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Exploring Sudden Gains in Behavioral Activation Therapy for Major Depressive Disorder

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Abstract

Understanding the onset and course of sudden gains in treatment provides clinical information to the patient and clinician, and encourages clinicians to strive for these sudden clinical gains with their patients. This study characterizes the occurrence of sudden gains with Behavioral Activation (BA; Martell, Addis, & Jacobson, 2001), and the extent to which pre-treatment dysfunctional depressive thinking predicts sudden gains during treatment. We enrolled a sample of adults ($n = 42$) between ages 18–65 diagnosed with primary Major Depressive Disorder. All participants completed a 16-week course of BA, with clinical and self-report assessments at pre-, mid- and post-treatment. Results indicated that sudden gain and non-sudden gain participants showed differential improvement across treatment. No significant effects emerged for the dysfunctional cognitive style as a predictor of sudden gain status. Sudden gains may result from interaction of non-specific factors with the BA techniques implemented during early phases of therapy.

Keywords

Major Depression; Behavioral Activation; Sudden Gains

Introduction

The prevalence of Major Depressive Disorder (MDD) and its impact on functioning is well documented, with 12-month estimates in the U.S. of 6.7% and lifetime prevalence of 17% among adult patients (Kessler, Berglund, Demler, Jin, & Walters, 2005; Kessler, Chiu, Demler, & Walters, 2005). With such a major public health impact, approximately 52% of depressed individuals seek psychiatric treatment in any given 12-month period, and yet, only 20% of depressed individuals receive minimally adequate treatment (Wang et al., 2005). Taking into account those who enter treatment, 40%–50% of samples drop out prematurely (Persons, Burns, & Perloff, 1988; Venturini, Sung, Nichol, & Sellner, 1999). The prevalence of MDD, along with the relatively low number of individuals who receive effective treatment, highlights the value of research focused on increasing positive outcomes, reducing negative effects, and identifying factors that help patients experience relief once they are in treatment.

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The challenge with this kind of research is that symptoms of depression vary during the course of treatment, and the propensity to respond well to a specific evidence-based treatment is difficult for the patient and the clinician to estimate. About half of patients with MDD treated with behavioral psychotherapy report partial treatment response (Gollan, Gortner, & Dobson, 2006), and up to 75% achieve response or remission at long-term follow-up after a course of behavioral therapy (Dimidjian et al., 2006; Hopko et al., 2011). Identifying predictors of positive changes during behavioral therapies will improve our understanding of differential course of improvement during treatment. Given the dynamic and complex nature of response to treatment, and the individual differences in the trajectories of improvement, it is noteworthy that one form of treatment response is a rapid improvement of symptoms between therapy sessions, or sudden gains (Tang & DeRubeis, 1999).

In an analysis of within-individual depression changes with standardized research-based Cognitive Behavioral Therapy (CBT; Beck, Rush, Shaw, & Emery, 1979), a pattern was revealed in which some patients demonstrated a sudden, substantial drop in depression severity score in one between-session interval. This drop in symptoms accounted for much of the patient's total symptom improvement across treatment, indicating its significance beyond normal symptom fluctuation. The authors developed specific measurable criteria for what they termed "sudden gains": (a) the drop in score was at least 7 points on the Beck Depression Inventory (BDI; Beck, Steer, & Garbin, 1988); (b) the drop represented a 25% reduction from the pre-gain session score; and, (c) the mean BDI score in the three sessions prior to the drop was significantly greater than the mean BDI score in the three sessions following the drop.

Subsequent studies further explored the presence of the sudden gain phenomenon. For example, data for sudden gains emerged first from a study using two efficacy trials (Elkin et al., 1989; Hollon et al., 1992) with adult participants with MDD who received up to 20 sessions of CBT (Tang & DeRubeis, 1999). Of 61 participants, 24 experienced a sudden gain during treatment. These participants experienced a larger drop in BDI score compared with participants who did not experience a sudden gain. These participants also experienced better outcomes at end-of-treatment compared to those who did not experience such gains, defined by significantly higher recovery rates (Tang & DeRubeis, 1999).

