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## Sudden gains in cognitive behavioral treatment for depression: when do they occur and do they matter?

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### Abstract

Despite the use of efficacious treatments for depression, individuals differ in both the degree to which they recover and the rate at which recovery occurs. Tang and colleagues found that depressed patients who had sudden improvements in their symptomatology not only maintained these gains, but also enjoyed more improvement and higher rates of recovery than those without sudden gains (*J. Consulting Clin. Psychol.* 67(6) (1999) 894; *J. Consulting Clin. Psychol.* 70(2) (2002) 444). Our study examined the role of sudden gains in a cognitive-behavioral group treatment for depression. Results indicated that 41.9% of patients experienced sudden gains. Furthermore, sudden gains occurring in the first third of treatment appear to have special importance. Participants enjoying early sudden gains had significantly larger changes in depressive symptom scores over the course of treatment than those without sudden gains and were marginally more likely to be treatment responders compared to those without early sudden gains. In contrast to Tang and DeRubeis (*J. Consulting Clin. Psychol.* 67(6) (1999) 894), however, sudden gains were not associated with cognitive changes.

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## 1. Introduction

It is often assumed that psychological interventions for depression lead to a steady, gradual improvement in symptomatology over the course of treatment. Although researchers are aware that the severity of individuals' depressive symptoms often fluctuates throughout treatment, it is commonly thought that these fluctuations reflect random variability centered around a gradual linear decline in severity. However, recent research has demonstrated that for a sizable percentage of clients, this assumption of steady, linear change is erroneous (Tang & DeRubeis, 1999). In this research, Tang and DeRubeis found that 37.3% of depressed patients experienced sudden large improvements (sudden gains) that accounted for 50% of their total improvement over the course of treatment. More importantly, they discovered that these sudden gains were typically maintained over time and were associated with more favorable treatment outcomes. Thus, for these clients, sudden gains were neither random nor clinically meaningless.

Tang and DeRubeis (1999) also explored possible mechanisms that may drive these sudden gains. Drawing from the cognitive mediation model of cognitive-behavioral therapy (CBT), Tang and DeRubeis hypothesized that improvements in negative thinking patterns should precede sudden gains in depression. They argued that shifts in cognitive style from negative, depressogenic thinking would result in rapid, precipitous improvements in depressive symptomatology. In fact, Tang and DeRubeis (1999) found that sudden gains frequently followed substantial cognitive changes evidenced in the previous session. They concluded that cognitive changes likely caused these sudden gains and prompted a "positive spiral" leading to superior gains over the course of treatment. Later research, however, showed that sudden gains also occur in non-cognitive treatments for depression, although these gains did not predict better outcomes at 6 months post-treatment whereas those found in cognitive-behavioral interventions did (Tang, Luborsky, & Andrusyna, 2002). Given these mixed findings, it seems important to explore factors associated with sudden gains more closely. In particular, it may be that individuals who enjoy sudden gains in treatment for depression are those with more adaptive cognitive styles to begin with. As such they may have more flexibility in their thinking patterns initially which better prepare them for making changes regardless of the type of treatment they are given.

The current study sought to replicate the findings of Tang and his colleagues in a sample of depressed outpatients participating in a cognitive-behavioral, psychoeducational group treatment for depression. First, we examined the frequency of sudden gains in our treatment sample and their association with outcome. In addition, we examined the pattern of sudden gains to determine the role early sudden gains may have in eventual treatment outcome. In their review, Iardi and Craighead (1994) suggest that early gains in treatment may be instrumental in treatment outcome, as improvement in the first 4 weeks of treatment accounted for 60–70% of total improvement in the studies they reviewed. As such, we were especially interested in exploring the impact of early sudden gains on treatment outcome. We hypothesized that sudden gains occurring early in treatment may be especially related to treatment outcome in that they may provide the impetus and improved functioning to continue to comply with treatment. Conversely, not experiencing a marked improvement in symptoms early in treatment may act to confirm hopelessness thus complicating treatment. Lastly, our sample represented a unique opportunity to examine the role of cognitive changes in sudden gains. Our protocol called for the systematic presentation of behavioral and cognitive skills in a set order. As such, it was possible to examine the impact of

potential cognitive changes on sudden gains to further evaluate Tang and DeRubeis' hypothesis that cognitive change impacts sudden gains. Furthermore, data were collected on several measures of cognitive style before treatment and self-esteem was evaluated on a weekly basis throughout treatment. We were therefore able to examine the relationship between cognitive style and sudden gains to determine if particular cognitive styles are associated with having a sudden improvement in depressive symptoms.

