DEPRESSION SYMPTOM DISCONTINUITIES OVER THE COURSE OF TREATMENT FOR CHRONIC POSTTRAUMATIC STRESS DISORDER

by

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Submitted in partial fulfillment of the requirements

for the degree of Master of Arts

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January, 2012
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Table of Contents

Acknowledgements........................................................................................................6
Abstract..........................................................................................................................7
Introduction.....................................................................................................................8
    Study Aims................................................................................................................16
Methods.........................................................................................................................17
    Participants...............................................................................................................17
    Measures..................................................................................................................18
Overview of Treatment..................................................................................................20
Procedure.......................................................................................................................21
Results............................................................................................................................22
Discussion.......................................................................................................................30
References.......................................................................................................................43
Figures

Figure 1. Depression Symptom Discontinuities over the Course of PTSD Treatment ...............................................................37
Tables

Table 1. Means and Standard Deviations for Self-Reported PTSD Severity, Depression Severity, and Social Support at Pre-treatment

Table 2. The Association Between Pre-Treatment Social Support and Depression Symptom Discontinuities Over the Course of PTSD Treatment

Table 3. Timing and Magnitude of Depression Symptom Discontinuities Between PE and SER

Table 4. Pre-treatment Psychopathology and Sudden Gains in Depression Symptoms as Predictors of Post-treatment PTSD Severity

Table 5. Pre-treatment Psychopathology and Sudden Gains in Depression Symptoms as Predictors of Post-treatment Depression Severity
Acknowledgements

Preparation of this manuscript was supported by grants to Drs. Zoellner and Feeny from the National Institute of Mental Health (R01 MH066347, R01 MH066348). The investigative team on the grants included: Peter Roy-Byrne, MD, Matig Mavissakalian, MD, Jason Doctor, Ph.D., Joshua McDavid, MD, and Nora McNamara, MD. I also acknowledge the support of The William T. Dahms, M.D. Clinical Research Unit, funded under the Cleveland Clinical and Translational Science Award (UL1 RR024989). Thank you to Norah Feeny, Ph.D., for serving as the chair of my Master’s committee and her help and feedback on this project. Thank you to Julie Exline, Ph.D. and Amy Przeworski, Ph.D. for serving as members of my Master’s committee.
Depression Symptom Discontinuities over the Course of Treatment for Chronic Posttraumatic Stress Disorder

Abstract

by

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Changes in symptoms during depression treatment occur discontinuously and such discontinuities predict better outcome (Hayes et al., 2007a). While depression commonly co-occurs with PTSD, we know little about how change unfolds in depression symptoms during PTSD treatment. This study examined transitions in depression symptoms during PTSD treatment, namely sudden gains and depression spikes. Social support was examined as a predictor of these symptom fluctuations. At pre-treatment, 200 participants with a primary diagnosis of PTSD completed measures of PTSD and depression severity, and social support. During 10 weeks of prolonged exposure or sertraline, depression was assessed. Overall, 18% of participants experienced a depression sudden gain and 22.5% experienced a transient depression spike. Higher negative trauma-related support was associated with experiencing a depression spike. Similar rates of depression discontinuities occurred between treatments. Sudden gains predicted better treatment outcome. Clinicians should be encouraged that transient depression worsenings were not associated with poorer outcome.
Depression Symptom Discontinuities over the Course of Treatment for Chronic PTSD

Currently, effective psychological and pharmacological treatment options exist for posttraumatic stress disorder (PTSD; e.g., Foa, Keane, Friedman, & Cohen, 2008). However, not everyone with PTSD benefits from these treatments, and drop-out rates remain significant (e.g., Brady et al., 2000; Davidson et al., 2001; Foa et al., 1999; Foa et al., 2005; Foa, Rothbaum, Riggs, & Murdock, 1991). Despite the empirical support for a variety of PTSD treatments, little is known about the underlying processes of change that lead to symptom improvement. These considerations highlight the need to better understand patterns of symptom change over the course of treatment, and predictors of these patterns. Researchers have argued (e.g., Kazdin, 2007) that such process oriented research is the “next-step” in improving treatment delivery. Examining how a given treatment works, which is the question at the core of process research, involves examining when change is occurring. This information can reveal predictors of symptom change at important transition points in treatment and what might be changing at those points (Hayes, Laurenceau, Feldman, Strauss, & Cardiaciotto, 2007b). Examining these transition points can inform treatment development and provide insight to clinicians on what components of treatment are most effective. Ultimately, this may lead to modification of treatments to better match individual client needs. Overall, examining process-oriented questions, such how symptoms change over the course of treatment, can help clinicians and researchers to better understand how PTSD treatments exert their effect.

Examination of pattern of symptom change throughout treatment focuses on points of symptom worsening or substantial gain over the course of treatment. Research
to date that has focused on symptom patterns suggests that symptom discontinuities, both rapid symptom decreases (e.g., Doane, Feeny, & Zoellner, 2010; Tang & DeRubeis, 1999) and transient symptom spikes (e.g., Hayes et al., 2007a) are common during psychotherapy for both mood and anxiety disorders, and may predict better treatment outcome. Such fluctuations in symptoms may be critical in pointing both researchers’ and clinicians’ focus to the components occurring during treatment at the time these changes are occurring; thus, a causal link can be made between specific components of treatment and symptom change (Hayes et al., 2007b). What is less known however is how change occurs in pharmacotherapy.

Although both psychotherapy and pharmacotherapy appear to be effective in the treatment of chronic PTSD (e.g., Brady et al., 2000; Foa et al., 2005) little is known as to the processes underlying symptom improvement in these dissimilar treatment regimens. Prolonged exposure (PE), a first line psychotherapy for PTSD (Institute of Medicine, 2007), has been shown to be efficacious in many trials and is well tolerated (e.g., Foa et al., 2008; Foa, Zoellner, Feeny, Hembree, & Alvarez-Conrad, 2002a). In comparison to other forms of cognitive behavioral treatment, PE has consistently demonstrated equal or greater effectiveness (Foa et al., 1999; Foa et al., 2005; Resick, Nishith, Weaver, Astin, & Feuer, 2002). In particular, PE uses behavioral strategies to reduce fear (Foa, Dancu, & Hembree, 2002b). More specifically, individuals are asked to repeatedly revisit their trauma memory and approach activities and places they have been avoiding because of the trauma (e.g., driving). Often, through repeated exposures, maladaptive cognitions (e.g., blame, guilt) are altered. On the other hand, sertraline (SER), an FDA approved selective serotonin reuptake inhibitor, is also an empirically supported intervention for
PTSD (Brady et al., 2000; Davidson et al., 2001). SER involves taking a daily dosage of medication and discussing side effects with a pharmacotherapist. Although these treatments appear very different, little is known regarding how similarly or dissimilarly change unfolds between psychotherapy and pharmacotherapy. Given the demand for a high level of emotional engagement in PE, there may be more opportunities for symptom discontinuities. In addition, researchers (e.g., Tang & DeRubeis, 1999; Tang, DeRubeis, Hollon, Amsterdam, & Shelton, 2007) have suggested that sudden improvements in symptoms are associated with a shift or correction in negative attitudes and beliefs. Since cognitive work is not a major focus of pharmacotherapy, there may likely be fewer opportunities for rapid symptom improvement. However, little attention has been paid to understanding symptom trajectories and discontinuities during pharmacotherapy for PTSD. Overall, examining differences in the patterns and processes between psychotherapy and pharmacotherapy can help researchers to understand how these treatments work.