Initial CBT trials examining sudden gains proposed that these sudden drops in depression severity result from the patient's insight into the cognitive model of depression (Tang & DeRubeis, 1999; Tang, DeRubeis, Beberman, & Pham, 2005). Consistent with this hypothesis, sudden gains were predicted by patient cognitive changes that occurred in "critical sessions," or sessions just prior to the sudden gain (Tang & DeRubeis, 1999). These findings were later strengthened using a more psychometrically sound form of the original cognitive change measure (Patient Cognitive Change Scale; Tang & DeRubeis, 1999; Tang et al., 2005).

Sudden gains have been captured in efficacy studies using different forms of psychotherapy, suggesting that gains may be independent of treatment approach and theoretical model. Sudden gains have been observed with Supportive Expressive Psychotherapy (Tang, Luborsky, & Andrusyna, 2002), group-based Cognitive Therapy (Kelly, Roberts, & Ciesla, 2005), Interpersonal Psychotherapy (Kelly, Cyranowski, & Frank, 2007), as well as with pharmacotherapy (Vittengl, Clark, & Jarrett, 2005). Results have been mixed, however, regarding the long-term implications of sudden gains. Participants exhibiting sudden gains have a lower risk of relapsing than those who do not experience such gains (Tang & DeRubeis, 1999; Tang, DeRubeis, Hollon, Amsterdam, & Shelton, 2007), though not all studies replicate these results (Busch, Kanter, Landes, & Kohlenberg, 2006).

Efficacy of behavioral treatments for depression has been researched for some time, beginning with Ferster's (1973) functional analytic theory of depression and Lewinsohn and colleagues' (1972) development of a specific treatment protocol. Lewinsohn's theory and interventions were based on the reinforcement explanation of depression, in that mood changes as a function of Response Contingent Positive Reinforcement (RCPR). According to this theory, depression is viewed as resulting from a decrease in/loss of positive reinforcement in the patient's environment, which causes reductions in frequency, intensity, and quality of previously reinforced healthy or non-depressed behavior (Dimidjian, Barrera, Martell, Munoz, & Lewinsohn, 2011; Hopko, Lejuez, Ruggiero, & Eifert, 2003).

Behavioral Activation (BA), emphasizing the functional relationship between reinforcement, behavior, and mood, has been tested as an effective stand alone approach for treatment of depression as a result of an important component analysis study (Jacobson et al., 1996). The results of this study were significant in that the least complex of the three interventions tested, BA, produced equivalent results to CT. Examining the efficacy of BA versus derivations of CBT to prevent relapse in this trial also indicated that all three components had comparable rates of relapse and recovery up to two years after treatment discontinuation (Gortner, Gollan, Dobson, & Jacobson, 1998).

BA has been shown to be effective for treatment of severe major depression, showing improved rates of response and remission (60–75%) when compared to CT (50%) (Dimidjian et al., 2006). In another clinical trial, those receiving prior BA or CT and those continued on medication had lower rates of relapse than those receiving medication discontinued on placebo at one year follow up. In addition, at two years after treatment discontinuation, BA was at least as efficacious as continued medication in preventing relapse (Dobson et al., 2008).

Sudden gains have been observed in treatment modalities using Behavioral Activation strategies (e.g., Andrusyna, Unpublished work; Hopko, Robertson, & Carvalho, 2009). With Jacobson's 16-session model of Behavioral Activation, sudden gains participants showed significantly lower depression severity on self-report and clinician-rated measures, and sudden gain responders showed significantly lower relapse rates compared with non sudden gain responders. However, within-session cognitive change, quantified with the Patient Cognitive Change Scale (PCCS; Tang & DeRubeis, 1999), was not significantly related to sudden gains (Andrusyna, Unpublished Work).

Complementary work using Behavioral Activation Treatment for Depression (BATD; Lejuez, Hopko, & Hopko, 2001)¹, a shorter behavioral treatment founded on matching theory (Herrnstein, 1970) and basing behavioral activation on a highly structured values assessment, measured sudden gains using the Tang and DeRubeis (1999) criteria. Results indicated that in a sample of 26 depressed outpatients diagnosed with cancer, 13 exhibited sudden gains across nine sessions of BATD. Sudden gains were associated with improved treatment response at end-of-treatment, and gains were maintained at 3-month follow-up (Hopko et al., 2009), with half of participants received training in cognitive and mindfulness techniques.

