Published in final edited form as:
J Consult Clin Psychol. 2014 February ; 82(1): 102–111. doi:10.1037/a0035286.

Depression Sudden Gains and Transient Depression Spikes During Treatment for PTSD

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Abstract

Objective—We know little about how change unfolds in depression symptoms during PTSD treatment or how patient characteristics predict depression symptom change. This study examined critical transition points in depression symptoms during PTSD treatment, namely depression sudden gains, which are rapid symptom improvements and transient depression spikes, which are transient depression worsenings. Social support, one of the strongest predictors of PTSD development, was examined as a predictor of depression symptom discontinuities.

Method—At pre-treatment, 200 participants (76.6% female; 64.9% Caucasian; age (M = 37.1 (SD = 11.3) years) completed measures of PTSD severity (PTSD Symptom Scale-Self Report), depression severity (Beck Depression Inventory), general social support (Inventory of Socially Supportive Behaviors; Social Support Questionnaire), and trauma-related social support (Social Reactions Questionnaire). During 10 weeks of prolonged exposure (PE) or sertraline, depression was assessed weekly.

Results—Overall, 18.0% of participants experienced a depression sudden gain and 22.5% experienced a transient depression spike. The presence of a depression sudden gain predicted better treatment outcome, $\beta = -4.82$, $SE = 1.17$, $p = .001$, 95% CI [−6.79, −2.90]. Higher perceptions of negative trauma-related reactions, albeit modestly, were associated with experiencing a transient depression spike ($r = .18$, $p = .01$). There were no differences in rates of depression sudden gains or transient depression spikes between treatments.

Conclusions—Encouragingly, rapid improvements in depression symptoms are beneficial for PTSD treatment outcome but transient spikes in depressive symptoms do not strongly influence outcome. Understanding symptom discontinuities may help us to personalize current PTSD treatment options.

Keywords

PTSD; transient depression spikes; sudden gains; social support; exposure; sertraline

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 (e.g., Brady et al., 2000; Foa et al., 2005). 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,

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2007) that such process-oriented research is the “next-step” in improving treatment delivery. Examining process-oriented questions, such as how symptoms change over the course of treatment, can help clinicians and researchers to better understand *how* PTSD treatments exert their effect.

No studies to date have examined rapid depression symptom fluctuations over the entire course of PTSD treatment for adults. Examining patterns of depression symptoms for patients with PTSD is important for a number of reasons. First, comorbidity of PTSD with another Axis I or Axis II disorder is the norm in the aftermath of a traumatic experience (e.g., Creamer, Burgess, & McFarlane, 2001). Second, major depression (MDD) is one of the most commonly co-occurring conditions associated with PTSD (e.g., Creamer et al., 2001; Nixon, Resick, & Nishith, 2004), with cross-sectional studies reporting comorbidity rates of approximately 50–80% (e.g., Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Hankin, Spiro, Miller, & Kazis, 1999). 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 may possibly benefit less from treatment (e.g., Green et al., 2006; Nixon et al., 2004). Fourth, tracking depression over the course of treatment for individuals with PTSD may provide clinicians with a deeper, more broadly focused picture of patient functioning. Specifically, a worsening in depression may make it more difficult for patients to engage effectively or become motivated to actively participate in a PTSD treatment regimen. Finally, clinicians often have concerns continuing with exposure therapy if patients experience symptom increases (Becker, Zayfert, & Anderson, 2004; Foa, Zoellner, Feeny, Hembree, & Alvarez-Conrad, 2002; van Minnen, Hendriks, & Olff, 2010). In a survey of licensed psychologists, Becker and colleagues (2004) found that 37% of respondents reported that any co-morbid diagnosis was a factor that would deter them from administering exposure therapy. Thus, examining depression symptoms for patients with PTSD can provide a more nuanced portrait of patient functioning and provide insight to clinicians regarding the impact of these symptom fluctuations.

Prior research tracking patterns of depression symptom change throughout treatment for depression has primarily focused on points of symptom worsening or substantial improvement. Symptom discontinuities, both rapid symptom decreases (e.g., Tang & DeRubeis, 1999) and transient symptom spikes (e.g., Hayes, Feldman, Beevers, Laurenceau, & Cardaciotto, 2007) are common during psychotherapy for depression and both may predict better treatment outcome. However, what is less known is how change unfolds in pharmacotherapy. This manuscript examined both sudden improvements, termed *depression sudden gains*, and transient worsenings, termed *transient depression spikes*, over the course of both exposure and sertraline treatment for chronic PTSD.