## 2. Method

### 2.1. Participants

Participants were 31 non-psychotic adult outpatients (61.3% female;  $n = 19$ ) seeking treatment for depression. All had a primary diagnosis of current major depressive disorder (based on the mood module of the structured clinical interview for DSM-IV diagnoses; SCID; First, Spitzer, Gibbon, & Williams, 1997) and an initial Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979; Beck, Steer, & Brown, 1996) score greater than or equal to 15. The sample was predominantly Caucasian ( $n = 30$ ) and relatively well-educated. Twenty-five percent of the sample ( $n = 8$ ) had some post-baccalaureate education or held advanced degrees, 10 (32.3%) were college graduates, 11 (35.5%) had some college education and 2 (6.5%) were high-school educated. The majority of participants were single ( $n = 16$ ; 51.6%), 10 were married (32.3%) and five were divorced (16.1%). Mean age of participants was 41.6 years (range 23–75). Nine participants (29.0%) were receiving concurrent pharmacotherapy, two were (6.5%) were receiving concurrent psychological treatment, and six (19.4%) were receiving both concurrent psychological treatment and pharmacotherapy.

### 2.2. Measures

Participants completed the Beck Depression Inventory (BDI; Beck, Steer, Ball, & Ranieri, 1996; Beck, Steer, & Brown, 1996) both pre- and post-treatment, as well as on a weekly basis prior to each group meeting. The BDI is a widely used self-report measure of depressive severity and has been shown to have strong psychometric properties. Due to a change in protocol, the first 21 participants received the BDI while the remainder received the BDI II<sup>1</sup>. Depressive severity was assessed at each session and participants' first two and last two scores were averaged to create initial (Time 1) and termination (Time 2) scores used in analyses. These aggregates were used in order to increase the reliability of our measurement of depressive severity (see Roberts, Shapiro, & Gamble, 1999; Ciesla & Roberts, 2002).

Participants were also asked to complete several measures of cognitive style. These included the Attributional Style Questionnaire (ASQ; Peterson et al., 1982), a measure of depressive cognitive style associated with the hopelessness theory of depression, the Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978), a measure of the cognitive style hypothesized by Beck to related to

<sup>1</sup>We converted BDI-II to BDI scores based on item-level norms (Beck et al., 1996 Beck, Steer, Ball, & Ranieri, 1996). For ease of communication, we will refer to both the BDI and the BDI II as the "BDI".

vulnerability to depression, the Rosenberg Self-esteem Scale (RSE; Rosenberg, 1979), a widely used measure of global self-esteem, and the Beck Hopelessness Scale (BHS; Beck, Weissman, Lester, & Trexler, 1974). The ASQ is a widely used measure of the depressive cognitive style associated with the hopelessness theory of depression. It has shown good internal reliability across attributions (Chronbach's alpha = .72 for negative events) and strong test–retest correlations ( $r = .64$  for negative events; Peterson et al., 1982). In this protocol, participants only rated negative events. The DAS is a measure of the cognitive style hypothesized by Beck to contribute to the onset and maintenance of depression. It is commonly used and has demonstrated good test–retest reliability as well as good internal consistency and validity (Oliver & Baumgart, 1985). The RSE is a widely used measure of global self-esteem that has been reported to have good reliability as well as moderate to strong convergent and divergent validity (Rosenberg, 1979). The BHS was developed as a measure of pessimism and has been shown to have good reliability (coefficient alpha = .93) and good convergent validity with clinician ratings and other measures of hopelessness (Beck et al., 1974). Each measure was administered prior to treatment. The RSE was administered weekly, prior to each group meeting.