**Depression Symptom Discontinuities**

The current manuscript examined discontinuities, both transient worsenings and sudden improvements, in depression symptoms over the course of both PE and SER for chronic PTSD. Examining fluctuations in depression symptoms for PTSD clients is relevant for a number of reasons. First, comorbidity of PTSD with another Axis I or Axis II disorder is the norm rather than the exception in the aftermath of a traumatic experience (Creamer, Burgess, & McFarlane, 2001; Hankin, Spiro, Miller, & Kazis, 1999; Kessler, Sonnega, Hughes, & Nelson, 1995). Second, major depression (MDD) is one of the most commonly co-occurring conditions associated with PTSD (Breslau et al.,
1997; Creamer et al., 2001; Nixon, Resick, & Nishith, 2004; Orsillo, Weathers, Litz, Steinberg, & Keane, 1996) with a number of cross-sectional studies reporting comorbidity rates of approximately 50-80% (e.g., Hankin, Spiro, Miller, & Kazis, 1999; Shore, Vollmer, & Tatum, 1989). These high rates have been observed among various traumatized samples including victims of interpersonal violence (e.g., Nixon et al., 2004), combat (Orsillo et al., 1996; Shalev et al., 1998), and natural disasters (e.g., Green & Lindy, 1994). Third, individuals with PTSD and MDD, as compared to individuals with either PTSD or MDD alone, tend to report higher levels of symptom severity and benefit less from treatment (e.g., Green et al., 2006; Nixon et al., 2004; Post, Zoellner, Youngstrom, & Feeny, in press; Shalev et al., 1998). Fourth, examining sudden increases or decreases in depression over the course of treatment for individuals with PTSD may provide clinicians with a deeper, more broadly focused picture of client functioning. In addition, sudden spikes in depression may present potential challenges or impediments to symptom improvement in PTSD. For example, a worsening in depression may make it more difficult for clients to engage effectively or become motivated to actively participate in the PTSD treatment regimen. Finally, clinicians often have concerns continuing with exposure therapy (Becker, Zayfert, & Anderson, 2004; Foa et al., 2002a; van Minnen, Hendriks, & Olff, 2010) when clients express PTSD or depression symptom increases. In fact, Becker and colleagues (2004), examining a survey of licensed psychologists, found that 37% of respondents reported that any co-morbid diagnosis was a factor that would deter them from administering PE. Thus, examining the impact of sudden depression symptom worsenings or improvements can provide a more nuanced
portrait of client functioning and provide insight to clinicians regarding the potential impact of these symptom fluctuations.

**Depression Sudden Gains.** A growing body of research has begun to examine "sudden gains" (Tang & DeRubeis, 1999) which are rapid, large decreases in symptoms that occur in one between-session interval. The majority of sudden gains research has been conducted on psychotherapy for depression. In a study conducted by Hardy and colleagues (2005), approximately 40% of clients experienced sudden gains and the presence of these sudden gains predicted better end-of-treatment outcome; other studies have reported similar findings (Busch, Kanter, Landes, & Kohlenberg, 2006; Hopko, Robertson, & Carvalho, 2009; Kelly et al., 2005; Tang, DeRubeis, Beberman, & Pham, 2005; Tang, Luborsky, & Andrusyn, 2002; Vittengl et al., 2005). Only one study has compared the rates of sudden gains among depressed clients undergoing psychotherapy, pill placebo, or pharmacotherapy (Vittengl et al., 2005). Interestingly, findings suggest similar rates of sudden gains among all treatment conditions, with 25% of clients in pill placebo, 33% of clients in cognitive therapy, and 47% of clients in pharmacotherapy experiencing a sudden gain in depressive symptoms. Recently, Drymalski & Washburn (2011) examined the occurrence of sudden gains in a large sample of individuals receiving depression treatment in a partial hospitalization program, and again found that over 40% experienced a sudden depression gain and that such gains were related to better treatment outcome and better quality of life at post-treatment (Drymalski & Washburn, 2011). Overall, it appears that discontinuous improvements in depression symptoms appear to be related to better depression treatment outcome. In addition, recent evidence suggests that the benefits of experiencing sudden gains in depression symptoms may
affect broader levels of social functioning as well (e.g., quality of life). However, what is less known, is how discontinuities in depression symptoms affect treatment outcome when clients are being treated for an anxiety disorder, such as PTSD.

Much of the work on sudden gains has examined depression symptom changes. However, there are a few studies that have examined such gains in anxiety disorders (Clerkin, Teachman, & Smith-Janik, 2008; Doane, Feeny, & Zoellner, 2010; Hofmann et al., 2006; Kelly, Rizvi, Monson, & Resick, 2009; Present et al., 2008). Hofmann et al. (2006) examined the occurrence of sudden gains among individuals with social phobia undergoing either cognitive behavioral group or exposure group therapy. Findings suggested that 18% of clients experienced sudden gains, but these gains were not associated with a better end of treatment outcome. Two studies have examined sudden gains among those receiving CBT for PTSD, and findings were similar to those seen in the depression literature: 40-50% of clients experienced sudden gains in PTSD symptoms and those gains were associated with better PTSD outcome (Doane et al., 2010; Kelly et al., 2009). In addition, Kelly et al. (2009) found that sudden PTSD gains were also associated with lower levels of depression at post-treatment. Thus, sudden gains may be not only beneficial for treatment outcome in PTSD symptoms, but may positively impact broader levels of client functioning as well (e.g., depression). These inconsistent findings across the anxiety disorders highlight the need for continued research on the phenomenon of sudden gains among those with anxiety disorders, including PTSD.