Systematic reviews of the evidence support the efficacy of both psychotherapy and pharmacotherapy for PTSD (e.g., Bisson & Andrew, 2005; Stein, Ipser & Seedat, 2006). Prolonged exposure (PE), a first line psychotherapy for PTSD (e.g., Institute of Medicine, 2007), has been shown to be efficacious in many trials and is well tolerated (e.g., Foa et al., 2002; Foa et al., 2005). PE uses cognitive and behavioral strategies to reduce fear (Foa, Dancu, & Hembree, 2002). 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 also altered (Foa & Rauch, 2004). On the other hand, sertraline, an FDA-approved selective serotonin reuptake inhibitor, is also an empirically supported intervention for PTSD (e.g., Brady et al., 2000). Pharmacotherapy with sertraline involves taking a daily dosage, titration up to therapeutic levels, and discussing symptoms and side effects with a pharmacotherapist. Although these treatments appear very different, little is known regarding how similarly or dissimilarly therapeutic change unfolds. Tang and colleagues (2005) suggested that sudden

gains are associated with a shift or correction in negative attitudes and beliefs. Since changes in core belief systems are not the target of pharmacotherapy interventions and successful PE treatment is associated with cognitive change (Foa & Rauch, 2004) there may likely be fewer opportunities for sudden gains in pharmacotherapy. Also, symptom discontinuities during pharmacotherapy may be associated with factors shared between both treatments such as therapeutic alliance or a discussion of symptoms (e.g., psychoeducation, potential side effects). Overall, examining differences in the processes between exposure therapy and pharmacotherapy can help us better understand how these treatments work.

Sudden gains in depression (Tang & DeRubeis, 1999) are rapid, large *decreases* in symptoms that occur in one between-session interval. In psychotherapy for depression, sudden gains are common, with 30–50% of patients experiencing sudden depression improvements, and these sudden gains predict better treatment outcome (e.g., Drymalski & Washburn, 2011; Tang, DeRubeis, Beberman, & Pham, 2005). Only one study has compared the rates of sudden gains among depressed patients undergoing psychotherapy, pill placebo, or pharmacotherapy (Vittengl et al., 2005). Similar rates of sudden gains were found across treatment conditions. No studies however, have examined depression sudden gains among patients receiving treatment for a primary diagnosis of chronic PTSD.

Exposure-based therapies for anxiety disorders theorize that symptom improvement results from activation of the relevant fear network and the introduction of corrective information (Foa & Kozak, 1986) into that network, termed emotional processing. The introduction of corrective information may result in a transient increase in arousal or “anxiety spikes” (Heimberg & Becker, 2002). Applying these principles to the treatment of depression, Hayes and colleagues (2005, 2007) identified “depression spikes.” 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 is conceptualized by Hayes and colleagues (2007) as being associated with a disruption of old, maladaptive cognitive processes, followed by an increase in emotional processing. Hayes and colleagues (2007) examined the transient depression spike pattern in 29 individuals undergoing an exposure-based cognitive therapy for major depressive disorder. Over 60% of the sample experienced at least one transient 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., 2007). Among individuals with PTSD, only one study to date has examined depression symptom worsenings. Foa and colleagues (2002) examined depressive symptom exacerbation during the onset of imaginal exposure in a sample of women receiving PE or PE plus cognitive restructuring for chronic PTSD. Only a minority of patients in either condition experienced a reliable symptom worsening and these worsenings were not related to worse treatment outcome or dropout (Foa et al., 2002). Thus, further research is needed to understand the impact of transient depression spikes on PTSD treatment outcome.

Examining patient characteristics associated with specific patterns of change can provide valuable insight for clinicians and potentially alter and guide their treatment approach. Social support is one patient characteristic that may potentially impact the therapeutic process. Patients with PTSD often report relationships characterized by difficulties with trust and feelings of stigmatization (e.g., Schumm, Briggs-Phillips, & Hobfoll, 2006). Long-standing evidence has highlighted the interpersonal nature of depression (e.g., Haeffel, Voelz, & Joiner, 2007), with some suggesting that interpersonal factors are the strongest predictors of depression onset, course, and duration (e.g., Lara, Leader, & Klein, 1997; Whisman, 2001). There is also growing evidence to suggest that low levels of support are associated with poor treatment outcome for those with PTSD (e.g., Price, Gros, Strachan, Ruggiero, & Acierno, 2013; Thrasher et al., 2010) and depression (e.g., George, Blazer,

Hughes, & Fowler, 1989). Given that low levels of support are associated with PTSD and depression development and course, patients with lower levels of support may be more likely to transient depression spikes during PTSD treatment. Specifically, during PE (e.g., approaching feared and previously avoided situations/thoughts/feelings during *in vivo* homework), many individuals complete assignments with their social network (e.g., going to dinner with a friend, calling a family member). Additionally, many patients in both psychotherapy and pharmacotherapy share their treatment experiences with their social network (e.g., telling partner about the successes or difficulties with PTSD treatment). However, feeling detached from others is a core symptom of PTSD. Thus, individuals with poor or few social relationships, particularly surrounding their trauma, may have difficulty effectively engaging in therapy or utilizing their support network. These difficulties may lead an individual to experience an increase in sadness, self-disappointment or guilt, and irritability towards others or themselves which are all symptoms of depression. Examining patient factors such as social support may help to identify factors associated with particular change patterns and provide insight into processes that underlie therapeutic improvement.

