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Sudden Gains in Group Cognitive-Behavioral Therapy for Panic Disorder

Elise M. Clerkin, Bethany A. Teachman, and Shannan B. Smith-Janik

University of Virginia

Abstract

The current study investigates sudden gains (rapid symptom reduction) in group cognitive-behavioral therapy for panic disorder. Sudden gains occurring after session 2 of treatment predicted overall symptom reduction at treatment termination and some changes in cognitive biases. Meanwhile, sudden gains occurring immediately following session 1 were not associated with symptom reduction or cognitive change. Together, this research points to the importance of examining sudden gains across the entire span of treatment, as well as the potential role of sudden gains in recovery from panic disorder.

Keywords

sudden gains; anxiety; panic disorder; cognitive bias

Many clinicians have worked with patients who improve tremendously from one session to the next. This abrupt improvement is often surprising for therapists – accompanied by amazement or even disbelief. However, it is increasingly clear that symptoms do not always improve in a gradual, linear fashion. Instead, research suggests that “sudden gains” (i.e., large improvements in symptoms that occur in a single between-session interval) may account for a sizeable percentage of total symptom improvement (Tang & DeRubeis, 1999).

In their influential paper, Tang and DeRubeis (1999) first demonstrated that sudden gains were prevalent in individual cognitive-behavioral therapy (CBT) for depression, accounting for approximately 50% of total improvement. Individuals who experienced a sudden gain (versus those who did not) displayed better outcomes at treatment termination and at 6- and 18-month follow-ups. Furthermore, results indicated that substantial changes in cognition predicted the gains. Since then, a variety of studies have examined sudden gains. While the focus has typically been on sudden gains in individual, time-limited treatment for depression, researchers have also examined gains in the context of group CBT for depression (Kelly, Roberts, & Ciesla, 2005), group CBT for social phobia (Hofmann, Schulz, Meuret, Moscovitch, & Suvak, 2006), and individual treatment in clinical settings for individuals with mixed psychiatric diagnoses (Stiles et al., 2003). In general, studies confirmed the existence of sudden gains, and demonstrated that individuals who experienced sudden gains, when compared to those who did not, had greater overall symptom reduction at treatment termination.

Correspondence concerning this paper should be addressed to Elise Clerkin at the University of Virginia, P.O. Box 400400, Charlottesville, VA 22904-4400. Phone: 434-243-7646, Fax: 434-982-4766, E-mail: emc2t@virginia.edu.

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In the current study, our primary goal is to examine sudden gains in group CBT (CBGT) for panic disorder. To our knowledge, this will provide the first evaluation of the frequency and impact of sudden gains within CBGT for panic. In her unpublished dissertation research, Pham (2006) also investigated sudden gains in panic disorder, but did so in the context of individual treatment. Unfortunately, she did not include a panic-specific symptom measure, but rather used the Anxiety Sensitivity Index (ASI; Reiss, Peterson, Gursky, & McNally, 1986). Sole use of the ASI is not ideal, however, as it is also a measure of cognitive bias and does not assess frequency or intensity of panic. Thus, we include two measures of cognition (including the ASI), as well as a separate measure of panic symptoms.

A secondary goal of this research is to examine whether there are differences between sudden gains occurring at the outset of treatment (i.e., gains occurring between sessions 1 and 2; referred to as Session 1 SG), and gains occurring later in the treatment process (i.e., gains occurring at any point after session 2; referred to as Session 2+ SG). While researchers theorize that early symptom improvement is crucial in treatment outcome (e.g., Ilardi & Craighead, 1994), few studies have examined Session 1 SG. Notable exceptions include Kelly et al. (2005) and Busch, Kanter, Landes, and Kohlenberg (2006), who both found that early gains were prevalent in treatment for depression and were associated with better treatment outcome. However, Kelly et al. failed to find a relationship between sudden gains and cognitive changes and Busch et al. did not examine this relationship. Further, Kelly and colleagues did not compare Session 1 SG to Session 2+ SG. Instead, they compared gains that occurred during the first third of treatment to gains occurring later, finding that individuals experiencing gains in the first third of treatment were more likely to be identified as treatment responders.