Depression Spikes. Exposure-based therapies for anxiety disorders are based on the premise that the relevant fear network must be activated and corrective information introduced in the context of affective arousal and emotional engagement (Foa, Huppert,
& Cahill, 2006; Foa & Kozak, 1986). Emotional processing is characterized by a transient and curvilinear pattern of affective arousal and “anxiety spikes” (Heimberg & Becker, 2002) during treatment. Applying these principles to the treatment of depression, by examining shape of symptom change, Hayes and colleagues (2005, 2007a) identified a “depression spike.” This spike pattern represents a sudden, large increase and then decrease in depression symptoms during treatment. Thus, these “spikes” are transient, and eventually decrease. The depression spike pattern is conceptualized by Hayes and colleagues (2007a) as being associated with a disruption of old, maladaptive cognitive processes, followed by an increase in emotional processing. Hayes and colleagues (2007a) examined the depression spike pattern in 29 individuals undergoing an exposure-based cognitive therapy for major depressive disorder. Overall, over 60% of the sample experienced at least one depression spike during the exposure-activation phase of therapy, and the presence of a spike during this portion of treatment was associated with more emotional processing and predicted better treatment outcome (Hayes et al., 2007a). Among individuals with PTSD, only one study to date has examined symptom exacerbations. Foa, Zoellner, Feeny, Hembree, & Alvarez-Conrad (2002a) examined depressive symptom exacerbation during the onset of imaginal exposure in a sample of women receiving either PE or PE plus cognitive restructuring for chronic PTSD. Findings suggested that only a minority of clients in either condition experienced a reliable symptom worsening, and these worsenings were not related to treatment outcome or in-treatment dropout (Foa et al., 2002a). Thus, further research is needed to understand the impact of transient depression symptom worsenings on PTSD treatment outcome.

Predictors of Depression Symptom Discontinuities
Examining client characteristics associated with specific patterns of symptom change (e.g., symptom gains or spikes) can provide valuable insight for clinicians and potentially alter and guide their treatment approach. Understanding specific pre-existing client characteristics associated with particular patterns of change may allow clinicians to modify the delivery of PTSD treatment to better fit individual client needs. Social support is one client characteristic that may potentially impact the therapeutic process, particularly for clients with PTSD. Meta-analyses have found that lack of social support is in fact, one of the strongest predictors of PTSD (Brewin, Andrews, & Valentine, 2000; Ozer, Best, & Lipsey, 2003) development. PTSD clients tend to have relatively impoverished social support networks, reporting relationships characterized by difficulties with trust and feelings of stigmatization (e.g., Browne & Finkelhor, 1986; Schumm, Briggs-Phillips, & Hobfoll, 2006). Social support is a very broad and multifaceted construct (Norris & Kaniasty, 1996; Schumm, et al., 2006). Thus, in order to best understand the influences of support on treatment processes, examining multiple aspects of an individual’s network (e.g., positive vs. negative support, objective vs. perceived support) can provide clinicians and researchers with a deeper, more nuanced understanding of client well being.

A critical next step is to examine how social functioning may carry-over and influence therapeutic change and treatment processes. Only a few studies to date have examined the impact of social support on PTSD treatment outcome, and none have looked at the association between social support and symptom discontinuities during PTSD treatment. Tarrier and colleagues (2000), using an objective measure of social support, found that living alone predicted worse PTSD treatment outcome. More recently,
Thrasher and colleagues (2010), using a measure of perceived social support, found that higher levels of trauma-related support predicted better treatment outcome for individuals receiving cognitive restructuring and/or exposure therapy for PTSD. Unpacking the broad construct of social support can help to provide clearer evidence regarding unique roles that individual types of social support may have on PTSD treatment processes. Overall, examining pre-existing client factors, such as social support network, can help researchers to identify factors associated with particular change patterns and provide insight into the processes that underlie therapeutic improvement.

**Study Aims**

In the present study, we examined the occurrence of depression symptom discontinuities, namely sudden gains in depressive symptoms and depression spikes, in a sample of men and women receiving either psychotherapy (i.e., prolonged exposure; Foa et al., 1999; Foa et al. 2005) or pharmacotherapy (i.e., sertraline; Brady et al., 2000; Davidson et al., 2001) for chronic PTSD. There were four main goals of the present study. First, we examined the individual trajectories of depression symptoms to classify clients with a depression sudden gain or depression spike. Given findings from the depression literature (e.g., Hayes et al., 2007a; Tang et al., 2002; Tang et al., 2005), we hypothesized that approximately half of the participants would experience a sudden gain or depression spike. Next, we examined multiple facets of social support as potential predictors of depression symptom discontinuities. Given that higher support (e.g., Tarrier et al., 2000; Thrasher et al., 2010) and the presence of symptom spikes (e.g., Hayes et al., 2007a) and gains (e.g., Tang et al., 2007) have been associated with better treatment outcome, we hypothesized that higher levels of support would be associated with the
presence of a depression symptom discontinuity. Third, we compared the patterns (e.g., timing, magnitude) of depression sudden gains and depression spikes in individuals receiving either PE or SER. Given the level of emotional engagement and processing that occurs during PE, we hypothesized that more participants in PE would experience a depression symptom discontinuity than those in SER. Finally, we examined the relationship between depression symptom discontinuities and treatment outcome. In line with findings from the depression literature (e.g., Hayes et al., 2007a; Tang et al., 2007), we hypothesized that both the experience of a depression symptom spike and depression sudden gain would be associated with better treatment outcome.

Method

Participants

Two hundred women (75.5%, \( n = 151 \)) and men (24.5%, \( n = 49 \)) with a primary diagnosis of PTSD participated in the study. Participants were recruited for a PTSD treatment outcome study via referrals from medical professionals, local victim assistance agencies, and media advertisements. Participants had to be between the ages of 18-65 and meet DSM-IV criteria for a diagnosis of chronic PTSD. Exclusion criteria included: a) a primary DSM-IV diagnosis other than chronic PTSD; b) a current diagnosis of schizophrenia, other psychotic disorder, unstable bipolar disorder, substance dependence, or depression requiring immediate psychiatric treatment (e.g., suicidal intent or plan); and c) an ongoing relationship with the perpetrator, if the trauma was assault related. Of the individuals who were seen for an intake evaluation, 35% were not eligible for the study, most often because PTSD was not the primary diagnosis or they did not meet PTSD diagnostic criteria. An additional 16% were eligible but declined participation or did not
follow through with entering the study. The remaining individuals entered the study ($N = 200$).

On average, participants were 37.4 years old ($SD = 11.3$). Approximately 70% of the sample was not college educated. Approximately half of the sample (48.5%) had an annual household income of less than $20,000. The majority of the sample was Caucasian (65.5%), followed by African American (21.5%), and other ethnic backgrounds (13.0%). The most common primary trauma was adult sexual assault (31.0%), followed by childhood physical or sexual assault (24.0%), adult non-sexual assault (22.5%), accident (13.5%), death or violence to a loved one (6.5%), and combat/war (2.5%)

**Interview Measures**

Independent evaluators who received standardized training on the administration of the PTSD Symptom Scale-Interview (PSS-I) and Structured Clinical Interview for DSM-IV (SCID-IV) completed interview measures. Before serving as an independent evaluator, they must have met 80% reliability criterion for each interview measure. All interviewers were trained mental health professionals ranging from doctoral candidates in Ph.D. programs to Ph.D. level psychologists.