In the present study, we tracked depression symptom patterns in a sample of men and women receiving either PE or sertraline for chronic PTSD. There were four main goals of the present study. First, we examined the individual trajectories of depression symptoms. We assessed whether patients with PTSD experience depression sudden gains or transient depression spikes during PTSD treatment. Next, we examined multiple facets of social support as potential predictors of these depression symptom discontinuities. We hypothesized that lower levels of support would be associated with the presence of transient depression spike. Third, we compared the patterns (e.g., timing, magnitude) of depression sudden gains and transient depression spikes in individuals receiving either PE or sertraline. We hypothesized that more individuals in PE would experience depression symptom discontinuities than those in sertraline, based on the sudden gains literature. Finally, we examined the relationship between depression symptom discontinuities and treatment outcome. We hypothesized that both the experience of a depression sudden gain and a transient depression spike would be associated with better treatment outcome. We also hypothesized that the experience of a depression symptom discontinuity (i.e., transient depression spike or depression sudden gain) would mediate the relationship between pre-treatment and post-treatment symptom severity.

Method

Participants

Two hundred women (75.5%, $n = 151$) and men (24.5%, $n = 49$) with a primary diagnosis of chronic PTSD participated in the study. Participants were recruited as part of a PTSD treatment outcome study via referrals from medical professionals, local victim assistance agencies, and media advertisements. Participants were between the ages of 18–65 years old and met 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., current suicidal intent or plan, or a suicide attempt in the previous three months); and c) an ongoing relationship with the perpetrator, if the trauma was assault related. Of the individuals who were seen for an intake evaluation ($N = 426$), 53% ($N = 226$) were not eligible for the study or declined participation. The most common reason for ineligibility was because PTSD was not the primary diagnosis or they did not meet PTSD diagnostic criteria. The remaining individuals entered the study ($N = 200$).

On average, participants were 37.41 years old (*SD* = 11.30). Approximately 70% of the sample was not college educated and almost 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 index 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%). Average time since index trauma was 11.97 years (*SD* = 12.69).

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. All interviewers were trained mental health professionals.

PTSD Symptom Scale-Interview Version (PSS-I; Foa, Riggs, Dancu, & Rothbaum, 1993)—The PSS-I is an interview measure consisting of 17-items. This measure yields 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 when compared to the PTSD section of the Structure Clinical Interview for DSM-IV Disorders (ρ = .73; Foa & Tolin, 2000) and also demonstrates high levels of inter-rater reliability (.91-.93; Foa & Tolin, 2000). In the current study, 10% of cases were rerated for inter-rater reliability; reliability was high for PTSD severity scores (ICC = .985) and PTSD diagnosis (κ = 1.00).

Structured Clinical Interview for DSM-IV (SCID-IV; First, Spitzer, Gibbon, & Williams, 1995)—The SCID-IV, a semi-structured interview, was administered at pre-treatment and 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 (κ = .80).

Self-report Measures

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 high internal consistency (.81-.86) and concurrent validity with other measures of depression (Beck, Steer, & Garbin, 1988); internal consistency in the current sample at pre-treatment was .88. This measure was used to determine the presence of depression sudden gains and transient depression spikes.

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). Internal consistency in the current sample was .85 at pre-treatment.

Received general social support—The Inventory of Socially Supportive Behaviors (ISSB; Barrera, Sandler, & Ramsey, 1981) is a 40-item self-report used to assess social support received from others. The ISSB measures how often someone has done a certain

activity (e.g., watched their possessions, loaned money, expressed concern) in the past two weeks. Responses range from 1 (*not at all*) to 5 (*about everyday*), with higher scores indicating higher support. The ISSB shows high test-retest reliability and internal consistency (Barrera et al., 1981). Internal consistency in the current sample was .80.

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 and was administered at pre-treatment. 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*). Adequate internal consistency (ranging from .77 – .93) and test–retest reliability (ranging from .64 – .81) have been previously reported in sexual assault samples (Ullman, 2000). Internal consistencies were adequate in the current sample (SRQ positive α = .78; SRQ negative α = .80).

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. Patients are asked to list individuals 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?"). They are also asked to rate their level of satisfaction with their support 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). Internal consistency in the current sample was .80.

Overview of Treatment

Treatment consisted of 10 weeks of either exposure therapy or sertraline. All psychotherapists were doctoral-level clinicians with at least a master's degree. All pharmacotherapists were board certified psychiatrists. Clinicians received standardized clinical training via training workshops and ongoing clinical supervision or consultation.

Psychotherapy—Prolonged exposure (PE; Foa, Hembree, & Dancu, 2002) consisted of 10 weekly, 90–120 min sessions, which include psychoeducation, 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) and processing of this exposure beginning at Session 3. Patients were assigned weekly homework including listening to their imaginal exposure tapes and practicing *in vivo* exposure exercises.

Pharmacotherapy—Pharmacotherapy consisted of 10 weeks of sertraline, monitored by a study psychiatrist who followed a treatment manual (Brady et al., 2000). Sessions were up to 30 min, with the first session lasting approximately 45 min. 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. The mean dosage at the end of treatment was 115.00 mg/day (*SD* = 78.00). During visits, the psychiatrist monitored side effects and adjusted medication dosage as well as provided general encouragement and support.

