patients who experienced sudden gains expressed more hope and had more cognitive-emotional processing than those who did not exhibit sudden gains, suggesting positive hope and cognitive-emotional processing are key elements for discontinuous change. These are the first findings of potential predictors and processes of change associated with sudden gains in PTSD treatment, and perhaps these elements can be enhanced to promote further therapeutic improvement in patients. Sudden gains occurred

similarly in both PE only and PE combined with sertraline treatment. However, when examining pre-gain in-session content, patients receiving PE only had more cognitive-emotional processing than patients receiving PE combined with sertraline, highlighting potential differential mechanisms between the two treatments. Absorption was the only predictor of sudden gains, suggesting that individuals who pay more attention to negative emotional states and thoughts are more likely to experience a sudden gain, possibly due to more emotional engagement in treatment. All together, the findings of this study bring a better understanding of the sudden gain phenomenon and elucidate what are the key elements of change in PTSD treatment.

Sudden Gains Associated with Better Treatment Outcome

As predicted, the experience of sudden gains was associated with better treatment outcome at termination, constituting for 48.2% of the total reduction in PTSD symptoms. The sudden gains were also associated with better outcome on secondary measures of depression symptoms, accounting for 24.5% of total symptom reduction. Our findings are consistent with the idea that sudden gains are not transient and inconsequential fluctuations. Instead, they are significant and stable changes that contribute to substantial symptom improvement (e.g., Aderka et al., 2012). Applying the dynamic systems theory (Thelen 1995; Thelen & Smith, 1994) to psychotherapy, critical fluctuations can occur when new interventions and elements are introduced to challenge and destabilize old system patterns in an individual (Hayes et al., 2007b). Thus, sudden gains could mark critical fluctuation points that can guide researchers to the important segments of treatment for further examination that are likely to reveal the crucial elements that mobilize and inhibit change, rather than examining all treatment sessions or randomly selected sessions without considering symptom trajectories of change (Hayes et al., 2007b).

Prior In-Session Therapy Content as Predictors of Sudden Gains

The lack of pre-treatment predictors of sudden gains in depression and PTSD treatment (e.g., Aderka et al., 2011; Doane et al., 2010; Gaynor et al., 2003; Hardy et al., 2005; Jun et al., 2013; Kelly et al., 2009) points to the need of examining more proximal factors to the sudden gains. The few studies that did examine in-session content of pre-gain sessions (e.g., Tang & DeRubeis, 1999; Tang et al., 2005) were more promising than studies that examined changes in weekly self-reports that were completed at the beginning of each treatment session (e.g., Hofmann, Schulz, Meuret, Moscovitch, & Suvak, 2006; Kelly et al., 2005), emphasizing the importance of the measurement interval and its proximity to the sudden gain. This was the first study to examine more proximal in-session factors and change processes that might be associated with the occurrence of sudden gains in PTSD treatment. Patient and therapist content in the discussion after imaginal exposure to the trauma memory of matched pre-gain sessions revealed that patients who experienced sudden gains expressed more cognitive-emotional processing and more positive hope than those who did not exhibit sudden gains. This is the first finding of potential predictors and processes of change eliciting sudden gains in PTSD treatment. These findings are generally consistent to that of Hayes and colleagues (2007a) who examined the discontinuous change patterns of early, rapid responses and depression spikes in depression treatment and coded narratives about the patients' depression with the CHANGE coding system. They found that the narratives prior to the symptom change of early, rapid responders to the treatment were coded as having more positive hope than narratives of nonrapid responders. However, they found the narratives of individuals exhibiting a depression spike (temporary worsening of symptoms that is related to better treatment outcome) had more cognitive-emotional processing than those without a spike during the spike sessions. Thus, greater

cognitive-emotional processing and positive hope seem to be consistent elements that when introduced to an individual's dynamic system, perturbation and critical fluctuations of discontinuous change occurs.

Cognitive-emotional processing. Cognitive-emotional processing also occurred at a higher level in therapist-patient interactions after imaginal exposure for patients who experienced rapid, large PTSD symptom improvements by the next session than those who did not. This is generally consistent with other studies in cognitive therapy and CBT for depression that found sudden gains were immediately preceded by substantial in-session cognitive changes (e.g., Tang & DeRubeis, 1999; Tang et al., 2005). Other studies utilizing the CHANGE coding system have consistently found peak levels of cognitive-emotional processing were strong predictors of good treatment outcomes (Hayes et al., 2005, 2007a). This suggests that cognitive-emotional processing is a possible key mechanism of change that is eliciting sudden gains. In fact, in PE for PTSD, reductions in negative PTSD-related cognitions led to subsequent improvement in PTSD, as well as improvements in depression symptoms (Zalta et al., 2014). Following the emotional processing theory (Foa & Kozak, 1985, 1986), cognitive-emotional processing is thought to have occurred by activating the pathological fear structure and integrating corrective information that is inconsistent with the fear structure containing erroneous trauma-related beliefs. Thus, the goal would be to have affective engagement and arousal, cognitive analysis, and meaning making. It is important to note that cognitive-emotional processing is a dynamic category that captures both the cognitive and affective change. It was designed to capture insights ranging from fleeting, superficial realizations to more in-depth, substantial understandings, which are distinct nuances that cognitive-change self-report measures would not detect. Notably, there were no differences in pre-gain negative trauma-related beliefs (PTCI)

between patients who experienced a sudden gain compared to those who did not, and pre-gain negative beliefs were not associated with cognitive-emotional processing. This suggests that the sudden gains and cognitive-emotional processing in this study is not composed of just general changes in trauma-related beliefs prior to the gain, but instead are driven by dynamic changes localized to the processing section of therapy. One factor that may differentiate an individual from having cognitive-emotional processing versus not is psychological flexibility, as a certain willingness and openness needs to be present in order to start exploring and questioning difficult experiences and emotions (Ciarrochi, Bilich, & Godsell, 2010). Moderate levels of flexibility must be present within a patient's dynamic system in order to be open to adapt change (e.g., Hollenstein, Lichtwarck-Aschoff, & Potworowski, 2013; Lunkenheimer, Olson, Hollenstein, Sameroff, & Winter, 2011). A dynamic system that is too rigid is characterized by patterns that perseverate and repeat over time and are insensitive to perturbations and the incorporation of new information (Hayes, Yasinksi, & Barnes, 2014a).

In order to better understand the concept of cognitive-emotional processing that is associated with sudden gains, examining actual coded content may help to understand how it is being elicited. The following are examples of cognitive-emotional processing statements from this study: "The imaginal is better. I could remember more details. I realize I'm not avoiding as much" (coded as low cognitive-emotional processing); "The part where I was really chocked up… I remember my sister snuck back in. I love her. She's amazing. I realized she was supportive. I did have support" (coded as medium cognitive-emotional processing); "I realize I'm afraid of being happy because I'm afraid of rejection. I tend to only focus on the negative things in life. I'm afraid to show happiness" (coded as high cognitive-emotional processing). The range of content that cognitive-emotional processing captures is wide. It can start from

fleeting, superficial realizations of changes that occurred with the imaginal exposure. It can also include significant realizations and shifts in perspectives about the trauma memory. Finally, it can be more global and consist of a better understanding of oneself and their patterns of behavior. Thus, a patient can gather insights from various domains in treatment. However, the substantial and in-depth processing seem to surround direct trauma-related beliefs and understanding of one's current and past behaviors, where the insights are often accompanied by emotions and may really serve to drive the symptom change. The current PE manual's guidelines (Foa et al., 2007) of how to facilitate processing and insight are general and brief: normalize and help patients understand their reactions and behaviors in the trauma and its aftermath, ask open-ended questions about the patient's thoughts and feelings about the imaginal exposure experience, and begin to focus discussion on negative, unhelpful, inaccurate, or unrealistic beliefs that the patient holds that may be contributing to the maintenance of PTSD. What is lacking is further information of how to elicit insight when the patient presents with rigidity and perseveration of unhelpful beliefs and how to maintain insights and perspective shifts that are made. Since cognitive-emotional processing is a dynamic process that captures both cognitive and emotional change, if a patient is being resistant to exploring and questioning a problem area, a therapist can assess for what is reinforcing the rigidity. For example, it is possible that the patients are avoiding the negative emotions associated with the belief. In this case, cognitive-emotional processing could occur in a step process, engaging and exploring the difficult emotion first and then the content. A therapist should recognize that it may take several sessions and practice on the same content for patients to exhibit substantial cognitive-emotional processing. Once they do, it would be important for the therapist to help the patients consolidate the new learning, by helping patients summarize at the end of the session what the take away

themes or points are. This will help patients to organize their thoughts and rehearse the new perspective shifts. Therapists should be encouraged to revisit these beliefs and emotions in the next session to see if the insights were maintained.