### 2.3. Procedure

Participants were initially assessed on the mood module of the SCID, the BDI, and measures of cognitive style at a pre-treatment session. SCID interviews were conducted by trained graduate students. Analyses conducted on a subset of the larger sample indicated reliability coefficients of .69 for MDD, .64 for dysthymic disorder, and 1.0 for bipolar disorder. These coefficients are consistent with reliability scores found in a study conducted by the authors of the SCID (Williams, Gibbon, First, & Spitzer, 1992).

After initial assessment, participants attended up to 12 sessions of group treatment for depression based on Lewinsohn's "Coping With Depression" course (Lewinsohn, Antonuccio, Steinmetz, & Teri, 1984). This manualized treatment focuses on skill-building to ameliorate depression and prevent relapse. Skills include relaxation, increasing pleasant activities, cognitive restructuring, and social skills training. Treatment was administered in 12 weekly 2 hour sessions co-led by trained clinical psychology graduate students. The first two sessions concentrated on providing participants with the treatment rationale and educating them about a behavioral approach to depression (session 1) as well as providing information on how to make and carry out a self-change plan (session 2). Following that, participants were taught the 4 core skills in the order stated above. Each skill was the focus of two consecutive sessions (weeks 3–10). Following the skill components, participants discussed means of maintaining their gains (week 11) and long-term goals (week 12). Participants had to attend at least 6 of the 12 treatment sessions to be included in the current study. The average number of sessions attended by individuals in the current sample was 10.9.

### 2.4. Criteria for sudden gains

Tang and DeRubeis (1999) proposed three criteria to identify sudden gains over the course of treatment. They proposed that the change should be substantial in three ways: in absolute terms (more than 7 BDI points), relative to the previous score (more than 25% of the previous session BDI score), and relative to symptom fluctuations around the gain (significantly higher mean in the

three pre-gain scores than in the three post-gain scores). Because the third criterion makes it impossible to evaluate changes between the first and second sessions, potentially eliminating very early sudden gains, we modified the third criteria. Our third criterion requires that a gain must reflect at an improvement of at least 1.5 standard deviations from the individual mean so that it does not simply reflect the individual's normal variation. This modification allowed us to include sudden gains that occurred very early in the course of treatment.

### 3. Results

#### 3.1. Preliminary analyses

The average participant improved by 9.2 BDI points over the course of treatment,  $t(30)=6.0$ ,  $p<.001$ ,  $d=0.98$ . The T1 mean BDI was 25.1 ( $sd=7.3$ ), while the T2 mean BDI was 15.8 ( $sd=11.7$ ). In addition, we considered a 50% reduction in depressive symptomatology to be indicative of treatment response. Forty-two percent of the participants enjoyed at least a 50% reduction in their BDI scores from T1 to T2 and 58% of the participants scored below 15 on the BDI at T2.

Analyses were conducted to determine if demographic, clinical and cognitive variables were associated with either initial or termination BDI scores. *T*-tests were conducted on categorical variables, such as gender and depressive sub-type, and correlational analyses were conducted with continuous data. Because these analyses were exploratory in nature, two-tailed tests were used. Education level, gender, number of previous depressive episodes,<sup>2</sup> duration of index episode in months, melancholic sub-type were not significantly associated with initial BDI scores nor with termination BDI scores ( $p$ 's ranged from .15 to .98). Age was marginally correlated with Time 1 BDI score ( $r=-.32$ ,  $p<.08$ ), but not with Time 2 BDI. Chronically depressed individuals had higher Time 2 BDI scores compared to those without chronic depression (22.5 vs. 12.6),  $t(28)=2.26$ ,  $p<.05$ ,  $d=.84$  though they were not statistically different at Time 1 ( $p=.56$ ,  $d=.24$ ). Interestingly, although those with recurrent depression did not differ at Time 1 from those with single episodes ( $p=.66$ ,  $d=.18$ ), they showed *lower* BDI scores at termination (12.2 vs. 21.4),  $t(28)=2.20$ ,  $p<.05$ ,  $d=.81$ . Lastly, we examined if concurrent treatment, either pharmacotherapy or other psychotherapy, was associated with depression severity. Although those in concurrent psychotherapy had higher Time 1 BDI scores (27.8 vs. 20.0),  $t(12)=2.21$ ,  $p<.05$ ,  $d=1.28$  they did not reliably differ at Time 2 ( $p=.35$ ,  $d=.51$ ). Pharmacotherapy was not associated with depression severity at either Time 1 or Time 2 ( $p$ 's=.31 and .36, respectively,  $d$ 's=.40 and .35, respectively).