Supervision and Treatment Integrity—Treatment sessions were recorded via audio or videotape. PE supervision occurred weekly and included session tape review. Treatment integrity ratings were based on Foa et al. (2005) and Marshall et al. (2001). For each treatment condition, standard fidelity checklists were utilized to assess whether or not the treatment provider delivered required session elements (e.g., delivered an imaginal exposure rationale to the participant at Session 3) or completed protocol violations. Trained outside

raters viewed 10% of tapes. For essential components, PE providers completed 90% and SER providers completed 96%. No protocol violations were observed. PE sessions were rated for therapist competence (e.g., engaged in interactive exchange with patient) on a 3-point scale (1 = *Inadequate*, 3 = *Adequate or Better*). Overall PE therapist competence was very good (M = 2.73, SD = .32).

Treatment Completion—Participants who completed seven sessions or more of either treatment were considered "completers."

Procedure

Participants provided written informed consent during an initial intake interview with an independent evaluator. During this interview, demographic and diagnostic information were obtained. Primary diagnosis of chronic PTSD was determined via the PSS-I, and the SCID-IV was utilized to assess the presence of diagnostic comorbidity. Following this intake, if eligible, participants came for a randomization appointment, where treatment condition was determined. They also completed a physical and laboratory panel (e.g., urine sample) at this visit. Participants also completed a battery of pre-treatment self-report measures including measures assessing severity of PTSD (PSS-SR) and depression (BDI), and current support (ISSB, SRQ, SSQ). Following this visit, patients received 10 weekly sessions of PE or sertraline 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, blind to treatment received, who re-assessed their PTSD symptoms and diagnosis. Participants also completed a battery of self-report measures, including reassessing severity of PTSD (PSS-SR) and depression (BDI).

Defining Depression Sudden Gains—Depression sudden gains were measured as outlined by Tang and DeRubeis (1999). A sudden gain is a rapid, non-linear decrease in depression symptoms over the course of one-between session interval. A sudden gain was said to occur if: 1) the BDI score decreases by at least 7 points between session N and N+ 1. As suggested by Tang and DeRubeis (1999), large-scale psychotherapy trials of depression tend to produce reductions of about 12–15 points on the BDI. In addition, the standard deviation of BDI scores in clinical samples is approximately 7 points (Tang & DeRubeis, 1999). Thus, this criterion of a 7 points likely represents a clinically meaningful shift in symptoms; 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 two pre-or post-gain sessions were available. Sudden gains occurred when all three conditions were satisfied.

Defining Transient Depression Spikes—Depression spikes derived from Hayes et al. (2007) were measured using the same 7-point criteria as defined by Tang and DeRubeis (1999) above, with the exception of the third criterion[1]. Participants were classified as having a transient depression spike if they exhibited a transient increase in BDI symptoms, as defined by a 7-point increase, which subsequently decreased by at least 7 points during the remaining sessions of the 10 weeks of PTSD treatment.

[1]Depression spikes are not simply the opposite of Tang and DeRubeis (1999) sudden gains. The third criterion cannot be applied to the depression spike. Post-spike symptomatology does not have to be significantly lower than pre-spike symptomatology (e.g., 28, 25, **39**, 28, 25 is considered a spike, but would not qualify as such when utilizing the third t-test criterion) (Tang & DeRubeis, 1999).

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. Previous reports examining symptom discontinuities in depression trials use a last observation carried forward (LOCF) imputation method to handle in-session missing data (Tang et al., 2007). This method does not artificially inflate rates of depression fluctuations. Further, the U.S. Food and Drug Administration has traditionally viewed LOCF as the preferred method, considering it likely to be conservative and assuming no change (Hamer & Simpson, 2009).

Results

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

The mean scores for measures of pre-treatment psychopathology severity and social support can be seen in Table 1. In general, this sample of individuals with chronic PTSD showed moderate to severe levels of PTSD symptoms, moderate to severe levels of depression, moderate levels of general support, and relatively low levels of trauma-related support. Further, 54.0% of the sample met criteria for current major depression based on SCID-IV diagnosis. Additionally, 29.5% (n = 59) of the sample met criteria for a current co-occurring anxiety disorder at pre-treatment and 5.5% (n = 11) met criteria for current substance abuse.