Positive hope. Positive hope occurred at a higher level in therapist-patient interactions after imaginal exposure for those who experienced a rapid drop in PTSD symptoms by the next session than those who did not. Having an optimistic view of the future, motivation for change, and the belief that one can affect the change are important components of mental health (Ryff & Singer, 1998) and may facilitate further positive experiences (Fredrickson, 2001). Positive hope may facilitate change through increasing treatment buy-in and motivation to do treatment homework, which in turn may lead to the experience of sudden gains. In this study, positive hope and sudden gains were not strongly associated with pre-treatment levels of PTSD or depression. This suggests that the patients who experienced more positive hope were not simply those who were less depressed at the beginning of treatment. One aspect that may differentiate an individual from expressing positive hope versus not is having an attentional bias toward negative stimuli that is seen in PTSD (e.g., Cisler et al., 2011; Constans, 2005). Focusing one's attention more on negative information, such as distressing emotions and thoughts, prolongs negative mood states and heightens stress reactivity (e.g., Tran, Seimer, & Joorman, 2011). Patients will have more difficulty disengaging from the negative information, preventing them from paying attention to new positive information and changes, such as remembering that recalling the trauma memory is getting easier and less distressing. This would make it difficult for patients to engage with and benefit from positive emotions and thoughts. Notably, there were no differences in the opposite direction with negative hope, as one may expect the two coding variables to measure the inverse of the same construct. This is consistent with other studies that

found the positive and negative valence variables of the CHANGE coding system moved independently from each other (e.g., Hayes et al., 2005; 2007a). In fact, positive psychology emphasizes the importance of treatments not only focusing on reducing symptoms and negative emotion regulation but also on promoting health and positive emotion regulation (Carl, Soskin, Kerns, & Barlow, 2013). Thus, although related, positive and negative hope are distinct constructs and should be targeted separately in treatment, by not only focusing on reducing hopelessness but also actively building hopeful thoughts (Snyder, Rand, Sigman, 2002).

In order to better understand the concept of positive hope that predicts sudden gains, examining actual coded content may help to understand how it is being elicited. The following are examples of positive hope statements from this study: "It's a lot better. The memory is not as traumatic, and it's easier to tell the story" (coded as low hope); "The imaginal is better, but a bit boring. It makes me feel like I'm moving in a positive direction" (coded as medium hope); and "It was better. I am getting a lot better. I'm getting better on crowded buses. I can actually enjoy life now" (coded as high hope). The majority of the content of positive hope was around noticing positive improvements related to the experience of doing imaginal exposure. Thus, this category has an overlap with cognitive-emotional processing. Many of the patients commented on imaginal exposure getting easier and the recounting of the trauma memory no longer evoking as strong or distressing emotions for them. At times, the hope statement was more general to their overall functioning, commenting more globally on lifestyle changes on what they are no longer avoiding. Thus, positive hope captured the recognition of making progress in problem areas and the belief that they are capable of making changes, which then often led to optimistic statements about the future being better. This highlights the importance for patients to regularly evaluate themselves and their progress in treatment on a meta-level, taking note of the positive

changes, which often times may be obscure to the patient if their attention is centered on negative content, such as problematic areas that are still distressing to them where they see no change. As the PE manual (Foa et al., 2007) currently stands, there is no mention of positive hope, but it includes general, brief guidelines of how to facilitate the processing of imaginal exposure, through which hope could be elicited: provide positive feedback and acknowledgement of the patient's courage, try to stimulate thinking with open-ended questions, and comment on distress reduction within or across sessions. Therapists should enhance these techniques with the goal of eliciting positive hope in mind. Specifically, when positive changes have occurred, therapists can promote hope by explicitly asking patients how they brought on the changes and what impact the changes are having on them. Connecting changes to the patient's behavior will encourage the belief that they have the agency and are capable of executing the means of attaining goals. Therapists can also encourage generalization by asking patients how they can achieve similar changes in other problem areas to achieve goals, which will help build motivation and the commitment for change. When the patient has no hope or is unable to recognize positive changes, the therapist should help guide the patient's attention through open ended questions to assess the differences and progress made during treatment. When no overt changes have occurred for the patient, the therapist should keep in mind that hope can be achieved through various domains. Instead, the therapist can help the patient to recognize their ability to tolerate and engage with the imaginal exposure and treatment in general, and how this in itself reflects the patient's commitment and determination to make changes for themselves, emphasizing that change takes time. Thus, by identifying progress made, connecting it to the patient's behavior, generalizing the technique of change to other goals may help bolster positive hope for the patient.

Dynamic systems theory. The process of discontinuous change and the interactions of different key elements can be represented by the dynamic systems theory (Thelen, 1995; Thelen & Smith, 1994). Within the framework of dynamic systems theory, an individual's dynamic system consists of elements that interact and continually evolve over time. The dynamic systems can be viewed as consisting of hypothetical networks of interrelated cognitive, affective, behavioral, and physiological components (Hayes et al., 2014). In this study, prolonged exposure and sertraline interventions would be the vehicles to cause perturbations in an individual's dynamic system, providing the environment for new elements to be introduced into the system. Extending this theory to the present findings, in particular, cognitive-emotional processing and positive hope may be key elements that must be introduced into one's dynamic network system. When the amount or strength of cognitive-emotional processing and positive hope exceeds a certain threshold of accommodation and the system can no longer easily assimilate these new elements, a critical fluctuation of discontinuous change of a sudden gain occurs. During this critical fluctuation and destabilization, the system is open to new information and exploration of more adaptive associations and configurations among its hypothetical networks. Thus, changes may occur in the individual's cognitions (e.g., further changes in trauma-related beliefs), affect (e.g., decrease in depressive symptoms), and behavior (e.g., increased engagement in *in vivo* and imaginal exposure) in order to accommodate the new cognitive-emotional processing and positive hope elements. This in itself may be illustrative of the "upward spiral" that Tang and DeRubeis (1999) described, where a sudden gain triggers a positive feedback loop where cognitive changes can spark further reductions in symptoms.