These studies point to the potential importance of early gains for treatment outcome, though the meaningfulness of gains that occur after only one session are challenging to interpret. For instance, the initial therapy session in the current study was primarily focused on socializing participants to therapy, engendering hope for recovery, building rapport among group members, and providing psychoeducation and an overview of the primary components of treatment. In contrast, subsequent sessions (including session 2) delved more into the “active” ingredients of therapy. In fact, Tang and DeRubeis (1999) caution against including Session 1 SG in analyses because the content of session 1 is theorized to be so different from the content of other therapy sessions. While we agree that first session gains should likely be analyzed separately, we believe the potential differences between Session 1 SG and Session 2+ SG makes them interesting and worth examining. For instance, while cognitive mediation is theorized to underlie sudden gains occurring after session 2 (Tang & DeRubeis, 1999), it seems plausible that nonspecific factors (e.g., hope about treatment) may play a particularly important role in earlier gains.

Thus, to provide a more comprehensive picture of the role of sudden gains in panic disorder, our objective was to examine gains across the entire 12-week course of treatment. Given the similar treatment foci in CBTs for depression and panic (e.g., cognitive restructuring), we expect that sudden gains occurring after session 2 will be similar to gains found in previous research in terms of prevalence and impact. In particular, individuals who experience a sudden gain any time after session 2 are expected to have greater symptom reduction relative to people who do not have sudden gains. Further, given prior evidence that Session 2+ SG may be preceded by cognitive change (Tang & DeRubeis, 1999), these sudden gains are also expected to predict greater cognitive change from pre-treatment to treatment termination. Unfortunately, we do not have a weekly indicator of change in cognitive biases associated with panic, so are unable to directly test the cognitive mediation hypothesis. However, examination of pre- to post-treatment changes in cognition allows us to draw meaningful inferences about the relationship between sudden gains and changes in cognitive bias.

Our hypotheses regarding gains that occur immediately following session 1 are more exploratory due to the unique nature of the initial treatment session. Given the likely association between these sudden gains and nonspecific (versus cognitive) factors in treatment, it is possible that these gains will function differently from gains occurring after session 2. Specifically, it is plausible that the gains will be less related to changes in cognitive bias. However, following Busch et al. (2006), we tentatively predict that Session 1 SG will predict greater symptom reduction at treatment termination, relative to people who do not have sudden gains.

Method

Participants

Participants ($N=43$) were adult outpatients who participated in a 12 week CBGT intervention for panic disorder. Consistent with past research (e.g., Tang & DeRubeis, 1999; Hofmann et al., 2006), we only included participants who attended greater than seven therapy sessions to ensure that they had received sufficient CBT treatment. In addition, including participants with only seven or fewer sessions could lead to misleading results, as this would inadvertently inflate the percentage of individuals who failed to experience a sudden gain, simply because they did not have the opportunity to do so. This resulted in a final sample of 30 (70.0% female), with a mean age of 40.63 ($SD=14.93$), and an average of 15.67 years of education ($SD=2.51$). Ninety percent reported race or ethnicity as Caucasian, 6.7% as African-American, and 3.3% as 'other.'

All participants had a principal diagnosis of panic disorder (based on the Structured Clinical Interview for DSM-IV diagnoses, SCID-IV; First, Spitzer, Gibbon, & Williams, 1997). Additionally, 56.7% of participants had a current comorbid Axis I diagnosis at intake, including 36.7% with another anxiety disorder and 20.0% with a mood disorder. The mean duration between participants' first panic attack and intake was 177.23 months ($SD=178.96$, $range=2-732$ months). At intake, 53.3% of participants were taking medication for psychiatric problems, and 50.0% were taking medication for physical problems. Details regarding recruitment procedures can be found in Teachman, Smith-Janik, and Saporito (2007).

Treatment

Patients completed a 12-week manualized treatment protocol following the widely-used Panic Control Treatment (Barlow & Craske, 1994), which was modified for group treatment. Treatment involved structured, 90-minute weekly sessions, which focused on four principal areas: 1) psychoeducation, 2) relaxation training, 3) cognitive restructuring, and 4) exposures. There were nine treatment groups composed of 4 to 6 participants, each of which was co-led by a licensed clinical psychologist (the second author) or advanced doctoral students in clinical psychology who received extensive training in the treatment protocol. Sessions were either directly observed or reviewed via audiotape by the second author, who also provided weekly supervision. Six months following treatment termination, participants returned to the laboratory and completed all assessments for a final time.