Frequency of Depression Symptom Discontinuities over the Course of PTSD Treatment

Sudden gains—First, the presence of depression sudden gains 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 patients (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 reductions (22.7%) met this criterion. Overall, a total of 38 depression sudden gains were experienced by 36 out of 200 participants (18.0%), only two of whom experienced two depression sudden gains during treatment. The mean session of the depression sudden gain was 7.36 (SD = 2.29; Range = 3 – 10) and the average magnitude was 10.42 (SD = 3.22; Range = 7 – 19). Chi-square tests (with Yates continuity correction) indicated that a higher percentage of individuals with current MDD experienced a depression sudden gain[2] (n = 26, 24.1%) than patients without current MDD (n = 10, 10.9%), χ^2 (1, N = 200) = 5.01, p = .03, with no differences between PE and sertraline.[3]

Transient depression spikes—Of the participants in either PE or sertraline, 22.5% (n = 45) of individuals experienced a transient depression spike over the course of treatment. Of those experiencing a transient depression spike, 77.8% (n = 35) experienced one transient spike, and 22.2% (n = 10) experienced two transient spikes. The mean session number of the

[2]Post-hoc analyses examined whether depression sudden gains were associated with reductions in suicidality via Item 9 on the BDI. Item 9 ranges from 0 (*I don't have any thoughts of killing myself*) to 3 (*I would kill myself if I had the chance*). Consistent with the inclusion criteria, the mean score of item 9 in the full sample at pre-treatment was low, M = .46, SD = .55. Among the 36 participants with a depression sudden gain, less than half (n = 14; 38.9%) reported a 2 or greater on the BDI item at the pre-gain session. Of these 14 participants, 7 (50%) of them reported a 2 or greater at the post-gain session. Given the low level of suicidality in this sample and the fact that a sudden gain, by definition, results in a reduction of depressive symptoms (which may include suicidal ideation), it does not appear that a reduction in suicidality is driving these results.

spike was 4.67 (*SD* = 1.98; Range = 2 – 9) and the average magnitude of the spike was 10.78 (*SD* = 5.04; Range = 7 – 28). A similar percentage of individuals with MDD (24.1%, *n* = 26) and without MDD (20.7%, *n* = 19) experienced a transient depression spike during PTSD treatment, χ^2 (1, *N* = 200) = 0.17, *p* = .68, with no differences between PE and sertraline[3].

Overall discontinuities in depression—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 transient depression spike and depression sudden gain over the course of PTSD treatment. In general, it appears that a significant minority of patients with chronic PTSD experience symptom discontinuities in depression symptoms[4].

Pre-treatment Predictors of Depression Discontinuities During Treatment

A series of chi-square analyses were conducted to examine if demographic variables were associated with depression sudden gains or transient depression spikes. Those who did and did not experience a depression sudden gain did not significantly differ from one another on main demographic variables (e.g., age, education, minority status). Similarly, those who did and did not experience a transient depression spike did not differ on these demographic variables.

Pearson correlations were conducted to examine the association between social support variables and the presence of depression symptom discontinuities. See Table 2. Overall, social support was not strongly associated with the presence of a depression sudden gain. Those who experienced higher levels of negative trauma-related support were more likely to experience a transient depression spike (*r* = .18, *p* = .01) during treatment.

Patterns of Depression Symptom Discontinuities in PE vs. Sertraline

There was not a significant difference in the percentage of individuals experiencing a depression sudden gain between PE and sertraline, χ^2(1, *N* = 200) = .96, *p* = .33, with 20.6% of individuals in PE and 14.2% of individuals in sertraline experiencing a depression sudden gain. Similarly, there was not a significant difference in the percentage of individuals experiencing a transient depression spike between PE and sertraline, χ^2(1, *N* = 200) = 0.02, *p* = .89, with 23.3% of individuals in PE and 21.4% of individuals in sertraline experiencing a transient depression spike. Experiencing a transient depression spike was not strongly associated with higher final sertraline dose (*r* = .22, *p* = .06). Similarly, experiencing a depression sudden gain was not strongly associated with end sertraline dose (*r* = .19, *p* = .11).

A series of ANOVAs were conducted to examine if there were differences in timing or magnitude of depression symptom discontinuities between treatments. There were no significant differences on timing [*F*(1, 34) = 0.26, *p* = *.62*] or magnitude [*F*(1, 34) = 0.02, *p*

[3]Chi-square analyses were run separately for each treatment condition, comparing the percentage of participants with and without MDD that experienced a depression sudden gain or transient depression spike. For participants in PE (*n* = 116), 27.6% (*n* = 16) of participants with current MDD experienced a sudden gain in depression and 13.8% (*n* = 8) of patients without current MDD experienced a sudden gain, χ^2 (1, *N* = 116) = 3.36, *p* = .06. In sertraline (*n* = 84), 20% (*n* = 10) of individuals with current MDD experienced a depression sudden gain and 5.9% (*n* = 2) without current MDD experienced a sudden gain, χ^2 (1, *N* = 84) = 2.24, *p* = .13. No significant differences in percentage of those with and without MDD experiencing a transient depression spike in either PE, χ^2 (1, *N* = 116) = .001, *p* = 1.00 or sertraline, χ^2 (1, *N* = 84) = .18, *p* = .67.

[4]Randomization to choice of treatment vs. no choice of treatment did not significantly impact the presence of depression symptom discontinuities. A similar proportion of participants randomized to choice (*n* = 25, 25.7%) and no choice (*n* = 20, 19.4%) experienced a transient depression spike, χ^2 (1, *N* = 200) = 1.16, *p* = *.28*. A similar percentage of participants randomized to choice (*n* = 19, 19.5%) and no choice *(n* = 17, 16.5%) experienced a depression sudden gain, χ^2 (1, *N* = 200) = .32, *p* = *.57*.