It is interesting that there were no significant differences with the other coding variables in individuals experiencing sudden gains versus not. It could be that they take on secondary

roles to the changes in symptoms compared to cognitive-emotional processing and positive hope. This is not to say that cognitive-emotional processing and positive hope are the only critical elements associated with sudden gains. There could be other third variables that play a role, such as homework completion, patient's readiness for change, and life events. It is possible that once a patient's level of positive hope increases, this may in turn increase motivation and commitment to change and thus allow more engagement in *in-vivo* and imaginal homework exercises. Also, the amount of readiness for change and flexibility that a patient's dynamic network system has would determine whether the system would be perturbed and incorporate new information. Thus, it is important to understand that processes that not only facilitate change but also maintains rigidity and inhibit change. Finally, general emotion-laden life events may act as vehicles to cause perturbations in a patient's dynamic system, which can further facilitate the mechanisms of the intervention. Related, although the present study looked at the various coding variables independently, these therapist-patient interaction factors may actually be moving and working together in a network. Indeed, Hayes and colleagues (2014b) illustrated a quantitative method of examining the CHANGE coding variables as positive and negative networks of cognition, emotion, and behavior. They were able to depict the destabilization or dispersion, defined as the spread of positive and negative activation scores, of the networks and differential activation patterns throughout a treatment for depression. More cognitive-emotional processing during the exposure phase of a depression treatment was associated with greater dispersion of the dynamic system networks, and the positive and negative networks moved and became activated independently of each other. Thus, this is further evidence that cognitive-emotional processing is a key element to be introduced into a perturbed dynamic system.

Treatment Modality and Sudden Gains

Overall, the patterns of occurrence of sudden gains in PE only and PE combined with sertraline were similar. This is consistent with the two studies that found similar frequencies of patients exhibiting sudden gains in psychotherapy and pharmacotherapy (Jun et al., 2013; Vittengl et al., 2005). Notably, Jun and colleagues found 42.2% of patients receiving PE and 31.0% of those receiving sertraline alone experienced sudden gains. In this study, 42.9% of patients in PE alone and 60.0% of those in PE combined with sertraline exhibited rapid symptom improvements ($V = .17$). However, benchmarking across studies, the rate of sudden gain occurrence when PE was combined with sertraline is greater than that of sertraline alone. In addition, Jun and colleagues found a greater reversal rate of sudden gains that occurred in the sertraline only group. In contrast, PE only and PE combined with sertraline had similar frequencies of sudden gain reversals. This suggests that there may be some additive benefits of the combined treatment of psychotherapy with SSRIs, where patients are better able to maintain the rapid improvements with the addition of PE than if they were on sertraline alone. This may be due to the additive components of differential mechanisms of PE and sertraline.

Jun et al.'s (2013) and Vittengl et al.'s (2005) studies concluded that the occurrence of sudden gains in pharmacotherapy either challenges the notion that cognitive change is the predominant mechanism underlying rapid symptom improvement (Tang & DeRubeis, 1999) or suggests that cognitive insight is a shared process by both psychotherapy and pharmacotherapy. This study was the first to further examine possible change processes underlying sudden gains in psychotherapy and augmented treatment with psychotropic medication, in order to shed light to this question of similar or different mechanisms of change. When comparing the different mechanisms in PE only and PE combined with sertraline, the only difference that emerged was in cognitive-emotional processing. In contrast to the direction of our original hypothesis, coding

of the matched pre-gain sessions revealed that patients receiving PE only had more cognitive-emotional processing than patients receiving PE and sertraline. This finding was held after controlling for the age of the patients and years since trauma differences between the two treatment groups. It is possible that this might be indicative of the divergent therapeutic mechanisms for PE only and augmented treatment with SSRIs. For PE only, cognitive-emotional processing may be a key element to introduce to an individual's dynamic network system, in order to create destabilization and a cascade effect to generate critical fluctuations in symptoms. However, for PE combined with sertraline, cognitive-emotional processing may not be as central of an element for change. In fact, the results of two studies are consistent with this idea of the non-centrality of cognitive shifts for PTSD improvement with psychotropic medications. Cooper and colleagues (Cooper, Zoellner, Mavissakalian, & Feeny, 2014) examined whether cognitive changes preceded PTSD symptom change, moderated by treatment group of PE only or sertraline only. They found that although cognitive shifts predicted symptom change for both groups, the effect was large for those receiving PE alone but was small for individuals receiving sertraline alone, suggesting cognitive insight was not the primary mechanism of change for those receiving sertraline. Similarly, in a study comparing treatment effects of a modified PE plus placebo versus PE plus methylene blue (an atypical monamine oxidase inhibitor) for PTSD, changes in negative cognitive beliefs mediated changes in PTSD symptoms for individuals in PE plus placebo but not for those receiving PE plus methylene blue (Zoellner et al., 2014). It is possible that when an antidepressant is introduced, the chemical effects onto the neurobiological system and its resulting change might be more global in the domains of emotion regulation and learning processes.

Studies have proposed various pathways of change for antidepressants. Heller and colleagues (Heller, Johnstone, & Peterson, 2013) examined changes in neural circuits involved in emotion regulation in patients receiving antidepressant treatment for major depressive disorder. They found that patients who exhibited the greatest reductions in depression symptoms also showed the most rapid increases in prefrontal cortex engagement when regulating negative affect during an emotion regulation task. Other studies have also found evidence of antidepressants increasing amygdaloid-hippocampal and frontal activity during emotion processing tasks (Outhred et al., 2013). It may be for patients receiving PE combined with sertraline, the addition of the antidepressant is helping with better regulation of negative emotions. The changes in negative emotions are new elements that are introduced into the patient's dynamic network system, altering the hypothetical affective and physiological components (e.g., Hayes et al., 2014b). This contributes to the critical fluctuation of discontinuous change of a sudden gain. Thus, patients receiving PE combined with sertraline may not need similar amounts of cognitive-emotional processing in order to exceed the threshold of accommodation of the system as patients receiving only PE do. Another pathway of change for antidepressants is that they may reverse neuronal deficits by increasing neurogenesis, specifically increasing the volume of the hippocampus in PTSD (Thomaes et al., 2013); however, other studies do not support this (Hanson, Owens, & Nemeroff, 2011). The hippocampus, which is centrally involved in the processing of declarative memory, may have a role in the formation of contextual memories, which has been shown to have an inverse relationship with involuntary memories (Bisby, King, Brewin, Burgess, & Curran, 2010). The addition of sertraline to PE, may aid in decreasing the amount of intrusive trauma-related memories the patient experiences, which in effect would decrease the amount of distress related to the intrusions. These changes would be new elements

added to the cognitive and affective components of the patient's dynamic network system. Similarly, these new elements would contribute to the critical fluctuation of discontinuous change, thus, not necessitating the same amount of cognitive-emotional processing to exceed the accommodation threshold of the system as that in PE only. Thus, PE and sertraline could have similar yet divergent mechanisms of change. The addition of an antidepressant may add different, new elements to the patient's dynamic network system on top of what PE already introduces, decreasing the amount of cognitive-emotional processing needed to initiate a critical fluctuation of a sudden gain compared to treatment with PE only.

Distress Tolerance and Neuroticism as Predictors of Sudden Gains

Although pre-treatment predictors of sudden gains, across the literature, have been weak at best (e.g., Aderka et al., 2011; Doane et al., 2010; Jun et al., 2013; Kelly et al., 2009), this study examined the association of possible pre-treatment trait-like factors of distress tolerance and neuroticism with sudden gains, which no prior study has done. It was found that patients who had greater absorption, their attention was being absorbed more by negative emotions, were more likely to experience a sudden gain. Notably, the distress tolerance scales were highly associated with one another; and accordingly, this more likely reflects a broader distress tolerance construct than absorption alone. The treatment of PTSD is based on the emotional processing theory (Foa & Kozak, 1985, 1986) that suggests successful exposure treatment requires activation of the pathological fear structure and integration of corrective information that is incompatible with this structure. Indeed, therapists conducting PE encourage emotional engagement with the trauma memory in order to effectively process both the memory and associated emotions (Hembree, Rauch, & Foa, 2003). Thus, it is possible that patients who are paying more attention to the distressing emotions are exhibiting greater emotional engagement

during treatment, including processing of the imaginal exposure. This would promote perturbation of the patient's dynamic network system, facilitating the environment for new elements to be introduced. Another possibility for this finding may be that distress tolerance is not necessarily a measure of trait but rather the patient's maladapative beliefs about the experience of distress (Zvolensky, Vujanovic, Bernstein, & Leyro, 2010). Thus, distress tolerance levels may be influenced by symptom severity and also may change with treatment. In this case, greater changes in beliefs about the experience of negative emotional and other aversive states may be reflected in greater positive hope and cognitive-emotional processing leading to sudden gains.