#### 3.2. Sudden gains

We identified 15 sudden gains occurring to 13 participants. Two participants had a sudden gain early in the treatment and a second change that met criteria for a sudden gain in the middle part of

<sup>2</sup>Because estimates beyond 6 previous episodes may be unreliable, we collapsed individuals reporting 6 or more previous episodes into a single category.

treatment. Sudden gainers accounted for 41.9% of our participants, comparable to the 37.3% in Tang and DeRubeis (1999) and 43% in Tang et al. (2002). Examination of the pattern of sudden gains across sessions indicated that the majority of sudden gains took place early in treatment. Two-thirds (10) of all sudden gains occurred between the first and 5th treatment sessions (i.e., in the first third of treatment). An additional five sudden gains occurred in the middle third of the intervention and no sudden gains occurred in the last third of treatment. Following Tang and DeRubeis (1999) we considered a sudden gain to be reversed if the individual's BDI score rose by 50% of the gain at any point after the gain. Using this criteria, seven of the 13 individuals had reversals and four of those seven returned to within 50% of their sudden gain by the end of the treatment.

We conducted one-way ANCOVAs controlling for initial BDI scores in order to determine if those with sudden gains fared better at treatment termination than those who did not have sudden gains. In addition,  $\chi^2$  analyses were conducted to determine if the presence of sudden gains was associated with treatment response status. As stated above, we considered those with a 50% reduction in BDI scores over the course of treatment to be treatment responders. Because previous research indicates that those with sudden gains perform better at treatment end, we conducted one-tailed tests to assess this hypothesis. In contrast to Tang and DeRubeis' (1999) findings, results indicated that participants who had sudden gains did not have better outcomes than participants without sudden gains. Although sudden gainers showed larger improvements in BDI scores than others over the course of treatment (by an average of 3.7 points), a one-way ANCOVA showed that these differences were not statistically significant,  $F(1, 28) = 1.31, p = .13, \eta^2 = .01$ , one-tailed test. Likewise, those with sudden gains were no more likely to be treatment responders than those without sudden gains (53.8% vs. 33.3%),  $\chi^2(1, N = 31) = 1.30, \phi(30) = .21, p = .13$ , one-tailed test. As a point of comparison, Tang and DeRubeis (1999) found that 79% of gainers and 41% of non-gainers were treatment responders.

When those with early gains in the first third of treatment were compared to those who did not have early gains, early sudden gainers had significantly greater symptom reductions by the end of treatment when initial BDI score was controlled,  $F(1, 28) = 4.40, p < .05, \eta^2 = .03$ , one-tailed test (mean BDI change of  $-13.6$  vs.  $-7.2$ ). Likewise they fared marginally better in terms of treatment response (60.0% vs. 33.3%),  $\chi^2(1, N = 31) = 1.98, \phi(30) = .25, p = .08$ , one-tailed test.

A further analysis was conducted in which individuals who had a sudden gain in the first third of treatment but not at any other point in the course of treatment were compared to all other participants. Individuals who experienced a sudden gain during the first third of treatment and not later showed greater reductions in depressive symptoms controlling for initial BDI scores compared to those without sudden gains or those who had sudden gains later in treatment,  $F(1, 28) = 3.30, p < .05, \eta^2 = .02$  one-tailed test (mean decrease in BDI of 13.8 vs. 7.7 points). Furthermore, individuals with exclusively early sudden gains were more likely to be treatment responders (75%) compared to those without sudden gains or those who had gains later in treatment (30.4%),  $\chi^2(1, N = 31) = 4.84, \phi(30) = .40, p < .05$ , one-tailed test.<sup>3,4</sup>

<sup>3</sup>We were interested in performing similar analyses on those participants who had sudden gains exclusively in the middle third of treatment, but because only three participants had such a gain, these analyses were judged to be inappropriate due to small sample size.