= .*90*] between PE (session: *M* = 7.50, *SD* = 2.35; magnitude: *M* = 10.47, *SD* = 3.35) and sertraline (session: *M* = 7.08, *SD* = 2.23; magnitude: *M* = 10.33, *SD* = 3.08) of depression sudden gains. Similarly, no differences were observed between timing [$F(1, 43) = 0.59$, $p = .45$] or magnitude [$F(1, 43) = 0.02$, $p = .88$] of transient depression spikes between PE (session: *M* = 4.48, *SD* = 2.04; magnitude: *M* = 10.87, *SD* = 5.18) and sertraline (session: *M* = 4.94, *SD* = 1.89; magnitude: *M* = 10.64, *SD* = 4.98).

Symptom Discontinuities and their Association with Post-treatment Severity

Treatment completion—In the total sample, a higher percentage of treatment completers (25.0%) than non-completers (3.0%) experienced a depression sudden gain, $\chi^2(1, N = 200) = 14.96$, $p = .001$. Overall, 34 out of the 36 (94.0%) participants who experienced a depression sudden gain completed treatment. A higher percentage of treatment completers (29.0%) than non-completers (7.6%) experienced a transient depression spike, χ^2 (1, $N = 200$) = 12.58, $p = .001$. Overall, 40 out of the 45 (88.8%) patients who experienced a transient depression spike completed treatment. Attending a higher number of total sessions was associated with experiencing a depression sudden gain ($r = .33$, $p = .001$) and a transient depression spike ($r = .29$, $p = .001$).

Post-treatment severity—Analyses were conducted to explore whether or not 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 ($r = -.19$, $p = .02$) and depression severity ($r = -.19$, $p = .02$). Experiencing a transient depression spike was not strongly associated with post-treatment PTSD severity ($r = -.09$, $p = .25$) or depression severity ($r = -.11$, $p = .15$).

Depression discontinuities and post-treatment severity—Path analysis, an extension of the regression model, was used to examine the relationship between pre-treatment symptom severity, depression sudden gains, and post-treatment severity (PTSD or depression) using Mplus software (Muthén & Muthén, 2010). Path analysis was used to examine the comparative strengths of the direct and indirect relationships among this set of variables (Lleras, 2005). We did not examine a path analysis model of whether or not transient depression spikes predicted post-treatment symptom severity, given that Pearson correlations were not significant. Bias-corrected bootstrap confidence intervals were used to test the significance of the total, direct, and indirect effect estimates using a bootstrap sample of 5,000. Specifically, we ran two separate path analyses: one model with post-treatment PTSD severity as the dependent variable and a second model examining post-treatment depression severity as the dependent variable.

Prediction of post-treatment PTSD severity: In this model, we examined pre-treatment PTSD severity and depression sudden gains as independent variables, pre-treatment depression severity was entered as a covariate, and post-treatment PTSD severity was the dependent variable. We also examined whether depression sudden gains mediated the relationship between pre- and post-treatment symptom severity.

The overall model weighted root mean square residual (WRMR) = 0.01, indicating a good fit (Muthén & Muthén, 2010). Lower pre-treatment PTSD severity ($\beta = .36$, $SE = .18$, $p = .05$, 95% CI [.06, .65]) and the experience of a depression sudden gain ($\beta = -4.82$, $SE = 1.17$, $p = .001$, 95% CI [−6.79, −2.90]) significantly predicted lower post-treatment PTSD severity. The covariate, pre-treatment depression severity, did not significantly predict post-treatment PTSD severity ($\beta = .27$, $SE = .15$, $p = .08$, 95% CI [.03, .52]). There were non-significant direct effects of pre-treatment PTSD severity ($\beta = .02$, $SE = .01$, $p = .20$, 95% CI

[−.007, .05.]) and depression severity (β = .02, *SE* = .02, *p* = .23, 95% CI [−.005, .04]) on the experience of a depression sudden gain, indicating that our independent variables were not significantly associated with our hypothesized mediator. Finally, the indirect effect, examining depression sudden gains as a mediator between pre-treatment symptom severity and post-treatment PTSD symptom severity was not significant (β = −.09, *SE* = .09, *p* = .28, 95% CI [−.27, .0.2]). Overall, depression sudden gains during PTSD treatment predict lower post-treatment PTSD severity.

Prediction of post-treatment depression severity: In this model, we examined pre-treatment depression severity and depression sudden gains as independent variables, pre-treatment PTSD severity was entered as a covariate, and post-treatment depression severity was the dependent variable. We also examined whether depression sudden gains mediated the relationship between pre- and post-treatment depression severity.