Trait neuroticism was not strongly associated with the occurrence of sudden gains. Neuroticism is thought to reflect negative affectivity that is a stable and general trait dimension and can predispose an individual to a broad range of negative moods, cognitions and self-appraisal (Robichaud & Dugas, 2005). One study that looked at the associations between trait neuroticism and treatment response in PTSD (van Emmerik et al., 2011) found that trait neuroticism was neither related to treatment dropout or poor treatment response. One possibility for the lack of findings with neuroticism and PTSD treatment response is a restricted range of neuroticism level in the sample. If the majority of patients were high on neuroticism, the lack of variability would obscure any association with sudden gains. However, for this study, there seemed to have been an adequate range in neuroticism based on normative data. Another possibility is that pre-treatment neuroticism is a predictor that is too distal in time from the occurrence of sudden gains. This would be consistent with the lack of findings of other pre-treatment predictors (e.g., Aderka et al., 2011; Jun et al., 2013). Thus, this further supports that

patients are not necessarily predisposed to the experience of sudden gains, and it is what the patient is experiencing within treatment that is eliciting the discontinuous changes.

Fear Activation, Between-Session, and Within-Session Distress Reduction As Predictors of Sudden Gains

Fear activation, between-session distress reduction, and within-session distress reduction are all important components to emotional processing theory (Foa & Kozak, 1985, 1986). They are also more proximal elements to the occurrence of sudden gains. Thus, it was expected that out of the potential predictors being examined, these would most likely predict the experience of sudden gains. Interestingly, neither fear activation nor distress reduction during imaginal exposure in the pre-gain session was associated with later sudden gains. This is consistent with the growing literature that initial fear activation and within-session distress reduction are not necessary for better treatment outcome in the general anxiety literature (for a review see Craske et al., 2008). The finding that between-session distress reduction did not predict sudden gains is also consistent with other studies that found patients improved with treatment even though between-session distress did not decline across sessions in the general anxiety literature (e.g., Bluett et al., 2013; Tsao & Craske, 2000). Notably, greater within-session distress reduction during imaginal exposure was associated with more cognitive-emotional processing immediately after the imaginal exposure. It is possible that the reduction of distress in-session facilitated the occurrence of cognitive-emotional processing by helping the patient stay engaged during the processing of the imaginal exposure to explore and question various problem areas. It is also possible that this association is just reflective of greater insights surrounding the actual experience of imaginal exposure and recognition that the distress decreased. Overall, these findings bring to question how central these elements are to the emotional processing theory.

Experiencing distress reduction may be one avenue of gaining cognitive-emotional processing. However, it is not the only path to better treatment outcome. Thus, therapists should not put too much emphasis on these concepts, knowing that patients can still experience corrective learning without the peak fear activation and distress reduction.

Limitations and Future Directions

There were a few limitations to this study. The focus on coding of the processing of imaginal exposure may have restricted the examination of sudden gains. The earliest pre-gain session that could be coded was session 3, when imaginal exposure begins. This in turn meant that the earliest sudden gain occurrence that could be examined was session 4. Thus, earlier sudden gains occurring at sessions 2 and 3 were not investigated, and it is possible they could be qualitatively different from those happening later in treatment (e.g., Busch et al., 2006; Clerkin, Teachman, & Smith-Janik, 2008). However, sessions 4-10 consist of the majority of the treatment, and this study was able to systematically compare and examine possible predictors of the majority of sudden gains. The study analyses were planned to detect medium to large effect sizes. Thus, some of the analyses may not have been powered enough to detect findings of small effect sizes that could be present in the data. Keep in mind, though, that the overall goal was to utilize sudden gains to guide the research to important segments of the treatment in order to detect the critical elements to symptom improvement. Although interesting to find the small effect differences, small effects may point to auxiliary elements that do not hold as much prominence in discontinuous symptom change. Some of the CHANGE coding variables had reliabilities in the fair range, which may have increased variability and affected our effect sizes. Future studies will have to work on increasing the reliability among the coders. Coders of this study went through extensive training to criteria, coding practice tapes and approximately 10

hours of reliability tapes. Perhaps increasing the training time for coders and doing more frequent reliability checks will help improve the inter-rater agreements. However, the consistency among raters for cognitive-emotional processing was similar to that for the coding system utilized by Tang and DeRubeis (1999) as well as Hayes and colleagues (2005). Previous studies examining in-session content predictors of sudden gains were mainly within-subject designs (e.g., Tang & DeRubeis, 1999; Tang et al., 2005; Kelly et al., 2005), while the present study was a between-subjects design. Thus, the smaller effect sizes of the study's findings may be due to more variability factors between patients (e.g., demographics, beliefs, treatment condition, and therapist). However, the between-subjects design allows for a more stringent test, making the findings more meaningful and generalizable, allowing for the examination of possible individual differences associated with sudden gains. It is also worth noting that the effect sizes may have been attenuated due to the majority of the sample improving from the treatment, whether or not they experienced sudden gains. However, this is the nature of process-oriented research. It was already known that PE and PE combined with sertraline are both effective treatments. The goal was to gain a better understanding of how the treatments work in order to find the core elements of change that can be maximized to improve the treatments and make them more efficient and effective.

To date, the results across many studies examining predictors of sudden gains suggested that pre-treatment individual differences do not predict sudden gains, highlighting the need to examine temporally closer to the occurrence of sudden gains. This is the first study to date to examine in-session content to examine what precedes the occurrence of sudden gains in PTSD treatment. This study not only compared possible in-session factors and change processes between those who experienced sudden gains versus not, but also compared the occurrence of

sudden gains and proximal predictors comparing those in PE alone and those in PE augmented with sertraline. This study sheds light into the "black box" of treatment, revealing that cognitive-emotional processing and positive hope may be critical elements and change processes for rapid, large symptom improvement. Thus, clinicians should enhance their therapeutic techniques with the goal of promoting both of these elements. Specifically, therapists should identify the agents reinforcing a patient's rigidity to question and explore problem areas, such avoidance of associated negative emotions that could inhibit cognitive-emotional processing. Further, therapists should help patients practice consolidating new learning at the end of every session through summarization and repetition of the content. In order to bolster positive hope, therapist should help patients identify changes and progress made, connecting the changes to the patient's behavior in order to encourage the patient's belief of agency and capability of executing the means of attaining goals. Therapists should encourage further change by helping the patient generalize their behavior to other problem areas and goals.

Further research should investigate whether cognitive-emotional processing and positive hope are driven by the patient's behavior, therapist's behavior or both. Part of this may require a study design that incorporates a direct manipulation factor to elicit cognitive-emotional processing, positive hope, or the maintenance and upward spiral of the changes. Potential manipulation factors may be direct versus indirect therapeutic techniques, such as techniques to help the patient consolidate newly learned information, provided to the patient and homework assignments, such as writing out the content of cognitive-emotional processing to help strengthen learning. Future studies should also look at the patterns of positive and negative network systems in PTSD treatment. The coding variables were examined independently in this study and may only be showing part of the picture. To get a better understanding of how the dynamic

systems theory of change is occurring in the patients, examining the dispersions of networks may bring further insight to individual differences of change and also differential mechanisms of change in psychotherapy and pharmacotherapy. In addition, future studies should investigate factors causing patients' dynamic network systems to maintain rigidity and inhibit change and to what extent and type of intervention perturbation is needed to initiate change.