<sup>4</sup>Of the five individuals with middle sudden gains, four experienced relatively small shifts in BDI scores that just met

Additionally it is interesting to note that, despite Tang and DeRubeis' (1999) original hypothesis that cognitive change was driving these sudden and lasting gains, participants who had early gains did not receive instruction in cognitive techniques prior to those gains. These techniques are not addressed until the 7th and 8th sessions in this treatment protocol. Participants with early gains had been instructed in behavioral self-change and relaxation techniques.

### 3.3. Does change in self-esteem precede early sudden gains?

To further explore Tang and DeRubeis' (1999) hypothesis that sudden gains were related to changes in cognitive style, we examined changes in RSE scores (as a proxy for cognitive change) prior to sudden gains to determine if they were related to such gains. Tang and DeRubeis (1999) performed similar analyses by coding pre-gain and "pre-pre-gain" session videotapes to determine if significant cognitive changes had occurred in these sessions. They hypothesized and found that the pre-gain sessions showed greater cognitive changes than the pre-pre-gain sessions, which acted as control sessions. Although we were not able to examine within-session changes in cognition, we were able to examine changes in the weekly RSE for the pre-gain session and the pre-pre-gain session among sudden gainers. Results indicated no statistically significant differences between these two sessions in paired sample *t*-tests regardless of which subgroup of gainers (any gain, early gains, only early gains) was used (*t*'s ranged from 0.06 to 0.52, *p*'s ranged from .31 to .48, one-tailed tests, *d*'s ranged from .01 to .17).

We also sought to compare those with sudden gains to those without sudden gains with regard to RSE scores occurring around sudden gains. To do so, we calculated the average interval at which early and middle gains had occurred to those who had sudden gains, and also calculated the average session at which any sudden gains had occurred. The mean early gain occurred from group session 3 to group session 4 and the average gain across all time periods occurred from group session 5 to group session 6. Non-sudden gainers' scores from these sessions were used as comparisons for the sessions in which sudden gainers had had their gains. As above, independent sample *t*-tests did not reveal significant differences between those who had sudden gains and those who did not on their RSE scores just prior to a gain (*t*'s ranged from 0.16 to 0.53, *p*'s ranged from .30 to .44, one-tailed tests, *d*'s < .24. Thus, it appears that changes in self-esteem did not precede sudden treatment gains in our sample.

### 3.4. Factors associated with early sudden gains

We examined demographic, diagnostic and cognitive variables to determine if any of these were associated with the presence of early sudden gains. Because these were exploratory analyses, two-tailed tests were used. All analyses were conducted with those having sudden gains early in treatment and with those who had sudden gains exclusively early in treatment (i.e., the participant had no additional sudden gain). Because results of these two sets of analyses were virtually identical, we present the analyses conducted on those with early sudden gains.

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(footnote continued)

threshold for being classified as a sudden gain (shifts of 7 to 10 BDI points). In the two cases in which these middle gainers also had early gains, those early gains were also of small magnitude (shifts of 7 and 8 BDI points). In contrast, of the eight individuals with only early gains, seven had relatively large gains (shifts of 14–19 BDI points).

Participants who had sudden gains early in treatment were marginally younger (by 10 years; mean age 34.8 years vs. 44.8 years) compared to those who did not have early gains,  $t(29) = 2.04$ ,  $p = .05$ . Although 42.1% of women had an early sudden gain compared to 16.7% of men, this difference was not statistically significant,  $\chi^2(1, N = 31) = 2.18$ ,  $p = .14$ . Neither concurrent psychological treatment ( $p > .70$ ) nor concurrent psychotropic medication ( $p > .52$ ) were associated with early sudden gains. No other variables (education, number of previous depressive episodes, melancholic subtype, DAS, RSE, BHS, ASQ) were associated with sudden gains occurring early in treatment (all  $p$ 's  $> .13$ ).<sup>5</sup>