Measures¹

Panic and Mood Symptoms—The *Panic Disorder Severity Scale* (PDSS; Shear et al., 1997) is a 7-item scale that provides a composite severity score of frequency, distress, and impairment associated with panic attacks (four-point Likert scale format with total scores ranging from 0–28). The PDSS was originally designed as a clinician-administered instrument;

¹These assessments are part of a larger study evaluating a range of cognitive biases in panic disorder (see Teachman et al., 2007). Only those measures relevant to the current hypotheses are reported here.

however, several prior studies have also successfully used the PDSS as a self-report measure (e.g., Otto, Pollack, Penava, & Zucker, 1999). In the current study, we also had participants complete the PDSS in a self-report format, which was modified slightly to provide a definition of “panic attacks” so that participants could more easily complete the scale. In the current study, Cronbach’s alpha ranged from .78–.92. The average alpha across the 13 assessment points was .86.

The *Beck Depression Inventory-II* (BDI-II; Beck, Steer & Brown, 1996) is a 21-item self-report inventory of depression that has good reliability and validity. In the current study, the average alpha across assessment points was .89 (range: .83–.92).

Cognitive Biases—The *Anxiety Sensitivity Index* (ASI; Reiss et al., 1986) is a 16-item scale with adequate psychometric properties that assesses an individual’s concerns about the negative consequences associated with anxiety (e.g., “It scares me when my heart beats rapidly”). The average alpha in the current study was .89 (range: .88–.91).

The *Brief Bodily Sensations Interpretation Questionnaire* (BBSIQ; Clark et al., 1997) provides a measure of catastrophic misinterpretations of bodily sensations. Participants are provided with 14 ambiguous scenarios and asked to *rank* order three alternative explanations for why an event might have occurred. Participants are then asked to *rate* the likelihood of each explanation on a 0–8 scale. Half of the items reflect panic-specific explanations where one could make a catastrophic misinterpretation related to bodily cues. Only the *ratings* for these panic-specific items were used in the current study, to index the tendency to make panic-relevant interpretations of ambiguous situations related to bodily cues. Cronbach’s alpha ranged from .76–.93. The average alpha across assessment points was .87.

Procedure

Participants completed the PDSS at the start of every therapy session to provide a weekly measure of panic symptoms. Assessment of cognitive processing (using the ASI and BBSIQ) and mood (using the BDI-II) were completed at testing sessions held immediately prior to session 1, and then following sessions 3, 6, 9, and 12. All assessments were also completed at 6-month follow-up.

Plan for Analysis: Criteria for Sudden Gains

We applied the domains from Tang and DeRubeis’ (1999) original criteria: “the magnitude of a sudden gain should be large: (a) in absolute terms, (b) relative to depressive symptom severity before the gain, and (c) relative to symptom fluctuations preceding and following the gain” (p. 895).

Criterion A—Following previous research on sudden gains (e.g., Hofmann et al., 2006; Stiles et al., 2003), we used the *reliable change index* (RCI) to compute a gain that is large in absolute terms. We calculated the RCI by taking the average change score across participants between session 1 and the last session (session 12), divided by the standard error of the difference score (see Jacobson & Truax, 1991, p. 14). Using this methodology, we calculated an RCI of 5.9 points on the PDSS (i.e., $PDSS_N - PDSS_{N+1} > 5.9$, where N reflects the session before the sudden gain). This reflects a large change in panic symptoms, because 5.9 points is approximately 22% of the total possible range on the PDSS. Notably, this comprises a relatively larger improvement in symptoms than was required for Tang and DeRubeis’ (1999) original criterion A (i.e., they required an approximately 11% decrease in depression symptoms as assessed by the BDI).

Criterion B—Consistent with Tang and DeRubeis (1999), the gain must represent at least 25% of the pregain session's score (i.e., $PDSS_N - PDSS_{N+1} \geq 25\% PDSS_N$). This ensures that the gain reflects a substantive change in the person's current panic symptoms. Note, both criteria A and B were established by comparing *contiguous* sessions.

Criterion C—To ensure that the gain reflects a relatively stable change, Tang and DeRubeis (1999) used independent t-tests to compare the average of the three symptom scores before the gain with the average of the three symptom scores after the gain. However, this method makes it impossible to examine gains that occurred following the first session of treatment, and this technique has been criticized due to autocorrelation of the data (e.g., Hardy et al., 2005). Thus, we examined two methods to operationalize our third criterion. First, following Kelly et al. (2005), we required the gain to be at least 1.5 SD of the individual's mean PDSS score across all sessions. This allowed us to examine very early sudden gains that occurred after the first session (Session 1 SG). Second, we applied Tang and DeRubeis' original method to gains that occurred after session 2 (Session 2+ SG). To qualify for a gain using this second method, scores in the (up to) three sessions prior to the gain needed to be significantly greater than scores in the (up to) three sessions following the gain. We obtained very similar classifications based on these two methods (only one individual who qualified for Criterion C using the first method did not qualify using the second; similarly, one individual who qualified using the second method did not qualify using the first). Given our interest in examining very early sudden gains, we chose to report results based on the first method.