The overall model was a good fit as indicated by the WRMR < 0.01. Lower pre-treatment depression severity (β = .60, *SE* = .13, *p* = .001, 95% CI [.40, .83]) and the experience of a depression sudden gain (β = −4.13, *SE* = 1.01, *p* = .001, 95% CI [−5.81, −2.49]) significantly predicted lower post-treatment depression severity. There was no direct effect of pre-treatment PTSD severity on post-treatment depression severity (β = −.01, *SE* = .14, *p* = .93, 95% CI [−.25, .22]). The indirect effect, examining depression sudden gains as a mediator between pre-treatment symptom severity and post-treatment depression severity, was not significant (β = −.08, *SE* = .08, *p* = .27, 95% CI [−.24, .02]). Overall, depression sudden gains during PTSD treatment predict lower post-treatment depression severity.

Post hoc Analyses: The Role of High Negative Trauma Support

To examine whether or not depression spikes may negatively impact treatment outcome among individuals with more extreme levels of negative social support, we ran post-hoc correlational analyses to examine the relationship between transient depression spikes and post-treatment severity among participants with high negative trauma-related support. Among those with negative-trauma related support that fell at least one *SD* above the mean (*n* = 36), experiencing a transient depression spike was not strongly associated with number of sessions attended (*r* = .07, *p* = .67), post-treatment PTSD severity (*r* = −.27, *p* = .14) or post-treatment depression severity (*r* = −.33, *p* = .07).

Discussion

Patients with PTSD often present with co-occurring MDD (e.g., Creamer et al., 2001) and the presence of MDD may hinder overall PTSD treatment progress (e.g., Nixon et al., 2004). Recently, a growing body of research has examined the empirical nature of the symptom or diagnostic overlap between anxiety disorders, such as PTSD and depression (e.g., Watson, 2009). The present study was interested in examining depression symptom change among patients with a primary anxiety disorder, namely chronic PTSD. This was the first study to track session-by-session depression discontinuities among individuals with chronic PTSD. About one-third (34.0%) of individuals experienced a rapid fluctuation in their depression symptoms over the course of PTSD treatment, evidenced by either a depression sudden gain (18.0%) or a transient depression spike (22.5%). Rapid fluctuations in depression symptoms occurred at similar rates during both exposure therapy (PE) and pharmacotherapy (sertraline), suggesting that both empirically supported treatments for PTSD are characterized by sudden shifts in depression for a subset of individuals. The experience of a depression sudden gain predicted lower post-treatment PTSD and depression symptom severity. However, depression sudden gains did not mediate the relationship between pre- and post-treatment symptom severity.

On average, both PE and sertraline are effective in treating PTSD (e.g., Bisson & Andrew, 2005; Stein, Ipser, & Seedat, 2006). Contrary to our original hypothesis and despite potentially dissimilar treatment mechanisms, depression symptom patterns were similar between PE and sertraline. Vittengl and colleagues (2005) similarly found that individuals receiving pill placebo, cognitive therapy, or pharmacotherapy for depression experienced sudden gains at similar rates. These findings challenge the notion that Tang and colleagues (1999; 2005) put forth in which sudden symptom improvements result from a rapid shift in cognitive insight. However, it may be that cognitive shifts occur in patients receiving pharmacotherapy as well, despite such shifts not being a treatment target. Alternatively, shared factors between both treatments may be contributing to symptom discontinuities (e.g., therapeutic alliance). Finally, recent evidence suggests that there may be overlap among the neurobiological signaling pathways involved in both fear extinction and mood stabilization (Tronson et al., 2008). Tronson and colleagues (2008) suggest that the mechanisms involved in depression-like behaviors are similar to the mechanisms involved in fear extinction. Thus, both cognitive behavioral therapy, such as PE, and SSRIs, such as sertraline, may target a common neurobiological system (Tronson et al., 2008), partially accounting for similar patterns of depression symptom change between treatments. Although PE and sertraline may exert their effects through distinct mechanisms, these treatment options likely address the same fear circuitry system within the brain.

In this chronic PTSD sample, transient depression spikes were not associated with either better or worse treatment outcome. This is in contrast to the depression literature, which suggests that both transient depression spikes and depression sudden gains are associated with improved outcome (e.g., Drymalski & Washburn, 2011; Hayes et al., 2007; Tang et al., 2005). In treatment of depression, approximately 30–50% of patients experience sudden gains. However, in this sample of patients with a primary diagnosis of chronic PTSD, a smaller portion (18%) experienced a depression sudden gain. Contrary to our original hypothesis, as well as Hayes et al.'s (2007) findings, transient depression spikes were not strongly associated with improved treatment outcome. These dissimilar findings between the PTSD and depression literature suggest that the trajectory of depression symptoms during treatment may be different for those with a primary diagnosis of PTSD. Although 54% of individuals in this sample also met current criteria for current MDD and mean depression scores for the sample were moderate to severe, some patients with mild or no depression may not have had room for improvement in their depression severity, thereby lowering the rates of sudden gains. Also, treatment duration in this study was shorter (10 sessions) than other studies examining symptom discontinuities not allowing for more opportunities for gains or spikes (20 sessions; Tang et al., 2007).