By understanding what the key elements are to trigger substantial change and by identifying the treatment factors to elicit these elements, therapists can promote long-term psychological change. By monitoring cognitive-emotional processing and positive hope, therapists will be able to readily modify the session content to enhance therapeutic gains. Thus, understanding the key processes of change will help to better tailor the intervention for specific patients based on their needs and strengths. This study demonstrates that the processing of the imaginal exposure section can substantially affect the course of treatment for some patients in a meaningful way. Thus, identifying patient and therapist factors associated with sudden gains will allow us to pinpoint the critical sessions and session components. This will provide treatment development targets of where to further refine therapy, potentially increasing the efficiency and effectiveness of the treatment.

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Table 1

Comparisons on demographic variables between PE and PE + Sertraline groups

Variable	PE *M* or % (*SD*)	PE + Sertraline *M* or % (*SD*)	*t*-test or χ^2	Cohen's *d* or Cramer's *V*	*p*
Age	33.14 (*10.28*)	41.84 (*13.57*)	-2.83*	.74	.006
Gender			.14	.05	.71
Female	46.7	31.7			
Male	11.7	10.0			
Ethnicity			.21	.06	.65
Caucasian	38.3	25.0			
Minority	20.0	16.7			
Education			.85	.12	.36
Not College Educated	35.0	20.0			
4 Year or More College Degree	23.3	21.7			
Income			.12	.05	.73
Less than $20,000 Per Year	28.3	18.3			
More than $20,000 Per Year	30.0	23.3			
Target Trauma			2.74	.05	.73
Adult Sexual Assault	16.7	15.0			
Adult Non-Sexual Assault	20.0	8.3			
Childhood Sexual Assault	6.7	8.3			
Childhood Non-Sexual Assault	3.3	3.3			
Motor Vehicle Accident	3.3	3.3			
Other[a]	8.3	3.3			
Years Since Target Trauma	9.14 (*10.59*)	18.24 (*15.51*)	-2.70*	.71	.009
Pre-Treatment PTSD (PSS-I)	30.26 (*4.22*)	31.20 (*4.49*)	-.83	.22	.41
Pre-Treatment Depression (QIDS-C)	14.20 (*4.49*)	15.24 (*4.83*)	-.86	.23	.40

Note. * $p < .05$; [a](death or violence to a loved one, medical complications).

Table 2

Comparisons on demographic variables between sudden gains and no sudden gain groups

Variable	Sudden Gain *M* or % (*SD*)	No Sudden Gain *M* or % (*SD*)	*t*-test or χ^2	Cohen's *d* or Cramer's *V*	*p*
Age	37.07 (*13.45*)	36.47 (*11.54*)	-.19	.05	.85
Gender			.10	.04	.75
Female	38.3	40.0			
Male	11.7	10.0			
Ethnicity			1.15	.14	.28
Caucasian	35.0	28.3			
Minority	15.0	21.7			
Education			.61	.10	.44
Not College Educated	30.0	25.0			
4 Year or More College Degree	20.0	25.0			
Income			1.07	.13	.30
Less than $20,000 Per Year	20.0	26.7			
More than $20,000 Per Year	30.0	23.3			
Target Trauma			2.20	.19	.82
Adult Sexual Assault	18.3	13.3			
Adult Non-Sexual Assault	10.0	18.3			
Childhood Sexual Assault	8.3	6.7			
Childhood Non-Sexual Assault	3.3	3.3			
Motor Vehicle Accident	3.3	3.3			
Other[a]	6.7	5.0			
Years Since Target Trauma	12.17 (*13.67*)	13.70 (*13.57*)	.44	.11	.66
Pre-Treatment PTSD (PSS-I)	31.03 (*3.89*)	30.27 (*4.76*)	-.68	.18	.50
Pre-Treatment Depression (QIDS-C)	15.27 (*4.61*)	14.00 (*4.63*)	-1.06	.28	.29

Note. [a](death or violence to a loved one, medical complications).

Table 3

Correlations between sudden gains and demographic variables

	1	2	3	4	5	6	7
1. Sudden Gains[a]	--						
2. Age	.02	--					
3. Gender[b]	-.04	-.30*	--				
4. Ethnicity[c]	.14	-.10	-.10	--			
5. Education[d]	.10	-.03	-.15	-.20	--		
6. Income[e]	-.13	.22	-.08	-.19	.31*	--	
7. Trauma[f]	-.13	.08	-.17	-.16	.23	-.06	--
8. Years Since Target Trauma	-.06	.39*	-.08	.12	-.12	-.003	.05

Note. * $p < .05$; [a](0 = *No*, 1 = *Yes*); [b](0 = *Male*, 1 = *Female*) [c](0 = *Minority*, 1 = *Caucasian*); [d](0 = *4 year or more college degree*, 1 = *Not college educated*); [e](0 = *More than $20,000 per year*, 1 = *Less than $20,000 per year*); [f](0 = *Sexual assault*, 1 = *Non-sexual assault*).

Table 4

Correlations between sudden gains and treatment outcome measures

	1	2	3	4
1. Sudden Gains[a]	--			
2. Pre-Treatment PSS-I	.09	--		
3. Post-Treatment PSS-I	-.49*	.43*	--	
4. Pre-Treatment QIDS-C	.14	.27*	.04	--
5. Post-Treatment QIDS-C	-.34*	.42*	.71*	.33*

Note. * $p < .05$; [a] (0 = *No*, 1 = *Yes*). PSS-I = Posttraumatic Symptom Scale – Interview; QIDS-C = Quick Inventory of Depressive Symptomatology – Clinician Rating.

Table 5

Correlations among demographic variables, pre-treatment symptom severity, and CHANGE variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. Age	--															
2. Gender[a]	-.23*	--														
3. Ethnicity[b]	-.10	.10	--													
4. Education[c]	-.03	-.15	-.20	--												
5. Income[d]	.22	-.08	-.19	.31*	--											
6. Trauma[e]	.08	-.17	-.16	.23	-.06	--										
7. Years Since Trauma	.39*	-.08	.12	-.12	-.003	-.22	--									
8. Pre-PTSD (PSS-I)	.01	-.15	-.06	.25	.23	.03	.08	--								
9. Pre-Depression (QIDS-C)	.10	-.25	-.05	.26*	.04	-.02	-.02	.27*	--							
10. Cognitive-Emotional Processing (CHANGE)	.09	.16	.01	-.16	-.14	.06	-.06	-.002	-.13	--						
11. Positive Hope (CHANGE)	.11	.08	.01	.03	-.07	-.14	.02	-.04	.17	.31*	--					
12. Negative Hope (CHANGE)	-.13	-.02	.08	.06	.17	-.11	.13	-.07	.20	-.15	-.13	--				
13. Unproductive Processing (CHANGE)	.04	-.13	-.24	.19	.12	.14	-.15	.09	.21	-.20	-.16	.23	--			
14. Avoidance (CHANGE)	.13	-.13	-.21	.17	.23	.09	-.12	.15	.10	-.18	-.08	.21	.54*	--		
15. Positive Self (CHANGE)	.19	-.01	-.09	-.05	-.05	-.09	.24	.07	.05	.19	.25	.03	-.22	-.12	--	

16. Negative Self (CHANGE)	.02	.12	.06	-.06	-.06	-.22	.06	-.02	.20	.13	.07	.44*	.30*	.08	.14	--
17. Therapist Support (CHANGE)	-.08	.11	.05	.20	-.09	-.01	-.02	.06	.06	-.16	-.16	.17	.30*	.20	-.18	.20

Note. * $p < .05$; [a](0 = *Male*, 1 = *Female*); [b](0 = *Minority*, 1 = *Caucasian*); [c](0 = *4 year or more college degree*, 1 = *Not college educated*); [d](0 = *More than $20,000 per year*, 1 = *Less than $20,000 per year*); [e](1 = *Sexual assault*, 2 = *Non-sexual assault*). PSS-I = Posttraumatic Symptom Scale – Interview; QIDS-C = Quick Inventory of Depressive Symptomatology – Clinician Rating; CHANGE = Change and Growth Experiences Scale.