¹BA (Martell et al., 2001) and BATD (Lejuez et al., 2001) are behavioral therapies developed by separate groups during a similar time period; BA at the University of Washington by Jacobson and colleagues (1996) and BATD by Hopko and colleagues in Tennessee. BATD and BA share similarities in their grounding behavioral principles and techniques, but differ somewhat in emphasis. BATD has theoretical underpinnings in matching theory and bases activation strategies on a highly structured values assessment, whereas BA is based in Lewinsohn (1972) and Ferster (1973)'s behavioral theories and targets avoidance behaviors by replacing them with activities that generate mastery and pleasure. The approaches differ in number of sessions (6–8 in BATD vs. 16 in BA) and relative focus and hierarchy of varying behavioral techniques (Dimidjian et al., 2011; Hopko et al., 2003; Lejuez et al., 2001). Both have been shown to be efficacious in clinical trials (Dimidjian et al., 2006; Dobson et al., 2008; Hopko et al., 2011; Hopko, Lejuez, LePage, Hopko, & McNeil, 2003).

Sudden gains are associated with improved treatment response (Tang & DeRubeis, 1999; Tang et al., 2007), though the challenge remains to elucidate the extent to which processes that positively influence treatment outcome also increase the likelihood of sudden gains. One such predictor of change, dysfunctional cognitions, has been shown to affect outcome in CT, BA, and antidepressant treatment (Hamilton & Dobson, 2002; Jacobson et al., 1996; Pedrelli, Feldman, Vorono, Fava, & Petersen, 2008). Despite the absence of interventions to target cognitions, research participants in the component analysis who responded positively to BA also demonstrated significant reduction in depressive cognitive styles. That is, depressive cognitive styles were a mechanism of improvement in BA (Jacobson et al., 1996). Though cognitions influence a patient's participation and "buy in" to treatment, exposure to positively reinforcing events may be altering dysfunctional cognitions more effectively than explicit cognitive interventions (Jacobson et al., 1996). Because dysfunctional attitudes influence treatment outcome in CT and in BA, it is important to determine whether such attitudes influence a specific type of outcome, sudden clinical gains, in BA therapy. We therefore hypothesize that lower levels of dysfunctional thinking at the start of treatment, as measured by lower scores on the Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978), will predict sudden gains in BA.

Methods

Participants

Participants were forty-two adults, 13 males and 29 females, diagnosed with primary MDD according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (APA, 2000), enrolled in an open trial of Behavioral Activation treatment at an urban academic medical center. Healthy controls were also enrolled. The primary aims of the study are to evaluate prognostic indicators of treatment response to BA. All participants were recruited from the community, using online advertisements.

Measures

Structured Clinical Interview for the DSM-IV Axis I Disorders, Outpatient Version (SCID; First, Spitzer, Gibbon, & Williams, 1997b)—A trained evaluator issued all *DSM-IV* diagnoses using the SCID, a semi-structured interview reviewing lifetime and current *DSM-IV* Axis I psychiatric disorders. The SCID has adequate inter-rater reliability with kappa values for modules reported to be between 0.70 and 1.00 (First, Spitzer, Gibbon, & Williams, 1995). Inter-rater reliability estimates from this study's sample yielded kappa coefficients of 0.83 for the mood and 0.93 for the anxiety module of the SCID.

Quick Inventory of Depressive Symptomatology-Clinician Rated (QIDS-SR; Rush et al., 2003)—The QIDS-C is a 16-item clinician-rated interview assessing all core *DSM-IV* symptom domains (cognitive/mood, anxiety/arousal, vegetative) to diagnose MDD. This measure was used to assess clinician-rated clinical symptom severity at baseline (pre-treatment), mid-treatment, and post-treatment time points. Cronbach alphas for this measure in our sample were .94 at pre-treatment evaluation and .91 at post-treatment.

Quick Inventory of Depressive Symptomatology-Self Rated (QIDS-SR; Rush et al., 2003)—The QIDS-SR is a 16-item self-rated version of the QIDS-C with Cronbach alpha values in our sample of .86 at the pre-treatment assessment and .90 at end-of-treatment. The QIDS-SR also has demonstrated convergent validity, as evidenced by strong correlations ($r = .86$) with the Hamilton Rating Scale for Depression (HRSD) (Rush et al., 2003). This scale was selected for use in analysis of sudden gains because it was administered prior to each treatment session.²

Dysfunctional Attitudes Scale (DAS; Oliver & Baumgart, 1985)—We used the 40-item DAS to measure dysfunctional cognitions associated with depression at baseline. The items on the DAS are meant to reflect the characteristic assumptions and beliefs of psychopathology based on Beck's cognitive theory of depression, with higher overall scores indicating more dysfunctional thinking. The DAS has been shown to be a valid and reliable measure of dysfunctional thinking in depressed individuals, with a Cronbach alpha value of .83 in our sample.