**PTSD Symptom Scale-Interview Version (PSS-I; Foa, Riggs, Dancu, & Rothbaum, 1993).** The PSS-I is an interviewer administered measure consisting of 17-items and produces both a score of PTSD severity and diagnostic status. Items are rated on a scale based on frequency and severity of symptoms from 0 (*not at all*) to 3 (*5 or more times per week/very much*) in the past two weeks. This measure was used to determine PTSD diagnosis for the study. The PSS-I demonstrates good convergent validity and inter-rater reliability, .93-.95 (Foa, Cashman, Jaycox, & Perry, 1997; Foa &
Tolin, 2000). In the current study, over 10% of cases were rerated for inter-rater reliability; reliability was high for PTSD severity scores (ICC = .95) and PTSD diagnosis ($\kappa = 1.00$).

**Structured Clinical Interview for DSM-IV (SCID-IV; First, Spitzer, Gibbon, & Williams, 1995).** The SCID-IV, a semi-structured interview, was used to determine if other Axis I disorders were primary. This measure has good inter-rater reliability (Skre, Onstad, Torgersen, & Kringlen, 1991). In the current study, 10% of the SCID-IVs were rerated for inter-rater reliability; reliability across current diagnoses was acceptable ($\kappa = .80$).

**Self-report Measures**

**PTSD severity.** The PTSD Symptom Scale-Self-Report (PSS-SR; Foa, Riggs, Dancu, & Rothbaum, 1993) is a self-report measure consisting of 17-items rating DSM-IV PTSD symptom severity and frequency. Participants rate their symptoms on a scale of 0 (*not at all*) to 3 (*5 times per week/very much*). This measure has good reliability and validity (Foa et al., 1993).

**Depression severity.** The Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) is a 21-item self-report measure assessing depression severity. Each item consists of four statements scored 0 to 3, with increasing scores indicating greater severity of depression. The BDI demonstrates good reliability and validity (Beck, Steer, & Garbin, 1988).

**Received general social support.** The Inventory of Socially Supportive Behaviors (ISSB; Barrera, Sandler, & Ramsey, 1981) is a 40-item self-report measure that was used to assess level of social support received from others. The ISSB measures
how often someone has done a certain activity (e.g., watched their possessions, gave them money, expressed concern for their well-being) in the past two weeks. Responses range from 1 (not at all) to 5 (about everyday), with a higher score indicating a higher level of support. This measure show high test-retest reliability and internal consistency (Barrera, 1981). This measure was administered at pre-treatment.

**Trauma-related social support.** The Social Reactions Questionnaire (SRQ; Ullman, 2000) is a 48-item questionnaire that was used to assess trauma-related social support. This measure generates two total scores, frequency of positive reactions (e.g., "how often someone has told you it was not your fault") and frequency of negative social reactions (e.g., "how often someone has told you that you were to blame"). Each response is rated on a scale from 0 (never) to 4 (always). This measure demonstrates good reliability and validity (Ullman, 2000). This measure was administered at pre-treatment.

**Social Satisfaction.** The Social Support Questionnaire (SSQ; Sarason, Sarason, Shearin, & Pierce, 1987) is a 6-item questionnaire that assesses number of social contacts and satisfaction with current support. Clients are asked to list individual’s from whom they receive support in a variety of situations (e.g., “Whom can you count on to console you when you are very upset?”). In addition, they are asked to rate their level of satisfaction with the support they are receiving in each of these situations on a scale from 1 (very dissatisfied) to 6 (very satisfied). This measure demonstrates good reliability and validity (Sarason et al., 1987).

**Total number of sessions.** To examine overall treatment completion, total number of sessions completed was recorded, with scores ranging from 0 to 10.

**Overview of Treatment**
Treatment consisted of either 10 weeks of psychotherapy or pharmacotherapy. For psychotherapy, all clinicians had at least a master’s level clinical training. For pharmacotherapy, all clinicians were board certified psychiatrists. All clinicians received standardized clinical training, through multiple-day initial training workshops and ongoing clinical supervision.

**Psychotherapy Treatment.** Prolonged exposure (PE; Foa, Hembree, & Dancu, 2002b) consists of 10 weekly, 90-120 min sessions, which include psychoeducation involving common reactions to trauma exposure, breathing retraining, approaching avoided situations outside of therapy (i.e., *in vivo* exposure) starting in Session 2, and approaching the memory of the trauma repeatedly (i.e., imaginal exposure) beginning at Session 3. Clients are assigned weekly homework including listening to their imaginal exposure tapes and practicing *in vivo* exposure exercises.

**Pharmacotherapy Treatment.** Pharmacotherapy consisted of 10 weeks of sertraline, monitored by a study psychiatrist. Each session ranged from 20-30 minutes. Sertraline was adjusted based on a standardized titration algorithm (Brady et al., 2000), starting at 25mg/day and proceeding up to 200mg/day, if indicated. For this sample, the mean dosage at the end of treatment was 135.68 mg/day (*SD* = 66.80). During visits, the psychiatrist monitored side effects and adjusted medication dosage as well as provided general encouragement and support.

**Procedure**

Participants provided written informed consent during an initial intake interview with an independent evaluator. During this interview, both demographic and diagnostic information were obtained. Primary diagnosis of chronic PTSD was determined via the
PSS-I and SCID-IV. Following this initial intake, if eligible, they came for a randomization appointment in which their treatment condition was randomly assigned. Participants also completed a battery of self-report measures including measures assessing current social support (ISSB, SRQ, SSQ) and severity of both PTSD (PSS-SR) and depression (BDI). Following this visit, patients received 10 weekly sessions of psychotherapy, PE, or pharmacotherapy, SER, for their chronic PTSD. Participants rated their depression symptom severity (BDI) at each treatment session. Following the completion of treatment, participants returned for a post-treatment evaluation with an independent evaluator who re-assessed their PTSD symptoms and diagnosis. Participants also completed a battery of self-report measures re-assessing current social support (ISSB, SRQ, SSQ) and severity of PTSD (PSS-SR) and depression (BDI).

Results

Data Analytic Strategy

Missing Data. The Last Observation Carried Forward (LOCF) imputation method was used in cases where in-session data was either missing or incomplete. The Food and Drug Administration (FDA) has traditionally viewed LOCF as the preferred method of analysis, considering it likely to be conservative, assuming no change (Hamer & Simpson, 2009). Thus, the use of this method did not artificially inflate the rates of depression spikes or gains. In addition, previous reports examining symptom discontinuities in PTSD trials have used this imputation method (e.g., Doane, Feeny, & Zoellner, 2010).

Power and Preliminary Analyses. A priori, we determined medium effect sizes (Cohen’s $d = .3$ or above) to be potentially clinically meaningful. Using the G-Power 3
software (Faul, Erdfelder, Lang, & Buchner, 2007), given the number of variables and our sample size, we were well powered (.80 and above) to detect such effect sizes for each study aim.

Prior to regression analyses, data was screened using SPSS REGRESSION and SPSS FREQUENCIES for evaluation of assumptions. A histogram of the residuals of each variable was plotted against the normal curve to determine normality. No transformations were necessary.