Table 6

Comparisons of CHANGE variables in patients with no sudden gain and those with sudden gain

Variable	No Sudden Gain *M* (*SD*)	Sudden Gain *M* (*SD*)	*t*-test	*p*	Cohen's *d*
Cognitive-Emotional Processing	1.37 (*.71*)	1.65 (*.63*)	-1.64*	.05	.42
Positive Hope	.57 (*.54*)	.85 (*.68*)	-1.78*	.04	.46
Negative Hope	.65 (*.68*)	.47 (*.43*)	1.24	.11	.32
Unproductive Processing	.58 (*.64*)	.65 (*.66*)	-.34	.35	.11
Avoidance	.45 (*.72*)	.38 (*.55*)	.40	.35	.11
Positive Self	.38 (*.49*)	.45 (*.51*)	-.52	.30	.14
Negative Self	.78 (*.78*)	.78 (*.77*)	.00	.50	.00
Therapist Support	2.05 (*.20*)	2.02 (*.09*)	.83	.41	.19

Note. * = $p < .05$.

Table 7

Comparisons of pre-gain negative trauma-related beliefs (PTCI) in patients with no sudden gain and those with sudden gain

Variable	No Sudden Gain *M* (*SD*)	Sudden Gain *M* (*SD*)	*t*-test	*p*	Cohen's *d*
Self	3.39 (*1.54*)	3.40 (*1.25*)	-.02	.98	.01
World	5.12 (*1.34*)	5.05 (*1.36*)	.19	.85	.05
Blame	3.13 (*1.20*)	3.64 (*1.90*)	-1.00	.32	.32
Total Score	122.67 (*44.72*)	124.88 (*39.25*)	-.20	.84	.05

Note. * = $p < .05$.

Table 8

Correlations among CHANGE variables and pre-gain negative trauma-related beliefs

	1	2	3	4	5	6	7	8	9	10	11
1. Positive Self (CHANGE)	--										
2. Negative Self (CHANGE)	.14	--									
3. Positive Hope (CHANGE)	.25	.07	--								
4. Negative Hope (CHANGE)	.03	.44	-.13	--							
5. Avoidance (CHANGE)	-.12	.08	-.08	.21	--						
6. Cognitive-Emotional Processing (CHANGE)	.19	.13	.31	-.15	-.18	--					
7. Unproductive Processing (CHANGE)	-.22	.30	-.16	.23	.54	-.20	--				
8. Therapist Support (CHANGE)	-.18	.20	-.16	.17	.20	-.16	.30	--			
9. Self (PTCI)	-.27	.22	-.10	.27*	.21	-.30	.34	.34	--		
10. World (PTCI)	-.07	.05	-.07	.06	.29	.01	.22	.25	.52	--	
11. Blame (PTCI)	-.27	.20	.03	.01	.04	-.15	.22	.16	.63	.34	--
12. Total score (PTCI)	-.27	.21	-.08	.20	.22	-.24	.34	.34	.96	.67	.75*

Note. * $p < .05$; CHANGE = Change and Growth Experiences Scale; PTCI = Posttraumatic Cognitions Inventory.

Table 9

Comparisons of sudden gains in PE and PE + sertraline

Variable	PE *M* or % (*SD*)	PE + Sertraline *M* or % (*SD*)	*t*-test or χ^2	*p*	Cohen's *d* or Cramer's *V*
Sudden Gains	42.9	60.0	1.71	.19	.17
Multiple Sudden Gains	14.3	16.0	.03	.86	.02
Median Session of Occurrence (4-10)	6	7			
Modal Session of Occurrence (4-10)	6	7			
Magnitude of Gains	11.53 (*5.14*)	10.07 (*3.24*)	.94	.36	.36
Sudden Gains as Percent of Total Improvement	52.1	42.1	1.16	.25	.52
Sudden Gains Reversed	11.4	16.0	.26	.61	.07

Note. * = $p < .05$.

Table 10

Comparisons of CHANGE variables in PE and PE + sertraline

Variable	PE *M* (*SD*)	PE + Sertraline *M* (*SD*)	*t*-test	*p*	Cohen's *d*
Cognitive-Emotional Processing	1.64 (*.74*)	1.32 (*.54*)	1.85*	.03	.49
Positive Hope	.64 (*.58*)	.80 (*.69*)	-.96	.17	.25
Negative Hope	.63 (*.68*)	.46 (*.38*)	1.23	.11	.31
Unproductive Processing	.66 (*.68*)	.56 (*.60*)	.57	.29	.16
Avoidance	.43 (*.74*)	.40 (*.48*)	.17	.43	.05
Positive Self	.34 (*.45*)	.52 (*.55*)	-1.37	.09	.36
Negative Self	.77 (*.75*)	.80 (*.82*)	-.14	.44	.04
Therapist Support	2.06 (*.20*)	2.00 (*.00*)	1.41	.08	.42

Note. * = $p < .05$.

Table 11

Correlations between sudden gains, distress tolerance, and neuroticism

	1	2	3	4	5	6
1. Sudden Gains[a]	--					
2. Tolerance (DTS)	.03	--				
3. Absorption (DTS)	-.20	.82*	--			
4. Appraisal (DTS)	-.20	.68*	.73*	--		
5. Regulation (DTS)	-.02	.61*	.54*	.60*	--	
6. Total (DTS)	-.11	.91*	.90*	.86*	.79*	--
7. Neuroticism (EPQ-R)	.05	-.57*	-.58*	-.67*	-.46*	-.65*

Note. * $p < .05$; [a] (0 = *No*, 1 = *Yes*); DTS = Distress Tolerance Scale; EPQ-R = Eysenck Personality Questionnaire Revised.

Table 12

Direct logistic regression analyses of absorption and neuroticism predicting occurrence of sudden gains

					95% Confidence Interval for Odds Ratio	
Variables	***B***	***SE B***	**Wald χ^2-test**	**Odds Ratio**	*Lower*	*Upper*
Absorption (DTS)	-.71	.34	4.31*	.49	.25	.96
Neuroticism (EPQ-R)	-.12	.08	2.37	.89	.77	1.03
(Constant)	3.79	2.00	3.58			

Note. * $p < .05$. $R^2 = .09$ (Cox & Snell), .11 (Nagelkerke). DTS = Distress Tolerance Scale; EPQ-R = Eysenck Personality Questionnaire Revised.

Table 13

Correlations among sudden gains, between-session distress reduction, within-session distress reduction, and pre-gain peak distress

	1	2	3	4
1. Sudden Gains[a]	--			
2. Between-Session Distress Reduction (N-1 and N)	.08	--		
3. Between-Session Distress Reduction (N and N+1)	-.12	-.44*	--	
4. Within-Session Distress Reductions	.24	-.24	.07	--
5. Pre-Gain Peak Distress	.03	-.62*	.41*	.24

Note. * $p < .05$; [a] (0 = *No*, 1 = *Yes*).