Behavioral Activation Treatment Scale (BATS; Dimidjian, In preparation)—The Behavioral Activation Treatment Scale is a 16-item measure developed to assess core competencies based on the Behavioral Activation model of treatment (Martell et al., 2001). Ratings are assigned on a scale of 1 to 6 (1=Poor, 6=Excellent), and assess core areas of competency including: structural and stylistic strategies, conceptualization, and application of BA techniques. In addition, a rating is provided based on overall therapist skill. Competent administration of BA is defined as a score > 60. This scale is in development and results of psychometric properties are in preparation. Mean competency among therapists was 68.26.

Procedure

Prospective volunteers responded to online advertisements by calling one of our research coordinators, all graduate students in clinical psychology. The graduate coordinator obtained verbal informed consent and conducted a brief, 20–30 minute eligibility questionnaire by phone. If eligible, potential volunteers were then invited to the laboratory. At baseline, participants provided written informed consent and completed self-report questionnaires and clinician rated measures. All evaluators were Clinical Psychology Ph.D. graduate students, all of whom had 1–3 years experience with psychiatric interviews. All were trained in the conduct of interview protocols and other assessment devices according to criteria outlined by First and colleagues (1995), and were required to have achieved adequate inter-rater reliability ($Kappa > .80$) prior to conduct of interviews. Evaluators were blind to screening and treatment progress information and supervised to maintain blind and prevent rater drift.

Inclusion Criteria—Participants were right-handed males and females, fluent in English, between ages 18 and 65 years, unmedicated and able to pass a urine toxicology test, with normal or corrected-to-normal vision.³ All participants met criteria for current MDD according to the Structured Clinical Interview for *DSM-IV* (SCID; First et al., 1997b) and scored higher than a 9 on the QIDS-C (moderate severity). Of 42 participants, $n=9$ (21%) met criteria for a comorbid anxiety disorder considered to be secondary to depression in severity and onset.

Exclusion Criteria—Individuals were ineligible if they had SCID-rated lifetime or current bipolar I or II disorder, schizophrenia, delusional disorder, organic brain disorder, obsessive-compulsive disorder, post traumatic stress disorder, or Axis II disorders, as rated by the Structured Interview for *DSM-IV* Personality Disorders (SID-P; Pfohl, Blum, & Zimmerman, 1995) or Structured Clinical Interview for Axis II Disorders Questionnaire, 2nd edition (SCID-II; First, Gibbon, & Spitzer, 1997a). Other exclusionary criteria included alcohol or drug abuse or dependence within the last six months, imminent risk of suicide or homicide, use of psychotropic medications or involvement in concurrent psychotherapy,

²A subset of participants ($n=19$) completed the BDI-II. Scores were converted to QIDS-SR using the Rush et al. (2003) method to ensure consistency of measurement across treatment.

³These criteria pertained to another category of research questions related to affective processing and physiological response (EEG, EMG, EDA) in depression, requiring right-handedness, normal visual acuity, and absence of recent substance use or intoxication.

general medical conditions associated with depression, or current pregnancy. Of participants consented on site, $n = 7$ (14.3%) were excluded for comorbid exclusionary axis I or II disorders ($n=4$ for PTSD; $n=3$ borderline personality disorder).

Treatment—Enrolled individuals were assigned to a research therapist for 16 weeks of BA therapy, following treatment strategies and principles as described by Martell and colleagues (2001; 2010). Participants averaged 14 therapy sessions ($SD = 1.80$), provided once weekly for fifty minutes in a private clinical room. Study clinicians ($n= 12$) were either in the process of completing their postdoctoral training in clinical psychology or were licensed clinical psychologists. To train, study clinicians completed one pilot case to achieve competency and adherence (reviewed by JG) using the BA conceptual framework, reviewing assigned homework from previous BA sessions, and assessing introduction of the new BA topic along with homework for the subsequent week. Research clinicians met with JG weekly for case review and planning, with supervisory feedback provided after hearing all audio taped sessions.