Results

Sample Characteristics and Comparison of Groups at Baseline

We first examined group differences to ensure that individuals in each SG group (No SG, Session 1 SG, Session 2+ SG) were comparable at baseline on key demographic variables. Chi-square tests revealed that groups did not differ by gender ($\chi^2=1.06, p>.10$), race ($\chi^2=3.48, p>.10$), or whether participants were taking medication for an emotional ($\chi^2=2.16, p>.10$), or physical ($\chi^2=.20, p>.10$) problem. Additionally, a univariate Analysis of Variance (ANOVA) test indicated that there were no significant group differences in age ($F_{(2, 27)}=.08, p>.10, \eta_p^2=.006$).

Frequency and Timing of Sudden Gains

A total of 10 individuals experienced only one SG and three experienced two, for a total of 16 sudden gains. Thus, 43.3% of the sample experienced at least one SG, which is consistent with Kelly et al. (2005) who found 41.9% in CBGT for depression. Among those who experienced a SG, 53.8% experienced a gain following the first session of treatment (Session 1 SG), and 46.2% experienced at least one gain following the second session of treatment (Session 2+ SG)². Therefore, many of our sudden gains occurred early in treatment: 46.7% occurred between session 1 and 2; 26.7% occurred between session 2 and 3; and 6.7% occurred between each of the following sessions: 4–5, 5–6, 8–9, and 10–11.

Reversal of Sudden Gains

Consistent with previous studies (Tang & DeRubeis, 1999), we considered a gain to be reversed if the individual's PDSS score rose by 50% of the gain (from the post-gain score) at any point in treatment following the gain. Using this rather inclusive criterion, 85.7% of individuals in the Session 1 SG group and 33.3% of individuals in the Session 2+ SG group experienced a

²One individual in the Session 1 SG group also experienced a gain later in treatment. However, this individual was included in the Session 1 SG group because their first gain had a larger magnitude than their second. This individual's subsequent SG was not included in analyses.

reversal. However, all of the individuals in Session 2+ SG and 66.7% of the individuals in Session 1 SG who had experienced a reversal returned to within 50% of the sudden gain (from the post-gain score) by the end of treatment, suggesting most gains were ultimately maintained.

Impact of Sudden Gains: Relationship to Symptom Change

To examine whether sudden gains were related to treatment outcome, we first analyzed group differences between individuals who experienced *any* sudden gain (regardless of timing) versus those who did not using a repeated measures ANOVA with one between-subject factor (SG group status: SG versus No SG) and one within subjects-factor (Time: Pre- versus Post-treatment PDSS score). There was not a significant main effect for SG group status ($F_{(1, 27)}=1.37, p>.10, \eta_p^2=.05$). However, there was a significant main effect for Time ($F_{(1, 27)}=102.0, p<.001, \eta_p^2=.79$). As expected, PDSS scores at treatment termination were significantly lower than pre-treatment PDSS scores ($t_{(28)}=8.34, p<.001, d=1.43$). Additionally, as anticipated, there was a significant Status by Time interaction ($F_{(1, 27)}=11.62, p=.002, \eta_p^2=.30$). Follow-up tests to evaluate the interaction indicated that the groups did not differ in PDSS scores at pre-treatment ($t_{(28)}=.02, p>.10, d=.006$), but individuals who experienced a sudden gain (versus those who did not) had significantly lower scores on the PDSS at treatment termination ($t_{(27)}=2.53, p=.02, d=.95$).