#### 4. Discussion

Tang and colleagues' (1999, 2002) observation of sudden gains in treatment for depression showed not only that sudden gains occur in the course of recovery from depression, but that these gains are associated with better treatment outcomes. We attempted to replicate their findings in a cognitive-behaviorally oriented group treatment for depression and to examine if gains in particular phases of treatment have a greater impact on outcome. Although our sample showed sudden gains at a similar frequency to those found by Tang and colleagues (1999, 2002), we did not find differences between sudden gainers and those without sudden gains in terms of treatment outcome. Importantly, this null finding does not appear to be due to limitations in power because the effect size of sudden gains on treatment outcome was small in our sample. In contrast, Tang and others who found medium to large effect sizes in similar analyses (Tang & DeRubeis, 1999; Stiles, Leach, Barkham, Lucock, & Iveson, 2003). However, we discovered that those whose gains occurred in the early part of treatment appear to enjoy a relatively favorable treatment response. Specifically, individuals who had an early gain had greater symptom reduction compared to those without an early gain. Additionally, those whose gains took place exclusively in the early part of treatment were more likely to be treatment responders when compared to all other participants. Future research needs to test whether there are important differences between individuals who have sudden gains (including early sudden gains) and those whose gains are achieved in a more gradual manner. It is possible that different processes contribute to early sudden gains compared to later sudden gains. For example, "common factors" such as hope may play a greater role in early sudden gains whereas mastery of behavioral skills may contribute to later sudden gains and gradual gains.

Despite their hypothesis that these gains were due to cognitive changes (Tang & DeRubeis, 1999), Tang and colleagues (2002) found similar gains in non-cognitive depression interventions, as have other researchers (Gaynor et al., 2003; Stiles et al., 2003). Similarly, our participants had no formal exposure to cognitive techniques in the early portion of the protocol when most sudden gains occurred. In addition, those with sudden gains did not differ significantly on several measures of cognitive style prior to treatment. Lastly, we did not find significant relationships between changes in self-esteem and treatment gains, indicating that sudden gains

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<sup>5</sup>Because of our interest in the impact of cognitive style on sudden gains, analyses were also conducted examining the relationship between any sudden gain and the cognitive variables. No significant relationships were found between initial scores on cognitive variables and having a sudden gain ( $p$ 's ranged from .37 to .74).



were not preceded with improvement in thoughts about the self. These effect sizes were negligible to small.

Our findings in combination with the discovery of sudden gains in non-cognitive focused interventions suggest that factors other than changes in cognition contribute to sudden gains in treatment for depression. In fact there may be a myriad of factors that contribute to the occurrence of sudden gains, many of which may be present in CBT as well as non-CBT oriented interventions. For example, sudden gains in depressive symptomatology may be caused by environmental factors or events in the individual's life. In their work examining the role of life events in depression, Brown and colleagues found a relationship between the resolution of ongoing difficulties and recovery from depression (Brown, Adler, & Bifulco, 1988; Harris, Brown, & Robinson, 1999). Similarly, Needles & Abramson (1980) found that positive life events interact with favorable attributional style to predict recovery from depression. Changes in client behavior resulting from treatment could contribute to these positive life events. Sudden gains in depression may also be caused by "common features" of psychological interventions, such as clinician warmth, the client's expectancies for change, and other factors attributed to the "therapeutic alliance" (Stiles, Shapiro & Elliott, 1986; Butler & Strupp, 1986). Lastly, "sudden gains" may in fact be attributable to the usually time-limited nature of depressive disorders. That is, sudden gains may be due to spontaneous remission that would have occurred regardless of treatment or other factors. Future research attending to these possible causes may be helpful in illuminating more clearly the causes of these apparently important changes in depressive symptoms.