Formal competency ratings were elicited by collecting two sets of independent ratings of adherence and competency. These ratings were assigned by two BA research clinicians with a version of the measure used in the Jacobson et al. (1996) and Dimidjian et al. (2006) trials. Drs. Christopher Martell and Ruth Herman-Dunn issued weekly independent ratings using the Behavioral Activation Treatment Scale, and presented these scores by weekly conference call to the clinicians. The mean score of competency was 68.26 across therapists (none less than 60). Competency is a score of > 60 on the Behavioral Activation Competency measure (Dimidjian, In preparation).

Data Analytic Plan

Definition of Sudden Gains—We defined sudden gains using Tang and DeRubeis' (1999) definition from their sudden gains validation study. We modified criteria (a) for use with the QIDS-SR measure. As Tang and colleagues describe, the first criterion was somewhat arbitrary, but was identified using frequency distributions and a reliable change index (RCI) of the BDI. Other researchers have modified the criteria for use with another measure (CORE-SF) using the RCI method (Stiles et al., 2003). We therefore defined this criterion using a validated standard of conversion created by the authors of the QIDS-SR (Rush et al., 2003) and checked our method with an RCI assessed on our sample. Criteria was defined as (a) a drop of four points on the QIDS-SR (equivalent to a reduction of seven points on the BDI). We identified participants who experienced a four-point drop between consecutive sessions.

Of these between-session interval drops, we used Tang and DeRubeis' (b) criteria to identify all of which qualified as at least a 25% reduction compared with the pre-gain QIDS-SR score. Finally, for each between-session interval that met criteria (a) and (b), we calculated Tang and DeRubeis' criteria (c) by conducting paired samples t -tests to determine whether the three post-gain session QIDS-SR scores were significantly lower than the three pre-gain session scores. If a participant's drop between sessions met all three of these criteria, it was considered a sudden gain session for the purposes of analysis.

Because we were interested in evaluating the significance of sudden gains that occur during all phases of treatment, we followed Bush and colleagues (2006) to include sessions that occur early in treatment.⁴ Thus, for gains that occurred following baseline assessment or

⁴Because the t test precludes examination of early gains, we followed Bush and colleagues (2006) to include analysis of gains that occurred following the baseline assessment and the first and second treatment sessions.

session one or two, 50% of the gain had to be maintained for at least two sessions following the gain.

Once sudden gains were identified, we identified reversals, and calculated the mean pre-gain session for each sudden gain participant, as well as mean baseline, post-treatment, and change scores from baseline to end of treatment for sudden gain and non-sudden gain participants. We also aimed to evaluate whether group differences existed in rates of response to treatment. Response ($\geq 50\%$ reduction in QIDS-SR) and remission ($\text{QIDS-SR} \leq 6$) rates at end of treatment were based on criteria defined by Rush and colleagues (2003).

Descriptive Analyses—Baseline differences in demographic and clinical characteristics were investigated using Analysis of Variance (ANOVA) for continuous variables and Chi-square tests of independence for categorical variables. Statistical significance for all analyses was defined as a 2-sided p -value < 0.05 .

Logistic Regression—To evaluate the contribution of baseline dysfunctional thinking (continuous variable) and sudden gains (categorical variable), we conducted a logistic regression, a technique that permits prediction of a dependent variable based on a continuous or categorical independent variable.

Profile Analysis—We conducted repeated measures linear regression analyses to assess the course of MDD symptoms across several time points during BA treatment (baseline, session six, and end-of-treatment). We used the tests of parallelism, flatness, and group differences to investigate symptom change across these pre-determined time points.

Results

Descriptive Statistics

Baseline sociodemographic characteristics are outlined in Table 1. Descriptive statistics are presented as means or percentages of the sample. Frequency differences were evaluated using Chi square analyses, which showed no differences for gender, $\chi^2 = 0.20$, $p = .65$, ethnicity, $\chi^2 (3) = 1.95$, $p = .58$, or employment status, $\chi^2 (3) = 4.15$, $p = .25$ by group (sudden gain vs. no sudden gain). Characteristics of clinical variables by sudden gain status are presented in Table 2. Of sudden gain participants, $n = 3$ (20%) met criteria for a diagnosis of comorbid anxiety, compared with $n = 6$ (22%) of nonsudden gain participants. Chi square analyses showed no differences for comorbid diagnosis of anxiety disorder $\chi^2 = .028$, $p = .87$ based on sudden gain status. One-way ANOVA analyses showed that participants also did not differ by self-reported ($F = 2.07$, $p = .16$) or clinician-rated ($F = 1.02$, $p = .32$) depression severity at baseline, dysfunctional cognitive style ($F = .004$, $p = .95$), or number of treatment sessions received ($F = .03$, $p = .87$).