Next, to determine whether timing of the sudden gain mattered, a repeated measures ANOVA was conducted with three levels for the between-subjects factor (SG group status: Session 1 SG, Session 2+ SG, No SG). Again, there was not a significant main effect for SG group status ($F_{(2, 26)}=.84, p>.10, \eta_p^2=.06$), but there was a significant main effect for Time ($F_{(1, 26)}=145.6, p<.001, \eta_p^2=.85$). Of primary interest, there was also a significant SG group status by Time interaction ($F_{(2, 26)}=12.47, p>.001, \eta_p^2=.49$). Follow-up tests revealed that the three groups did not differ in PDSS scores at pre-treatment ($F_{(2, 27)}=.13, p>.10, \eta_p^2=.01$), but there was a significant group difference at treatment termination ($F_{(2, 26)}=4.97, p=.02, \eta_p^2=.28$). Specifically, post-hoc Least Significant Difference (LSD) tests revealed that the Session 2+ SG group had significantly lower scores on the PDSS at treatment termination relative to the No SG group ($p=.004$). There was also a non-significant trend for individuals in the Session 2 + SG group to report fewer symptoms at treatment termination than individuals in the Session 1 SG group ($p=.09$). Session 1 SG and No SG were not significantly different at treatment termination ($p>.10$). See Table 1. Given the significant group difference at treatment termination, all subsequent analyses separated the three groups.

To address the concern that individuals experiencing a sudden gain were simply a proxy for treatment responders, we also examined symptom change among individuals who did *not* experience a sudden gain. Among individuals in the No SG group, PDSS scores at treatment termination were significantly lower than pre-treatment scores ($t_{(15)}=5.40, p<.001, d=.87$). Thus, treatment was also effective for many individuals who did not experience sudden gains; however, the response was more gradual and not as strong overall. Furthermore, we examined the effect of SG group status on PDSS scores over the course of treatment, with psychotropic medication use at intake as a covariate, given concerns that medication use may explain the group differences. Again, there was no significant main effect for SG status ($p>.10$), but there was still a significant SG status by Time interaction ($p<.001$), and a significant main effect for Time ($p<.001$). Finally, we conducted a repeated measures ANOVA examining the effect of SG status on PDSS scores at treatment termination, controlling for each participant's largest between-session gain (regardless of SG group status). This provides an especially stringent test of the impact of sudden gains (as opposed to other kinds of gains in treatment), because it evaluates whether the difference in overall change as a function of SG group status can be accounted for by the magnitude of one's largest gain. Even with this stringent control, there was still a significant SG group status by Time interaction ($F_{(2, 25)}=4.86, p=.02, \eta_p^2=.28$).

Together, these results enhance our confidence that the significant interaction between SG group status and Time is reliable given that the effect remained significant even with these additional checks.

Impact of Sudden Gains: Relationship to Cognitive Biases

To examine whether SG status predicts changes in cognitive processing, we examined the effects of SG Status on two forms of cognitive bias: panic-relevant threat appraisals (assessed with the ASI) and catastrophic misinterpretations of bodily sensations (assessed with the BBSIQ).

We first examined SG status on panic-relevant threat appraisals by conducting a repeated measures ANOVA with SG group status as the between-subjects factor and Time (Pre-Treatment versus Treatment Termination ASI scores) as the within-subjects factor. As expected, there was a significant main effect for Time, with fewer overall threat appraisals endorsed at treatment termination ($F_{(1, 26)}=25.88, p<.001, \eta_p^2=.50$). Additionally, there was a non-significant trend for SG group status ($F_{(2, 26)}=2.65, p=.09, \eta_p^2=.17$), and a significant Time by SG group status interaction ($F_{(2, 26)}=5.95, p=.007, \eta_p^2=.31$). Follow-up tests indicated that there were no group differences on the ASI at pre-treatment ($F_{(2, 27)}=.12, p>.10, \eta_p^2=.009$), but there was a significant group difference on the ASI at treatment termination ($F_{(2, 26)}=9.49, p=.001, \eta_p^2=.42$). Post-hoc LSD tests indicated that the Session 2+ SG group had significantly lower scores on the ASI at treatment termination than individuals in the Session 1 SG ($p=.003$) and No SG ($p<.001$) groups, who did not differ from one another. Notably, the Time by SG group status interaction remained significant even when controlling for PDSS symptom severity at treatment termination ($F_{(2, 25)}=6.11, p=.007, \eta_p^2=.33$).

A second repeated measures ANOVA was then conducted to examine catastrophic misinterpretations, with SG group status as the between subjects factor and Time (Pre-Treatment versus Treatment Termination BBSIQ scores) as the within-subjects factor. Again, there was a significant main effect of time ($F_{(1, 24)}=11.18, p=.003, \eta_p^2=.32$), with participants reporting fewer catastrophic misinterpretations at treatment termination compared to pre-treatment. However, there was no significant SG group status effect ($F_{(2, 24)}=.452, p>.10, \eta_p^2=.04$), nor a significant Time by SG group status interaction ($F_{(2, 24)}=1.12, p>.10, \eta_p^2=.09$).