Table 14

Direct logistic regression analysis of between-session distress reduction, within-session distress reduction, and pre-gain peak distress predicting occurrence of sudden gains

					95% Confidence Interval for Odds Ratio	
Variables	***B***	***SE B***	**Wald χ^2-test**	**Odds Ratio**	*Lower*	*Upper*
Between-Session Distress Reduction (N-1, N)	.02	.02	.98	1.02	.98	1.05
Within-Session Distress Reduction	.03	.02	2.45	1.03	.99	1.07
Pre-Gain Peak Distress	.01	.02	.58	1.01	.98	1.04
(Constant)	-5.58	.43	1.65			

Note. * $p < .05$. $R^2 = .07$ (Cox & Snell), .09 (Nagelkerke).

Table 15

Direct logistic regression analysis of between-session distress reduction, within-session distress reduction, and pre-gain peak distress predicting occurrence of sudden gains

					95% Confidence Interval for Odds Ratio	
Variables	***B***	***SE B***	**Wald χ^2-test**	**Odds Ratio**	*Lower*	*Upper*
Between-Session Distress Reduction (N, N+1)	-.02	.02	.64	.98	.94	1.03
Within-Session Distress Reduction	.03	.02	3.22	1.03	1.00	1.07
Pre-Gain Peak Distress	-.004	.01	.10	1.00	.97	1.02
(Constant)	-.19	.90	.05			

Note. * $p < .05$. $R^2 = .08$ (Cox & Snell), .10 (Nagelkerke).

Table 16

Correlations among distress reductions, peak distress, and CHANGE variables

	1	2	3	4	5	6	7	8	9	10	11
1. Between-Session Distress Reduction (N-1, N)	--										
2. Between-Session Distress Reduction (N, N+1)	-.44*	--									
3. Within-Session Distress Reduction	-.24	.07	--								
4. Pre-Gain Peak Distress	-.62*	.41*	.24	--							
5. Cognitive-Emotional Processing (CHANGE)	.07	.05	.29*	-.05	--						
6. Positive Hope (CHANGE)	-.04	-.24	.19	-.07	.31*	--					
7. Negative Hope (CHANGE)	-.02	-.07	-.14	.02	-.15	-.13	--				
8. Unproductive Processing (CHANGE)	.18	.18	-.01	.00	-.20	-.16	.23	--			
9. Avoidance (CHANGE)	.20	-.08	-.04	.02	-.18	-.08	.21	.54*	--		
10. Positive Self (CHANGE)	-.08	-.26	.003	-.11	.19	.25	.03	-.22	-.12	--	
11. Negative Self (CHANGE)	-.07	-.002	-.09	.01	.13	.07	.44*	.30*	.08	.14	--
12. Therapist Support (CHANGE)	-.05	.21	.05	-.16	-.16	-.16	.17	.30*	.20	-.18	.20

Note. * $p < .05$; CHANGE = Change and Growth Experiences Scale; PTCI = Posttraumatic Cognitions Inventory.