Sudden Gains

Over the 16 weeks of treatment, one third of the sample ($n = 15$, 35.7%) exhibited a sudden gain. More than half did not demonstrate the marked improvement of symptoms during treatment ($n = 27$, 64.3%). Among the sudden gains group, the mean pre-gain session was session 4 (range = baseline – session 14). The median pre-sudden gain session was session 1, and 73% of gains had occurred by session six. Frequencies of sudden gains by pre-gain session are as follows: baseline ($n=5$), session 1 ($n=4$), session 2 ($n=1$), session 6 ($n=1$), session 12 ($n=2$), session 14 ($n=2$).

To ensure gains did not represent transient improvement in symptoms, we calculated the number of participants who experienced a reversal in sudden gain status, meaning they gave

up at least 50% of the change exhibited following the gain. Of the 15 participants who experienced a sudden gain, only two exhibited a reversal, and both returned to their post-sudden-gain score before the end of treatment.

Sudden gain participants began treatment with mean QIDS-SR scores of 16.60 ($SD = 4.05$) and QIDS-C of 14.0 ($SD = 2.42$). Non-sudden gain participants began treatment with mean QIDS-SR of 14.30 ($SD = 5.41$) and QIDS-C of 13.07 ($SD = 3.06$). Although sudden gain participants did not differ from non-sudden gain participants in baseline depression severity score, the two groups did differ in average change across treatment $t(40) = -3.22, p < .01$, with sudden gainers reporting an average drop in QIDS-SR scores of 11. points ($SD = 3.58$) from baseline to end-of-treatment, compared to an average drop in QIDS-SR score of 6.15 points ($SD = 5.63$) for non-sudden gainers. Mean end of treatment QIDS-SR scores were 5.27 ($SD = 3.35$; mild depression) and 8.14 ($SD = 7.08$; mild depression) for sudden gain and non-sudden gain participants, respectively. The average magnitude of a sudden gain was 6.2 QIDS-SR points.

Finally, we conducted separate one-sample t tests for sudden gain and non-sudden gain groups to determine whether they differed significantly from the population mean hypothesized as zero using self-reported and clinician-rated depression estimates. One-sample t tests indicate that sudden gainer $t(14) = 6.092, p < .001$ and non-sudden gainer $t(26) = 5.98, p < .001$ self-reported depression severity at end of treatment significantly differed from zero.

Logistic Regression

We conducted a logistic regression analysis to test whether dysfunctional beliefs predicted sudden gain status, controlling for baseline depression severity. A test of the full model against a constant only model was not statistically significant, indicating that the predictor did not reliably distinguish between sudden gain and non sudden gain participants, $\chi^2(2) = 2.95, p = .229$. The Wald criterion demonstrated that there were no significant contributors to prediction in this model; in other words, neither dysfunctional cognitions ($p = .351$), nor depression severity ($p = .201$) at baseline accounted for sudden gains.

Profile Analysis

To evaluate progression of symptoms over time between and within groups, we conducted a post hoc profile analysis. We based our selection of time points for the within subjects factor on the literature (Tang & DeRubeis, 1999; Tang et al., 2007) showing that the median sudden gain tends to occur following session five of treatment. We therefore used treatment session one, treatment session six, and end-of-treatment QIDS-SR scores as time points for the repeated measure in the analysis.

Multivariate Analyses of Variance (MANOVA) was used to conduct tests of flatness, parallelism, and group difference using QIDS-SR estimates as the dependent variable. Figure 1 shows the profiles of QIDS-SR scores at each time point by sudden gain status. The Parallelism test indicated that sudden gain and non-sudden gain participants differed in their progress during treatment $F(2, 39) = 3.49, p < .05$. Significant results were therefore also found for the Flatness hypothesis $F(2, 39) = 38.73, p < .001$. Results of the Groups test indicated no main effect of group membership, sudden gain versus non-sudden gain $F(1, 40) = .276, p = .602$.