Impact of Sudden Gains: Six-month follow-up

We examined 6-month follow-up data for PDSS, ASI, and BBSIQ scores to investigate the longer term impact of sudden gains. Because power was more limited for these tests due to attrition, these analyses are not included here but are included in the Appendix for the interested reader. Briefly, individuals in the Session 2+ SG group (relative to the other two groups) displayed lower scores on the PDSS, ASI, and BBSIQ at 6-month follow-up. However, these group differences only reached significance for the ASI scores.

Discussion

In the present study, we examined sudden gains in group CBT for panic disorder. Our goals were to: 1) characterize sudden gains in panic disorder; 2) explore whether sudden gains differ as a function of when in treatment they occur; and 3) determine whether sudden gains are associated with cognitive changes. We found that gains occurring after session 2 occurred in 20% of our overall sample and were associated with better symptom outcomes at treatment termination. This finding adds to the mounting evidence that sudden gains are a general phenomenon in treatment that are found across a variety of disorders. A novel finding from the current study was that gains occurring immediately following session 1 were as prevalent

as later gains, but they did not predict overall symptom reduction. Moreover, in contrast to sudden gains occurring after session 2, gains occurring after session 1 were not associated with greater changes in cognitive biases.

The differences between Session 1 SG and Session 2+ SG may help to inform the presently unresolved questions regarding whether sudden gains are clinically meaningful (see Hofmann et al., 2006), and whether their timing is important (Busch et al., 2006). In contrast to Busch et al. (2006), we did not find a relationship between Session 1 SG and either symptom reduction or cognitive changes. This discrepancy may be due to differences between depression and panic. Although speculative, it is possible that certain nonspecific factors related to very early sudden gains (e.g., engagement in an activity, such as therapy) are more integral in treating depression, relative to panic. Alternatively, given the high level of reversals among our Session 1 SG group, the most parsimonious explanation may be that Session 1 SG were simply reflective of volatile symptom fluctuation in our sample.

In contrast, we found that Session 2+ SG were relatively stable and had predictive validity. We cannot verify what caused these sudden gains, though suspect that changes in threat appraisals may have played a role given the strong emphasis on cognitive restructuring in treatment, and the association between Session 2+ SG and changes on the ASI. Further, spontaneous recovery is not typically characteristic of panic disorder (Chambless & Gillis, 1993), so it seems unlikely that sudden gains following session 2 represent random symptom fluctuation (although see Bruce et al., 2006, for a discussion of the prospective course of panic disorder relative to other anxiety disorders). Nevertheless, it is curious why Session 2+ SG were *not* associated with changes in interpretation bias (on the BBSIQ). One explanation is that the ASI, which assesses a patient's fear of panic symptoms (i.e., fear of fear), taps both *symptoms* and *cognitions* (see Taylor, 1999), leading to closer ties with the PDSS. Alternatively, a floor effect may have contributed to the null finding given that there was generally low endorsement of catastrophic interpretations on the BBSIQ following treatment (see Table 1). It is also worth mentioning that Hofmann et al. (2006), who examined sudden gains in group treatment for an anxiety disorder, found no relationship between cognitive changes and sudden gains. Given the contradictory evidence regarding whether Session 2+ SG predict changes in cognition, future research is necessary to determine the extent that sudden gains will be associated with different types of cognitive change in panic disorder, and whether sudden gains are cognitively mediated.

Moreover, *why* sudden gains (as opposed to gradual symptom reduction) predict more positive symptom outcomes remains an important empirical question. We suspect this occurs because of the changes in self-efficacy that follow a large, dramatic improvement, which likely engenders hope for further recovery, and enhances commitment to the therapy. In fact, the sudden gain itself may confer a critical belief change regarding the patient's ability to overcome symptoms of panic.³ In this sense, mediation by so-called, non-specific factors and cognitive mediation are not at odds with one another, as both are potentially related to important changes in beliefs. Accordingly, we believe that a variety of factors were likely related to the predictive validity of sudden gains, including belief changes tied to panic-specific factors (e.g., fear-of-fear), as well as non-specific factors (e.g., possibility for future change given a SG).