**Depression Symptom Discontinuities.** For the first set of analyses, the presence or absence of sudden, non-linear depression symptom discontinuities over the course of treatment were examined over the course of 10 sessions for 200 individuals receiving PTSD treatment. Thus, 1800 between session intervals were examined for the presence or absence of sudden symptom gains or symptom worsenings. Individuals were classified as having a sudden gain in depression symptoms (1 = sudden gain; 0 = no sudden gain) using guidelines set forth by Tang & DeRubeis (1999). In addition, participants were classified as having a transient depression spike or not (1 = depression spike; 0 = no depression spike), using guidelines similar to those originally used by Hayes and colleagues (2007a).

**Sudden Gains in Depression Symptoms.** Sudden gains were measured as outlined by Tang & DeRubeis (1999). A sudden gain is a rapid, non-linear decrease in depression symptoms over the course of one-between session interval (see Figure 1). Three criteria are necessary to define a sudden gain; the gain must be 1) large in absolute terms, 2) large relative to pre-gain symptom severity and 3) large relative to symptom fluctuations before and after the gain (Tang & DeRubeis, 1999). Sudden gains in
depression (using the BDI scores from each session) were calculated. Sudden gains in depression utilized the exact criteria outlined by Tang & DeRubeis (1999); thus, a sudden gain will be said to occur if 1) the BDI score decreases by at least 7 points between session N and N+1, 2) the gain represents at least 25% of the pre-gain session’s score, and 3) the mean BDI score of the three (or two) sessions before the gain is significantly higher than the mean of the three (or two) sessions after the gain. This third criterion was tested using a two-sample t-test using critical values of $t(4) = 2.78$, $p < .05$ or $t(3) = 3.19$, $p < .05$, in cases where only 2 pre-or post-gain sessions were available. Sudden gains occurred when all three conditions were satisfied.

**Transient Depression Spikes.** Depression spikes (Hayes et al., 2007a) have been much less examined, and have never been examined among individuals receiving treatment for PTSD. Depression spikes are considered to be transient, sudden worsenings in depressive symptoms over the course of treatment, which eventually decrease (See Figure 1). Depression spikes will be measured using the same 7-point criteria as defined by Tang & DeRubeis (1999). Participants will be classified as having a depression spike if they exhibit a transient increase in BDI symptoms, as defined by a 7-point increase, which subsequently decreases by at least 7 points during the course of 10 weeks of PTSD treatment.

Pre-treatment PTSD Severity, Depression Severity, and Social Support

The mean scores for all measures of pre-treatment psychopathology severity and

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1 Depression spikes are not simply the opposite of Tang & DeRubeis (1999) sudden gains. The third criterion cannot be applied to the depression spike. Post-spikesymptomatology does not have to be significantly lower than pre-spikesymptomatology (e.g., 28, 25, 39, 28, 25 is considered a spike, but would not qualify as such when utilizing Tang and DeRubeis (1999) third t-test criterion).
social support can be seen in Table 1. In general, this sample of individuals with chronic PTSD showed moderate to severe mean levels of PTSD symptoms, moderate to severe levels of depression, moderate levels of general support, and relatively low levels of trauma-related support. Overall, 54% of the sample met criteria for current major depression based on SCID-IV diagnosis.

**Frequency of Depression Symptom Discontinuities over the Course of PTSD Treatment**

First, the presence of sudden gains in depression symptoms was examined. When examining reductions in depression (BDI) symptoms of at least 7 points or more in one between session interval, 177 out of 1800 (9.8%) between session intervals met the first criterion. This level of depression symptom reduction was seen in 58% of clients (n = 115). Next, these 177 observations were examined to determine the magnitude of the reduction. Overall, 167 out of the 177 session interval 7-point reductions exceeded 25% of the pre-gain session depression severity score. Then, we examined the third criterion, whether the depression symptom gains were large relative to pre-gain and post-gain symptom fluctuation, and found that 38 out of the 167 symptom (22.7%) reductions met this criterion. Overall, a total of 38 sudden gains in depression symptoms were experienced by 36 out of 200 participants (18%), only two of whom experienced two depression sudden gains over the course of treatment. The mean session of the depression sudden gain was 7.36 (SD = 2.29; Range = 3-10) and the average magnitude of the depression sudden gain was 10.42 (SD = 3.22; Range = 7-19 points).

Next, the presence of depression spikes was examined. Of 200 participants entering either PE or SER treatment for chronic PTSD, 22.5% (n = 45) of individuals
experienced a transient depression spike over the course of treatment. Of those experiencing a transient depression spike, 78% \((n = 35)\) experienced one spike, and 22% \((n = 10)\) experienced two spikes. The mean session of the spike was 4.67 \((SD = 1.98;\) Range = 2-9) and the average magnitude of the depression spike was 10.78 \((SD = 5.04;\) Range = 7-28 points).

Overall, 34.0% of participants \((n = 68)\) experienced some type of discontinuity in their depression symptoms over the course of PTSD treatment. Only a small percentage of participants \((n = 13; 6.5\%)\) experienced both a depression spike and depression sudden gain over the course of PTSD treatment. In general, it appears that symptom discontinuities in depression symptoms over the course of PTSD treatment are fairly common.

**Pre-Treatment Variables and Depression Symptom Discontinuities.**

Prior to examining the relationship between social support and depression symptom discontinuities, a series of chi-square analyses were conducted to determine if any demographic variables were associated with depression sudden gains or spikes. Those who did and did not experience a depression symptom discontinuity did not significantly differ from one another on any demographic variable \((0 = \text{no depression symptom discontinuity}; 1 = \text{experienced either a sudden gain or spike})\)\(^2\) including gender, \(\chi^2 (1, N = 200) = .68, ns, (0 = \text{female}, 1 = \text{male})\), education level, \(\chi^2 (1, N = 200) = .72, ns, (0 = \text{not college educated}, 1 = \text{college educated})\), or ethnicity, \(\chi^2 (1, N = 200) = .77, ns, (0 = \text{Caucasian}, 1 = \text{other})\).

\(^2\) Chi-square analyses were also run separately for each type of depression symptom discontinuity (e.g., sudden gains, depression spikes) and no significant differences in frequency of depression spikes or sudden gains were found for any demographic variable.
The presence of a depression sudden gain was associated with pre-treatment PTSD severity ($r = .17, p < .05$) and pre-treatment depression severity ($r = .18, p < .05$). However, the presence of a depression spike was not associated with pre-treatment PTSD severity ($r = .05, ns$) or pre-treatment depression severity ($r = .11, ns$). Thus, it appeared that higher level of pre-treatment psychopathology was associated with sudden improvements in depressive symptoms, but was not associated with experiencing a sudden worsening in depression symptoms over the course of PTSD treatment.

Next, Pearson correlations were conducted to examine the association between multiple aspects of an individual’s social support network and the presence of depression symptom discontinuities. See Table 2. Overall, social support was not associated with the presence of a sudden gain in depressive symptoms. There was, however, a trend for higher positive trauma-related support to be associated with the presence of a sudden gain in depression symptoms ($r = .13, p = .07$). Those who experienced higher levels of negative trauma-related support were more likely to experience a transient, depression symptom worsening ($r = .18, p < .05$) over the course of treatment. Overall, it appears that social support has a small, but significant impact on the occurrence of depression spikes, and not all types of social support impact symptom trajectory equally.