A large body of research suggests that lack of social support is one of the strongest risk factors for PTSD development (e.g., Brewin et al., 2003). However, no previous studies have examined how social support affects depression symptom trajectories during treatment, and few have examined the role of social support in PTSD treatment processes (e.g., Keller, Zoellner, & Feeny, 2010; Price et al., 2013; Thrasher et al., 2010). In the present study, negative trauma-related support, albeit modestly, rather than general support received, positive support, or an objective measure of support, size of social network, was associated with experiencing a transient depression spike. Clinically, it appears that improving the *quality* of how others relate to the trauma survivor surrounding the event itself may influence the processes of change during PTSD treatment. This may be particularly relevant for patients in exposure therapy, who may be encouraged to interact with others, in relation to their traumatic event, for their *in vivo* homework assignments (e.g., increasing intimacy with a significant other).

From a theoretical standpoint, 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., 2007). Previous researchers (Hayes & Strauss, 1998) have urged psychotherapy researchers to increase their focus on rapid destabilizations and transitions to better understand the theoretical nature and underlying mechanisms of psychological change. Depression discontinuities, such as sudden gains and transient spikes, may represent important transitions and reorganizations within the system (Thelen & Smith, 1994; Hayes & Strauss, 1998). Clinically, for patients with PTSD, clinicians should be attuned to both a patient's history and current conditions, as both factors can influence the likelihood of destabilization within a system and an individual's response to the destabilization (Keenan, 2010). Notably, pre-treatment co-occurring MDD was associated with an increased likelihood of experiencing a depression sudden gain. No other demographic or psychopathology factors reliably predicted either sudden gains or transient depression spikes. While this study suggests that pre-treatment social support may play a role in depression symptom discontinuities, there are likely multiple interacting factors that influence the occurrence of a depression sudden gain or transient depression spike during PTSD treatment. Future research should consider examining patterns of social support over the course of treatment as well as examining additional potential predictors of depression discontinuities. For example, future research may examine potential shared factors between treatments as well as additional patient factors (e.g., attachment style, interpersonal life events such as job functioning, presence of Axis II disorders, suicidality, menstrual cycle in females) that may be associated with both the likelihood of symptom fluctuations and response to these fluctuations. That said, for example, if menstrual cycle were a factor in depression discontinuities, we should expect then to see a pattern of spikes roughly on a monthly basis for some participants (e.g., on average, two to three spikes during 10 weeks of treatment), which was not the case. More fine-grained analyses, examining how depression symptom discontinuities map onto background states (e.g., a transient depression spike that occurs in the context of stable, increasing, or decreasing depression) would also be an important future step.

There were a few limitations of the present study. 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. A no-treatment control group was also not included in the present study. Thus, we were unable to examine the pattern of natural fluctuations in depressive symptoms over time. However, a previous examination of depression fluctuations during a no-treatment observation period (Kelly, Roberts, & Bottonari, 2007) among depressed college students indicated that those who experienced sudden gains during the observation period were no less likely to achieve remission at the end of the observation period than individuals without sudden gains. Thus, mechanisms of sudden gains within and outside of treatment may be distinct. Finally, we used the LOCF method to handle missing data. However, this method is considered to be conservative and therefore did not inflate our rates of depression symptom fluctuations. Despite these limitations, the study highlights the role of depression symptom fluctuations during treatment for chronic PTSD.

Encouragingly, depression sudden gains were associated with improved treatment outcome, both for PTSD and depression symptoms. Thus, clinicians may want to be particularly attentive to depression sudden gains for patients with PTSD. 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 spikes were not detrimental to treatment outcome for either sertraline or PE. Research examining therapeutic change processes, including symptom discontinuities, can potentially improve current treatment

options and fill the “gaps” (Bauer, 2007) in evidence based practice by modifying treatment protocols to better fit patients’ individual needs. Specifically, tracking symptom levels over the course of treatment can increase both patient and clinician awareness of progress or lack of progress. Second, for patients who are worried about their symptom spike, clinicians may want to normalize the concern while also reassuring the patient that a temporary increase in symptoms does not necessarily mean that they will benefit less from treatment. Third, by pinpointing exactly when change is occurring, clinicians may be better able to examine what happened either in-treatment (e.g., shift in cognitions, avoidance of in-session material) or outside of treatment (e.g., change in employment status, marital problems) that led to the sudden gain or transient spike in symptoms. Overall, understanding how depression symptoms change during PTSD treatment may help us to better tailor our current PTSD treatments to better fit individual patient needs.

Acknowledgments

Preparation of this manuscript was supported by grants to Drs. Zoellner and Feeny from the National Institute of Mental Health (R01 MH066347, R01 MH066348) and the William T. Dahms, M.D. Clinical Research Unit, funded under the Cleveland Clinical and Translational Science Award (UL1 RR024989).

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Table 1

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

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

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

	Depression Sudden Improvement	Transient Depression Spike
Positive Trauma-Related Support (SRQpos)	.13	.01
Negative Trauma-Related Support (SRQneg)	.03	.18*
General Support (ISSB)	.10	.05
Satisfaction with Current Support (SSQ)	.06	−.10
Number of Social Contacts (SSQ)	.03	.03

Note:

* $p < .05$