Finally, it is worth mentioning that the vast majority of sudden gains in the current study occurred very early in treatment. This is different than some other studies where sudden gains occurred most frequently at session 5 (e.g., social anxiety, Hofmann et al., 2006; depression, Tang & DeRubeis, 1999). However, this finding is largely consistent with Pham (2006), who found that session 2 was the modal pre-gain session among a group of patients undergoing

³Thank you to an anonymous reviewer for this useful suggestion.

individual CBT for panic disorder (she did not look at gains occurring between session 1 and 2). It is possible that there is something unique about panic disorder treatment that allows large gains to occur very rapidly. In particular, during session 2, participants learned about the adaptive nature of the fight or flight response and were taught to apply the “fear of fear” model to their own panic attacks to learn that they no longer needed to catastrophically misinterpret their physical sensations. This intervention is at the heart of the cognitive model for panic, and we speculate that this learning experience may be very powerful for the many individuals who have suffered with biased assumptions about panic for years, thus resulting in the early sudden gains.

Limitations and Conclusions

The present results should be interpreted in light of several limitations. First, while some of our effects were quite large and our sample was similar in size to several past studies evaluating sudden gains (see Tang, Luborsky, & Andrusyna, 2002; Kelly et al., 2005), our sample was small overall so some of our tests had very limited power. Given this, null findings need to be interpreted with considerable caution. Along these lines, we were unable to evaluate other session groupings in the current study (e.g., combining session 1 and 2 sudden gains versus sudden gains at other points in treatment, etc.) due to power limitations, but we believe this would be an interesting avenue for future research. Additionally, we did not have a weekly or within-session indicator of cognitive bias. Therefore, it is not possible to directly address the critical question of whether cognitive change mediates symptom change. Furthermore, without a waitlist control, we are unable to determine whether these findings resulted from the specific intervention, or if they would have occurred naturally over time. Finally, it is difficult to know the extent to which our findings will generalize to alternate modalities for examining symptom outcomes beyond self-report. However, it is important to note that the clinician-administered Global Assessment of Functioning at intake was significantly related to the self-reported PDSS scores at session 1 in the current study ($r = -.46, p = .01$). Additionally, in previous research we found that the current version of the PDSS was significantly related to multiple measures of cognitive processing (Teachman et al., 2007).

Despite these limitations, this study provides valuable time course information about CBGT for panic disorder. Sudden gains were prevalent both at the very beginning of therapy (immediately following session 1), as well as later in treatment (following session 2). However, only gains following session 2 were associated with better treatment outcomes and change in cognitive bias at treatment termination. These differential outcomes indicate that gains following session 1 may reflect less meaningful symptom fluctuations, while gains occurring after session 2 are perhaps more influenced by the active ingredients of cognitive-behavioral therapy. Regardless, the sheer number of sudden gains that occurred before the third session of treatment underscore the impact of these very early sessions. Together, this research points to the importance of examining sudden gains across the entire span of treatment, as well as the potential role of sudden gains in recovery from panic disorder.

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Appendix: Impact of Sudden Gains at 6-month Follow-up

At 6-month follow-up, the majority of participants maintained their improvement in symptom reduction on the PDSS. A 3 (SG group status) by 2 (Time: Treatment Termination versus 6 month follow-up) repeated measures ANOVA revealed no significant main effect for Time ($F_{(1, 24)}=.37, p>.10, \eta_p^2=.02$) and no significant Time by SG group status interaction ($F_{(2,24)}=.38, p>.10, \eta_p^2=.03$). Although there was a non-significant trend for SG group status ($F_{(2,24)}=3.01, p=.07, \eta_p^2=.20$), this was largely driven by the significant group differences at treatment termination described above. Indeed, a follow-up ANOVA revealed that the group differences in PDSS scores at 6-months follow-up did not reach significance ($F_{(2, 24)}=1.97, p=.16, \eta_p^2=.14$). [Note: A chi-square test revealed that there was no difference in likelihood of having sought further therapy between treatment termination and the 6-month follow-up as a function of sudden gain group ($\chi^2=1.70, p>.10$)].

Next, we evaluated 6-month follow-up outcomes for ASI scores. Specifically, a 3 (SG group status) by 2 (Time: Treatment Termination versus 6-month follow-up) repeated measures ANOVA indicated that there was no significant main effect for Time ($F_{(1, 24)}=1.69, p>.10, \eta_p^2=.07$) or Time by SG group status interaction ($F_{(2, 24)}=.06, p>.10, \eta_p^2=.005$). However, there was a significant main effect for SG group status ($F_{(2, 24)}=8.73, p=.001, \eta_p^2=.42$). In conjunction with the significant group differences at treatment termination discussed earlier, a univariate ANOVA indicated that there was also a significant main effect for SG group status at 6-month follow-up ($F_{(2, 24)}=6.06, p=.007, \eta_p^2=.34$). Specifically, individuals in the Session 2+ SG group displayed lower scores on the ASI relative to individuals in the No SG group ($p=.002$), and individuals in the Session 1 SG group ($p=.01$). The No SG and Session 1 SG groups did not differ significantly from one another ($p>.10$).