Differences in Patterns of Depression Symptom Discontinuities in PE vs. SER

Next, analyses were conducted to determine if there were differences in the presence, timing, or magnitude of depression symptom discontinuities between two treatment options: psychotherapy (PE) and pharmacotherapy (SER). First, a series of chi-square analyses were conducted to determine if there was a difference in the proportion of individuals who experienced a depression symptom discontinuity between PE and
SER. Overall, there was not a significant difference in the percentage of individuals experiencing a depression sudden gain between PE and SER, $\chi^2 (1, N = 200) = 1.35, ns$, with 20% of individuals in PE and 14% of individuals in SER experiencing a sudden gain in depression symptoms. Similarly, there was not a significant difference in the percentage of individuals experiencing a depression spike between PE and SER, $\chi^2 (1, N = 200) = .09, ns$, with 23% of individuals in PE and 21% of individuals in SER experiencing a transient spike in depression symptoms.

A series of ANOVAs were conducted to determine if there were differences in timing or magnitude of depression symptom discontinuities between PE and SER. See Table 3. Overall, there were no significant differences between PE and SER on the timing ($F(1, 35) = .26, ns$) or magnitude ($F(1, 35) = .01, ns$) of depression sudden gains. Similarly, no differences were observed between timing ($F(1, 44) = .59, ns$) or magnitude ($F(1, 44) = .02, ns$) of depression spikes between PE and SER.

Of those in SER, there was a trend toward significance of experiencing a depression spike being associated with a higher end dosage of SER ($r = .22, p = .06$). Experiencing a depression sudden gain was not significantly associated with end dosage ($r = .19, ns$).

**Symptom Discontinuities and their Association with Treatment Outcome**

In the total sample, experiencing a depression symptom discontinuity was associated with attending a higher number of treatment sessions. The presence of a depression sudden gain ($r = .33, p < .05$) and the presence of a depression symptom spike ($r = .29, p < .05$) were associated with a higher number of treatment sessions attended.

Finally, analyses were conducted to determine if experiencing a depression
symptom discontinuity over the course of PTSD treatment was associated with lower post-treatment PTSD and depression severity. Overall, the presence of a depression sudden gain was associated with lower post-treatment PTSD severity ($r = -.20$, $p < .05$) and post-treatment depression severity ($r = -.19$, $p < .05$). However, experiencing a depression spike was not associated with post-treatment PTSD severity ($r = -.09$, ns) or depression severity ($r = -.11$, ns). When looking at post-treatment PTSD diagnostic status, there was a trend toward a lower portion of those experiencing a sudden gain retaining their PTSD diagnosis, with 9% of individuals who experienced a sudden gain in depression retaining their PTSD diagnosis and 21% of individuals who did not experience a sudden gain retaining their PTSD diagnosis $\chi^2 (1, N = 200) = 2.98$, $p = .08$. Similarly, there was a trend toward a lower portion of individuals who experienced a depression spike to retain their PTSD diagnosis, with 10% of individuals who experienced a depression spike retaining their PTSD diagnosis, and 22% of individuals who did not experience a depression spike retaining their PTSD diagnosis $\chi^2 (1, N = 200) = 2.79$, $p = .09$.

In order to examine sudden gains in depression as a predictor of treatment outcome, two regressions were conducted. Regressions were not conducted to examine whether or not depression spikes predicted outcome, given that Pearson correlations were not significant. In the first regression, PTSD post-treatment severity was the dependent variable. In Step 1, we entered centered pre-treatment PTSD severity and pre-treatment depression severity scores. In Step 2, we entered depression sudden gains ($-0.5 = \text{no sudden gain}, 0.5 = \text{sudden gain}$). As shown in Table 4, both the experience of a sudden gain in depression symptoms ($\beta = -.32$, $p < .05$) and lower pre-treatment PTSD symptoms
(β = .21, p < .05) emerged as significant predictors of lower post-treatment PTSD severity.

In the second regression, post-treatment depression severity was entered as the dependent variable. In Step 1, we entered centered pre-treatment PTSD and depression severity scores. In Step 2, we entered depression sudden gains (-.5 = no sudden gain, .5 = sudden gain). As shown in Table 5, both lower pre-treatment depression severity (β = .49, p < .05) and the experience of a depression sudden gain (β = -.32, p < .05) emerged as predictors of lower post-treatment depression severity.

Thus, overall it appears that sudden symptom improvements in depression symptoms over the course of PTSD treatment predict lower post-treatment PTSD and depression severity. However, the presence of transient depression symptom worsenings over the course of PTSD treatment was not associated with treatment outcome.

**Discussion**

Overall, depression symptoms did not change linearly for a significant portion of individuals receiving PTSD treatment. In this sample, 34% of individuals experienced a discontinuity in their depression symptoms, evidenced by either a sudden gain in depression symptoms or a transient depression spike. This was the first study to examine sudden gains and transient spikes in depression symptoms among individuals receiving either psychotherapy or pharmacotherapy for PTSD. These results highlight the discontinuous nature of therapeutic change, and provide further evidence that periods of rapid change and turbulence may play an important role in overall symptom reduction (Hayes et al., 2007b).

Interestingly, it appears that not all discontinuities are equally beneficial for
clients with PTSD. This is contrary to findings in the depression literature, which suggest that both transient symptom spikes and rapid improvements are associated with improved treatment outcome (e.g., Drymalski & Washburn, 2011; Hayes et al., 2007a; Tang et al., 2002; Tang et al., 2005). In the treatment of depression, sudden gains are common, with 30-50% of clients experiencing sudden decreases in depressive symptoms. However, in this sample of clients with a primary diagnosis of chronic PTSD, a smaller portion of clients (18%) experienced a rapid depression symptom decrease. Encouragingly, in line with previous research (e.g., Drymalski & Washburn, 2011; Kelly et al., 2005; Tang et al., 2002; Tang et al., 2005; Vittengl et al., 2005), sudden gains in depression were associated with better PTSD and depression treatment outcome. Sudden, transient depression symptom worsenings also occurred, with 22.5% of participants experiencing a depression spike. Only one other study to date has examined depression spikes, and found that 62% of clients receiving an exposure-based psychotherapy for depression experienced a depression spike at the onset of exposure (Hayes et al., 2007a). Contrary to our original hypothesis, as well as Hayes et al.’s (2007a) findings, transient depression symptom worsenings were not associated with improved treatment outcome and occurred at a substantially lower rate than previously reported (e.g., Hayes et al., 2007a). These dissimilar findings between the PTSD and depression literature suggest that the trajectory of depression symptoms over the course of treatment may be different for those with a primary diagnosis of PTSD. There may be a number of reasons for these differences. First, depression was not the primary diagnosis for this sample, thus potentially lowering the rates of gains and spikes in symptoms. However, 54% of individuals in this sample also met current criteria for current MDD, and mean depression scores for the sample
were moderate to severe. Second, depression spikes may not be a sign of treatment improvement for those with primary PTSD, and instead, increases in depressive symptoms (e.g., lack of interest, sleep and concentration difficulties, sadness) may lead to decreased adherence or motivation to complete treatment. However, given that the experience of a depression spike was associated with completing more treatment, this hypothesis is unlikely. Finally, although depression spikes were not directly related to primary outcome measures, there may be other more subtle impacts of depression spikes on overall client functioning. Considering the tentative evidence that depression discontinuities, namely sudden gains, improve other areas of functioning (e.g., quality of life; Drymalski & Washburn, 2011), future studies should consider examining the impact of depression spikes on additional measures of client social functioning (e.g., family functioning, work functioning, etc.). Thus, overall, sudden gains in depression appear to be more influential than depression spikes in PTSD treatment outcome.