Figure 1

Sudden gain criterion 1

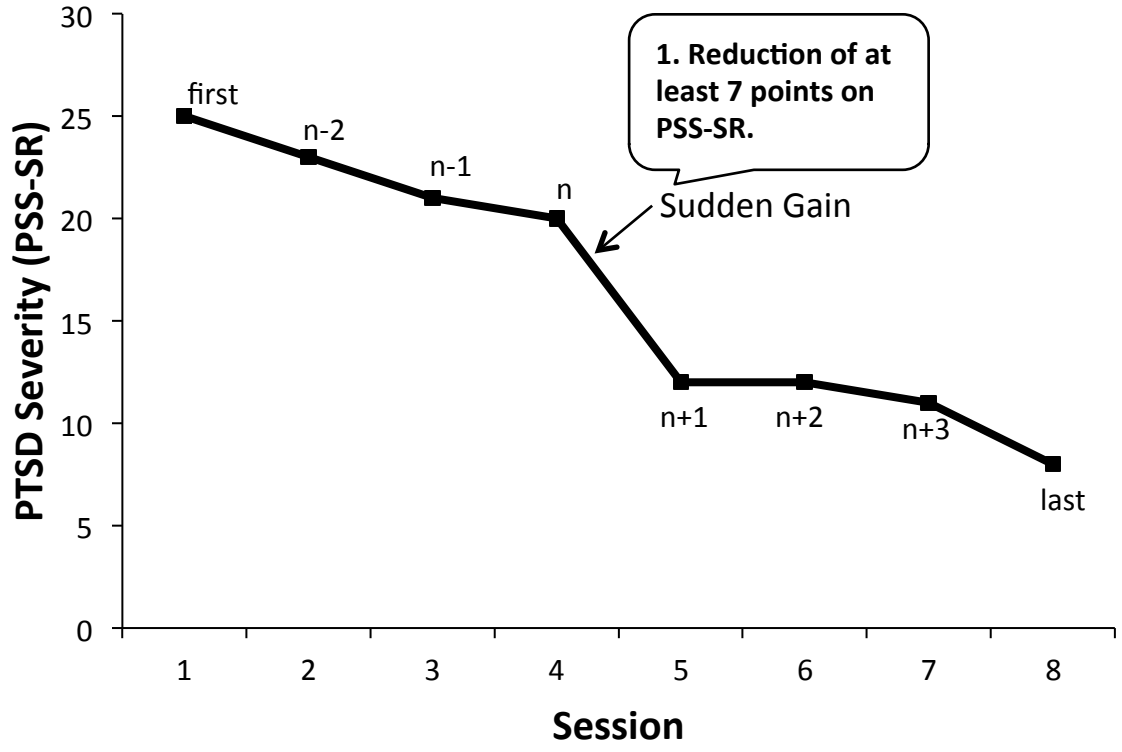

Figure 2

Sudden gain criterion 2

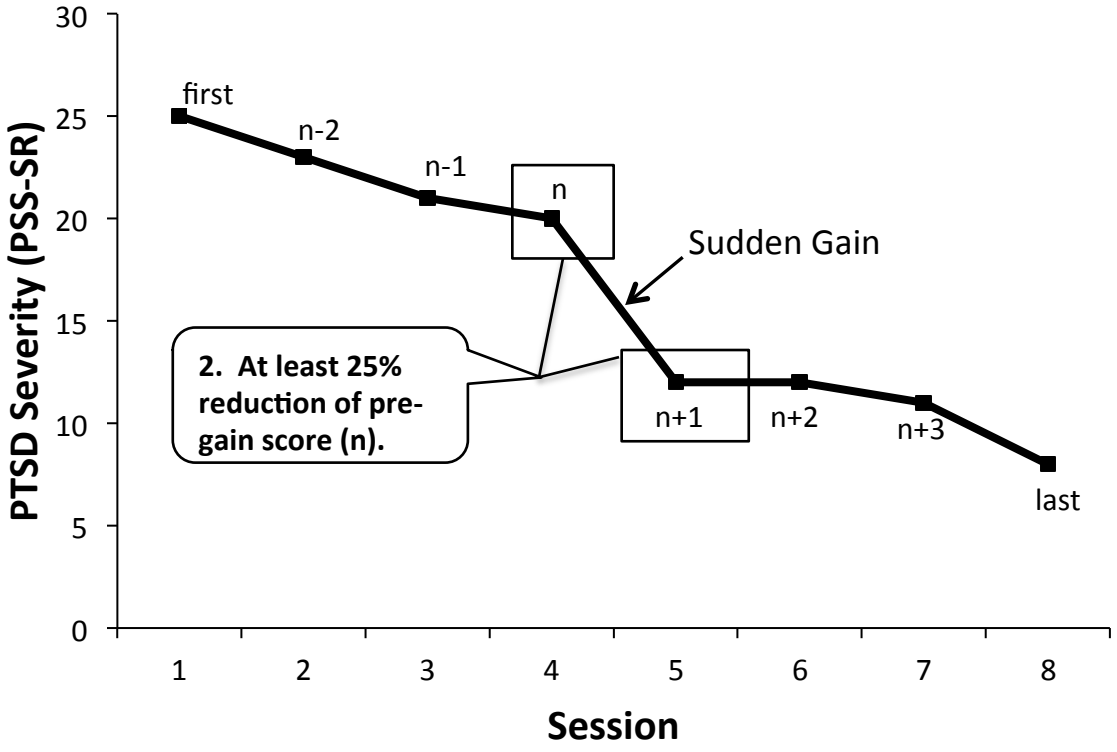

Figure 3

Sudden gain criterion 3

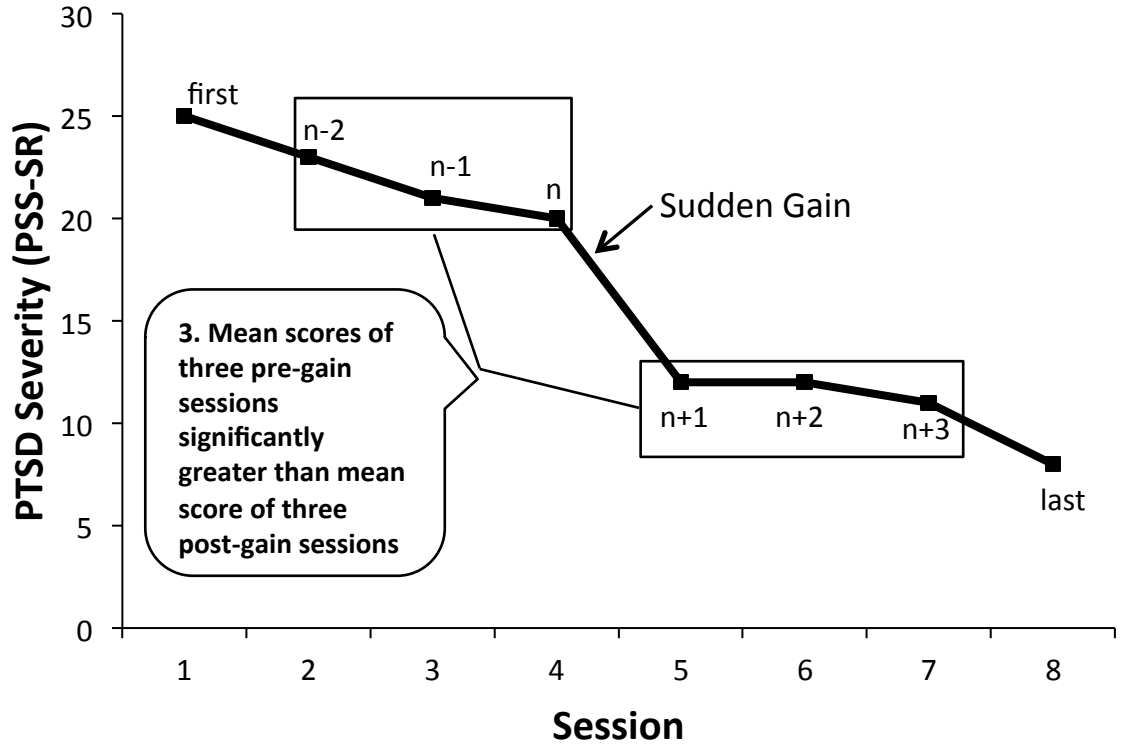

Figure 4

Information flow on sudden gain and no sudden gain patient selection

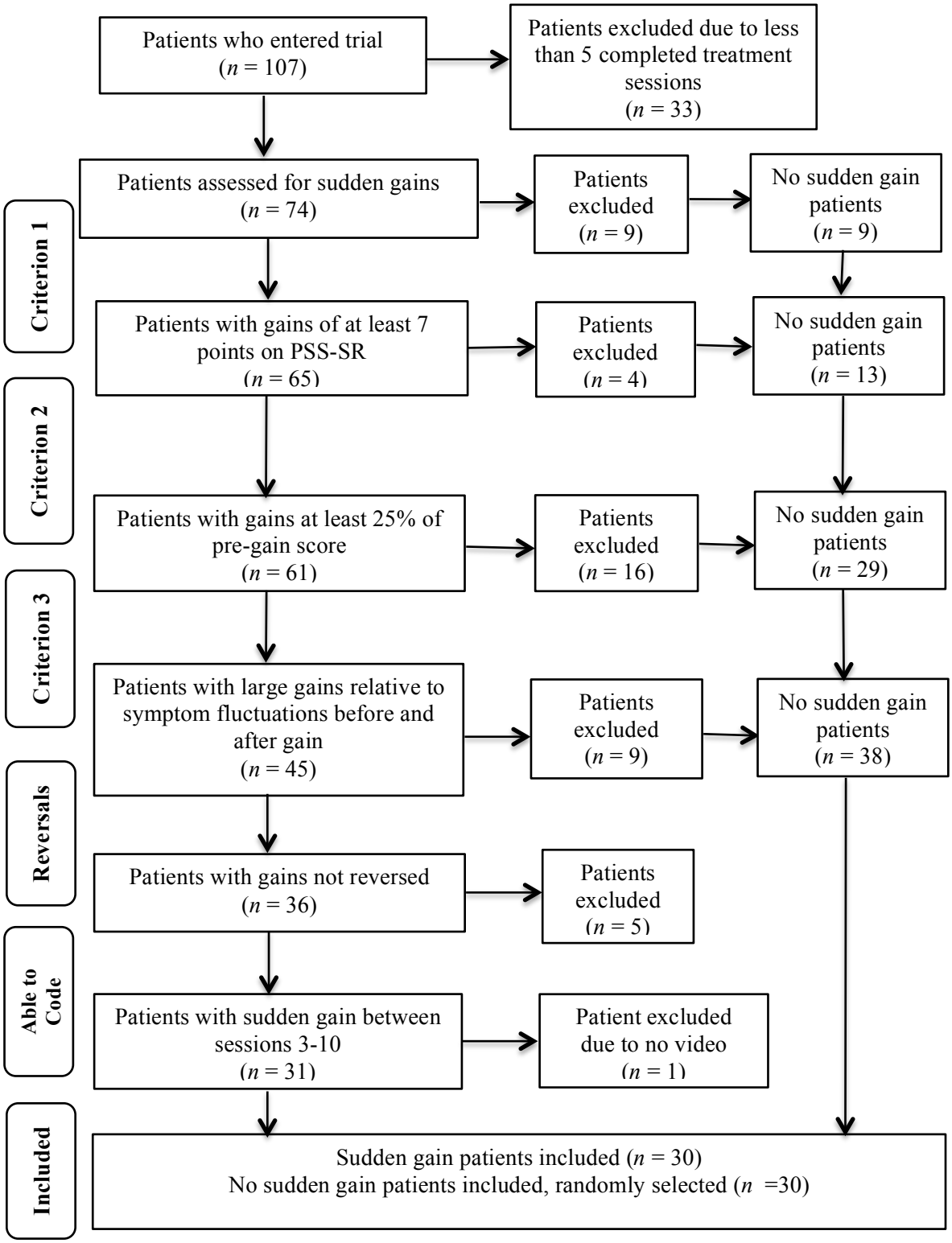

Figure 5

Probability of experiencing sudden gain by distress tolerance absorption scale