Finally, we examined 6-month follow-up for BBSIQ scores, as a function of SG group status. Again, we conducted a 3 (SG group status) by 2 (Time: Treatment Termination versus 6-month follow-up) repeated measures ANOVA. There was not a significant main effect for SG group status ($F_{(2, 22)}=.62, p>.10, \eta_p^2=.05$), nor was there a significant main effect for Time ($F_{(1,22)}=.41, p>.10, \eta_p^2=.02$). Interestingly, there was a non-significant trend for an interaction between Time and SG group status ($F_{(2, 22)}=3.07, p=.07, \eta_p^2=.22$). To further explore this interaction, we examined 6-month follow-up BBSIQ scores as a function of SG group status (recall that there was not a significant SG group difference in BBSIQ scores at treatment termination). At 6-month follow-up, the mean BBSIQ score for individuals in the Session 2+ SG group was lower than the mean scores for individuals in the other two groups, consistent with the results for the ASI (noted above), though this difference did not reach significance ($F_{(2, 24)}=2.17, p=.14, \eta_p^2=.15$). Also, within the Session 1 SG group, scores at 6-month follow-up were significantly worse than at treatment termination ($t_{(24)}=4.47, p=.007, d=1.24$), suggesting this group did not retain their gains in healthier interpretations, whereas the other two groups did maintain their gains (both $p>.10$).

As a whole, it is difficult to draw strong conclusions about the long-term predictive validity of sudden gains, because most people maintained their treatment gains, leaving little variability from treatment termination to 6-month follow-up. Further, we believe these results should be interpreted cautiously because power was more limited for these tests due to attrition at the 6-month follow-up assessment (we lost 2 individuals who were in the No SG group, and one who was in the Session 2+ group). At the same time, the ASI findings in particular add further credibility to the idea that sudden gains occurring in session 2 or later may be meaningfully related to long-term treatment outcome.

Table 1

Descriptive Statistics by Group

Pre-treatment	Full Sample		No SG		Session 1 SG		Session 2+ SG	
	M	SD	M	SD	M	SD	M	SD
Panic Disorder Severity Scale (Total Score)	11.83	4.12	11.82	5.14	11.29	2.29	12.50	2.59
Anxiety Sensitivity Index (Total Score)	29.27	11.23	30.06	12.11	29.00	13.77	27.36	5.31
Brief Bodily Sensations Interpretation Questionnaire (Mean Item Rating)	2.29	1.98	2.46	2.40	2.47	1.66	1.57	.36
Beck Depression Inventory -II (Total Score)	11.42	9.38	13.36	9.85	11.21	10.00	6.17	5.81
Treatment Termination	Full Sample		No SG		Session 1 SG		Session 2+ SG	
Panic Disorder Severity Scale (Total Score)	M	SD	M	SD	M	SD	M	SD
Anxiety Sensitivity Index (Total Score)	5.76	4.18	7.38	4.19	5.43	3.82	1.83	.68
Brief Bodily Sensations Interpretation Questionnaire (Mean Item Rating)	20.83	10.46	24.75	7.51	23.00	11.05	7.83	6.21
Beck Depression Inventory -II (Total Score)	1.33	1.15	1.59	1.39	1.10	.42	.80	.63
6-month follow-up	Full Sample		No SG		Session 1 SG		Session 2+ SG	
Panic Disorder Severity Scale (Total Score)	5.36	4.84	5.09	3.97	8.14	7.01	2.83	2.48
Anxiety Sensitivity Index (Total Score)	M	SD	M	SD	M	SD	M	SD
Brief Bodily Sensations Interpretation Questionnaire (Mean Item Rating)	5.93	4.70	6.83	5.03	6.57	4.08	2.30	3.15
Beck Depression Inventory -II (Total Score)	18.70	9.58	21.87	9.77	20.00	2.00	7.40	7.54
	1.35	.82	1.36	.90	1.73	.49	.78	.71
	6.49	7.16	6.74	8.59	8.71	5.12	2.60	2.88