In order to begin to understand factors that influence therapeutic change, pre-treatment social support was examined as a potential predictor of depression discontinuities. A large body of research suggests that lack of support is a risk factor for PTSD development (e.g., Ozer et al., 2000; Brewin et al., 2003). However, no previous studies have examined the role of social support in depression sudden gains or spikes and few have examined the role of social support in PTSD treatment processes (e.g., Keller, Zoellner, & Feeny, 2010; Tarrier et al., 2000; Thrasher, Power, Morant, Marks, Dalgleish, 2010). Thrasher and colleagues (2010), examining individuals receiving psychotherapy for PTSD, found that lower levels of pre-treatment trauma support predicted worse PTSD treatment outcome. Similarly, Keller, Zoellner, & Feeny (2010)
found that lower trauma related support was associated with lower early therapeutic alliance among individuals receiving PE or SER for PTSD. In line with these findings, (e.g., Keller et al., 2010; Thrasher et al., 2010), our results suggest that trauma related support, rather than general support received or an objective measure of support, size of social network, appears to be influential in predicting depression symptom discontinuities. Similarly, others have suggested that perceptions of support, rather than actual support received, seem to be more important in influencing psychopathology, especially following a trauma (Thompson et al., 2000; Yap & Devilly, 2004). Our results indicated that higher levels of negative trauma support were associated with the presence of a depression spike. In addition, there was a trend for higher positive trauma-related support to be associated with the presence of a sudden gain. Clinically, this suggests that therapists should pay particular attention to clients’ views of their social network, and encourage an increase in positive interactions and a reduction of negative interactions, particularly surrounding their traumatic event. Thus, rather than increasing the quantity of social interactions, it appears that improving the quality of current relationships may be more beneficial for therapeutic change. This may be particularly relevant for clients entering exposure therapy, who may be encouraged to interact with others, in relation to their traumatic event, for their in vivo homework assignments (e.g., driving to a feared location with a friend, increasing intimacy with a significant other). Overall, as others have suggested (Norris & Kaniasty, 1996; Schumm, et al., 2006), it appears that social support is a multi-faceted construct, and not all facets contribute equally to treatment processes. Clinicians should pay attention to multiple aspects of a client’s social network, and encourage a reduction of negative engagement with current supports.
Interestingly, patterns of depression symptom discontinuities did not substantially differ between psychotherapy (PE) and pharmacotherapy (SER). Despite potentially dissimilar treatment mechanisms, patterns of change in depression symptoms may actually be quite similar. Contrary to our original hypothesis, depression discontinuities did not occur at significantly higher rates in PE than SER. These findings are in line with Vittengl and colleagues (2005) findings, who found that sudden gains are not unique to psychotherapy. Similarly to our findings, Vittengl et al. (2005) found that clients receiving pill placebo, cognitive therapy, or pharmacotherapy for depression experienced sudden gains at similar rates. These findings challenge the notion that Tang & DeRubeis (1999) put forth in which sudden symptom gains are a result of a rapid shift in cognitive insight. However, it may be that cognitive shifts occur in clients receiving pharmacotherapy as well, despite cognitive work not being a treatment target.

Alternatively, shared factors between both treatments, rather than components of the treatment regimen, may be contributing to symptom discontinuities (e.g., client expectations, views of the self, therapeutic alliance, etc.). This is the first study to compare rates of depression spikes in a pharmacotherapy and psychotherapy for PTSD. Similar to sudden gains, rates of spikes did not differ between treatments. Given that some individuals report experiencing side effects to SER, it may be that depression spikes could be associated with an increase in side effects. Thus, despite similar rates of spikes between treatments, the process underlying the spike pattern may be quite different. Future studies may want to consider examining additional predictors of depression symptom discontinuities between these dissimilar treatment options to further understand the processes that drive symptom discontinuities. Clinically, these findings suggest that
both pharmacotherapists and psychotherapists should be attuned to depression symptom shifts in their PTSD clients, as there is a likelihood that clients in either treatment may experience a transient depression spike or sudden gain. Future studies may consider examining shared factors among psychotherapy and pharmacotherapy options and their impact on therapeutic change in these different treatment options.

There were a few limitations of the present study. First, both depression symptom severity and social support variables were self-report measures. Thus, the relationship between these variables may be exaggerated due to shared method variance. Additionally, we measured social support at pre-treatment, and did not track changes in support over the course of treatment. Future studies should consider tracking changes in support over the course of treatment, in order to assess whether the relationship between support and therapeutic change is causal in nature. Finally, although we examined the impact of symptom discontinuities on treatment outcome, we did not examine the impact of spikes or gains on secondary outcome measures. Thus, future research should consider examining the impact of spikes and gains on additional areas of client functioning (e.g., quality of life, social functioning, etc.).

Although clinicians are often concerned about sudden symptom worsening (Becker et al., 2004; Foa et al., 2002; van Minnen et al., 2010) as well as implementing PE among those with comorbid diagnoses such as depression (Becker et al., 2004), transient depression symptom worsenings that were subsequently resolved were not detrimental to treatment outcome. Encouragingly, rapid depression symptom decreases in depression were associated with improved treatment outcome. Thus, clinicians may want to be particularly attentive to sudden improvements in depression symptoms for
clients with PTSD, as these shifts may be a marker of overall treatment improvement. Future research examining therapeutic change processes, including symptom discontinuities, can potentially improve current treatment options by modifying treatment protocols for individual client characteristics. In addition, tracking patterns and processes over time allows researchers and clinicians to pinpoint components of the treatment regimen that are crucial in influencing change. Overall, these results suggest that discontinuities in depressive symptoms occur fairly often during PTSD treatment and may be indicators of treatment response.
Figure 1. Depression Symptom Discontinuities over the Course of PTSD Treatment
Table 1

*Means and Standard Deviations for Self-Reported PTSD Severity, Depression Severity, and Social Support at Pre-treatment.*