#### *4.1. Limitations and future directions*

Although the current study is largely in concert with previous work documenting the existence and clinical relevance of sudden gains, future research may improve on the methods used in the present study in several ways. First, although our sample size was approximately equal to the sample size examined in the Tang et al. (2002) study, it was small in an absolute sense and therefore had limited statistical power. Despite our relatively small sample size, we did find sudden gains in approximate proportion to those found by Tang and other researchers (see Gaynor et al., 2003; Stiles et al., 2003) suggesting that our sample was not dramatically different than those previously studied. Furthermore, the effect sizes associated with our null findings were generally of small magnitude. One area in which our sample differed from Tang and DeRubeis' (1999) is in "reversals" of gains. Our study revealed a greater proportion of reversals than was found in Tang and DeRubeis' sample of patients receiving individual CBT (54% vs. 17%). Interestingly, other samples receiving supportive-expressive therapy (Tang et al., 2002) or routine clinical care (Stiles et al., 2003) also appear to experience high rates of reversals, suggesting that individually delivered CBT may be more effective in maintaining sudden gains. Another potential shortcoming of the present research is that there may have been a floor effect at work making it more difficult to find sudden gains later in treatment (due to scores being too close to 0 to meet the criteria). In contrast to this possibility, the average T2 BDI score was approximately 15, leaving "room" for up to 2 additional sudden gains based on our first criteria for sudden gains (a decrease in score of 7 points). Relatedly, our sample as a whole demonstrated somewhat limited change on the BDI over the course of treatment (9.2 BDI points). In the context of relatively limited change, the frequency

of sudden gains would have been diminished in our sample compared to samples with greater overall change.

In terms of our ability to test factors that may drive sudden gains, we relied on session-by-session measures of self-esteem, rather than more specific indices of other aspects of cognitive style. Thus we may not have detected changes that were occurring in cognitive areas outside of people's views of the self, which may have a greater impact on sudden gains. Repeated measures of dysfunctional attitudes, hopelessness, and attributional style may have revealed a relationship between changes in cognitive style and sudden improvements in depressive symptoms. In addition, as a reviewer pointed out, our analyses focused on changes in self-esteem occurring while the participant was depressed. These analyses presumed that changes in self-esteem could occur up to 1 week prior to changes in depressive symptoms. In contrast, Tang and DeRubeis' (1999) design permitted a much briefer time lag between cognitive change and change in depression. Specifically, someone could be quite depressed coming into a session, but exhibit a large cognitive change during the session that in turn would have an immediate impact on reducing depression. Our design was unable to detect such effects. To do so, future studies would need to measure cognitive constructs on multiple occasions in each treatment session, for example immediately prior to and immediately following sessions.

In addition, it may be that the cognitive changes that facilitate sudden gains are subtler than we were able to detect with the measures used. For example, [Hollon \(1999\)](#) suggests that the realization that thoughts are merely thoughts may promote sudden gains. In other words, participants may begin to see their underlying thoughts about themselves as hypotheses rather than truisms simply through the suggestion that one can control one's mood and make changes in one's life. Thus, gains may be attributable to very subtle cognitive changes even when no specific cognitive restructuring skills have been addressed and no clear cognitive changes have been observed. It is also important to acknowledge that our measures of cognitive style were all self-report in nature.

The study presented here represents an uncontrolled trial, and as such may not have been the most stringent test of the relationships between cognitive change and the occurrence of sudden gains. Future research should examine the impact of cognitive interventions as compared to non-cognitive (perhaps behavioral-only) control groups. Such a design might provide better information about the relationship between cognitive change and symptom reduction, as well as providing a means to compare rates of sudden gains in cognitive and non-cognitive interventions. In addition, because of the naturalistic nature of the present study, participants were not excluded if they were in concurrent treatment for depression, either psychological or psychiatric. It would be important for future research to systematically examine the potential impact of concurrent treatment on sudden gains.

Lastly, this study was not intended to investigate all possible causes of sudden gains in treatment. As discussed above, future studies should also include measures of environmental factors such as the resolution of on-going stressors and the occurrence of positive life events. In addition, the systematic measurement of non-specific treatment factors such as the strength of the therapeutic alliance and client self-efficacy to remediate depressive mood may further illuminate the causes of sudden gains in treatment for depression.

Despite these limitations, our findings provide further evidence of the presence and importance of sudden gains in treatment for depression. Further, they suggest a significant role for gains that

occur early in treatment. Although our sample size is small, our findings in conjunction with Tang's and those of Gaynor and colleagues (Gaynor et al., 2003) suggest that sudden gains are a robust and significant phenomenon in treatment for depression. Future research should more closely examine factors that are related to the occurrence of sudden gains and their long-term impact on the course of depression.

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