<table>
<thead>
<tr>
<th>Self-Report Measures</th>
<th>Mean (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTSD Severity (PSS-SR)</td>
<td>34.47(8.00)</td>
<td>11-51</td>
</tr>
<tr>
<td>Depression Severity (BDI)</td>
<td>25.03(9.77)</td>
<td>4-48</td>
</tr>
<tr>
<td>General Social Support (ISSB)</td>
<td>86.86(30.63)</td>
<td>40-174</td>
</tr>
<tr>
<td>Social Support Questionnaire-Number of Contacts (SSQ)</td>
<td>13.20(10.32)</td>
<td>0-54</td>
</tr>
<tr>
<td>Social Support Questionnaire-Satisfaction (SSQ)</td>
<td>22.81(9.36)</td>
<td>5-36</td>
</tr>
<tr>
<td>Positive Trauma Related Social Support (SRQpos)</td>
<td>1.78(.76)</td>
<td>0-3.61</td>
</tr>
<tr>
<td>Negative Trauma Related Social Support (SRQneg)</td>
<td>1.26(.66)</td>
<td>.13-3.33</td>
</tr>
</tbody>
</table>

Note. PSS-SR = PTSD Symptom Scale – Self-Report (n = 200); BDI = Beck Depression Inventory (n = 200); ISSB = Inventory of Socially Supportive Behaviors (n = 194); SSQ = Social Support Questionnaire (n = 193); SRQpos = Social Reactions Questionnaire, positive support scale (n = 195); SRQneg = Social Reactions Questionnaire, negative support scale (n = 195).
Table 2

*The Association Between Pre-Treatment Social Support and Depression Symptom Discontinuities Over the Course of PTSD Treatment*

<table>
<thead>
<tr>
<th></th>
<th>Presence of a Depression Sudden</th>
<th>Presence of a Depression Spike</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Gain</td>
<td></td>
</tr>
<tr>
<td>Positive Trauma-Related Support</td>
<td>.13</td>
<td>.01</td>
</tr>
<tr>
<td>(SRQpos)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative Trauma-Related Support</td>
<td>.03</td>
<td>.18*</td>
</tr>
<tr>
<td>(SRQneg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>General Support (ISSB)</td>
<td>.10</td>
<td>.05</td>
</tr>
<tr>
<td>Satisfaction with Current Support</td>
<td>.06</td>
<td>-.10</td>
</tr>
<tr>
<td>(SSQ)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of Social Contacts (SSQ)</td>
<td>.03</td>
<td>.03</td>
</tr>
</tbody>
</table>

Note: *p < .05
Table 3

*Timing and Magnitude of Depression Symptom Discontinuities Between PE and SER*

<table>
<thead>
<tr>
<th></th>
<th>PE</th>
<th></th>
<th>SER</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Range</td>
<td>Mean (SD)</td>
<td>Range</td>
</tr>
<tr>
<td>Depression Sudden Gain</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Timing</td>
<td>7.5 (2.35)</td>
<td>3-10</td>
<td>7.08 (2.23)</td>
<td>3-9</td>
</tr>
<tr>
<td>Magnitude</td>
<td>10.47 (3.35)</td>
<td>7-19</td>
<td>10.33 (3.08)</td>
<td>7-18</td>
</tr>
<tr>
<td>Depression Spike</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Timing</td>
<td>4.48 (2.04)</td>
<td>2-9</td>
<td>4.94 (1.89)</td>
<td>2-9</td>
</tr>
<tr>
<td>Magnitude</td>
<td>10.87 (5.17)</td>
<td>7-28</td>
<td>10.63 (4.97)</td>
<td>7-25</td>
</tr>
</tbody>
</table>
Table 4

Pre-treatment Psychopathology and Sudden Gains in Depression Symptoms as Predictors of Post-treatment PTSD Severity

<table>
<thead>
<tr>
<th>Prediction of Post-treatment PTSD Severity</th>
<th>Step</th>
<th>$B$</th>
<th>SE($B$)</th>
<th>$\beta$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$\Delta R^2$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Step 1&lt;sup&gt;a&lt;/sup&gt;</td>
<td></td>
<td>.07*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-treatment PTSD Severity (PSS-SR)</td>
<td></td>
<td>.32</td>
<td>.16</td>
<td>.19*</td>
</tr>
<tr>
<td>Pre-treatment Depression Severity (BDI)</td>
<td></td>
<td>.13</td>
<td>.13</td>
<td>.09</td>
</tr>
<tr>
<td>Step 2&lt;sup&gt;b&lt;/sup&gt;</td>
<td></td>
<td>.10*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-treatment PTSD Severity (PSS-SR)</td>
<td></td>
<td>.37</td>
<td>.15</td>
<td>.21*</td>
</tr>
<tr>
<td>Pre-treatment Depression Severity (BDI)</td>
<td></td>
<td>.18</td>
<td>.13</td>
<td>.13</td>
</tr>
<tr>
<td>Sudden Gain in Depression</td>
<td></td>
<td>-11.54</td>
<td>2.43</td>
<td>-.32*</td>
</tr>
</tbody>
</table>

Note: Dependent Variable = post-treatment PTSD severity

<sup>a</sup>Step 1: $R = .25$, $F(2, 197) = 6.82$, $p < .05$

<sup>b</sup>Step 2: $R = .40$, $F(3, 196) = 12.56$, $p < .05$

* $p < .05$
Table 5

*Pre-treatment Psychopathology and Sudden Gains in Depression Symptoms as Predictors of Post-treatment Depression Severity*

<table>
<thead>
<tr>
<th>Prediction of Post-treatment Depression Severity</th>
<th>Step</th>
<th>$B$</th>
<th>$SE(B)$</th>
<th>$\beta$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$\Delta R^2$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Step 1$^a$</td>
<td></td>
<td>.17*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-treatment PTSD Severity (PSS-SR)</td>
<td></td>
<td>-.10</td>
<td>.13</td>
<td>-.07</td>
</tr>
<tr>
<td>Pre-treatment Depression Severity (BDI)</td>
<td></td>
<td>.56</td>
<td>.11</td>
<td>.46*</td>
</tr>
<tr>
<td>Step 2$^b$</td>
<td></td>
<td>.10*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-treatment PTSD Severity (PSS-SR)</td>
<td></td>
<td>-.06</td>
<td>.12</td>
<td>-.04</td>
</tr>
<tr>
<td>Pre-treatment Depression Severity (BDI)</td>
<td></td>
<td>.61</td>
<td>.10</td>
<td>.49*</td>
</tr>
<tr>
<td>Sudden Gain in Depression</td>
<td></td>
<td>-9.87</td>
<td>1.95</td>
<td>-.32*</td>
</tr>
</tbody>
</table>

*Note: Dependent Variable = post-treatment depression severity*  

$^a$Step 1: $R = .41, F(2, 197) = 20.30, p < .05$  

$^b$Step 2: $R = .52, F(3, 196) = 23.79, p < .05$  

* $p < .05$
References