Due to the differential progression observed across treatment based on group membership, Chi square analyses were conducted to evaluate differences in response and remission rates using the criteria defined by Rush and colleagues (2003). According to these criteria, a patient has responded to treatment if baseline depression severity score decreases across

treatment by at least 50%. Depression is considered to be remitted if post-treatment depression severity is less than or equal to 6 QIDS points. Sudden gain participants were significantly different from non-sudden gainers in rates of response to treatment $\chi^2 = 5.09$, $p < .05$, but were not significantly different in rates of remission at end-of-treatment $\chi^2 = .727$, $p = .394$.

Discussion

Frequency of Sudden Gains

Sudden gains occur with a variety of treatments, and this study replicates prior work (Andrusyna, Unpublished Work; Hopko et al., 2009) showing that sudden gains occur in behavioral treatments. The number of participants with sudden gains (35.7%) was comparable to the 34% found in 24 sessions of Interpersonal Psychotherapy (Kelly et al., 2007), and lower than reports using Tang and DeRubeis' (1999) definition of sudden gains. For example, 43% of participants experienced sudden gains in 20 sessions of Supportive Expressive Psychotherapy (Tang et al., 2002), and 43% of participants showed gains in 20 sessions of a modified version of CBT (using the data of the AT condition of Jacobson et al., 1996 study) (Tang et al., 2005). Even in pharmacotherapy (i.e., phenelzine sulfate) with clinical management, 46% demonstrated sudden gains (Vittengl et al., 2005).

The lower percentage of sudden gains may be attributable to a "treatment dosage" effect, specifically, the number of sessions accrued before the sudden gains. Participants in the current study averaged 14 treatment sessions once weekly, and in comparison, participants in the majority of sudden gains studies (Kelly et al., 2007; Tang & DeRubeis, 1999; Tang et al., 2005; Tang et al., 2007; Tang et al., 2002) accrued more sessions, starting twice weekly for the first half of treatment, for up to 24 sessions total. This increase in number of between-session intervals may increase the probability that a sudden gain will occur. However, sudden gains with BATD were evident among depressed outpatients with cancer with only nine treatment sessions (Hopko et al., 2009).

As a group, studies using the three criteria for sudden gains outlined by Tang and DeRubeis (1999) using the BDI or BDI-II, reported sudden gain percentages comparable to the original Tang and DeRubeis validation study. When using modified criteria (e.g., different measure of depressive symptoms, modifying criteria (c)), sudden gains results have differed (Busch et al., 2006; Kelly et al., 2007; Kelly et al., 2005; Stiles et al., 2003). Discrepancies in frequencies of sudden gains observed may also be an artifact of measurement differences.

Timing of Sudden Gains

Gains occurred across treatment in our sample, but the majority occurred earlier in treatment compared with CT trials (e.g., Hardy et al., 2005; Tang & DeRubeis, 1999), in which the majority of gains occur in sessions 4 through 10. Interestingly, in other behavior therapy trials, gains occur early. Modal sudden gain sessions were sessions 2 and 3 in Andrusyna (Unpublished Work) and Hopko et al. (2009), respectively. The timing of gains in our sample supports a 'treatment approach' interpretation, in that BA may be more influential at achieving sudden gains earlier than the cognitive component of CBT. In BA, early sessions focus on the role of contextual factors with the goal of increasing the rate of positively reinforcing behaviors (Jacobson, Martell, & Dimidjian, 2001). This early focus of treatment has the potential of affecting change quickly and rapidly. This is plausible given that cognitive therapy trials that did examine early treatment gains still report median sudden gain sessions as late as session 10 (e.g., Busch et al., 2006).

The early sudden gain phenomenon may also be attributed to patients' early socialization to models of activation, specifically, rehearsing activation in session followed by homework

assignment. Targeted activation, increased report of positive events, and increased engagement in pleasurable activities are associated with fewer depressive symptoms (Andrusyna, Unpublished Work). However, limited research to date has examined mechanisms of change in BA or demonstrated a temporal relationship between intervention, sense of accomplishment, and depression change (Dimidjian et al., 2011). As minimal research has examined behavioral variables as predictors of sudden gains, and these interventions are often introduced early in treatment, these variables deserve further investigation as a mechanism of sudden gain changes in treatment.

Alternatively, the early sudden gains observed have implications for examining nonspecific therapy factors such as patient's motivation for treatment, perceived support, previous therapy experiences, or patient-specific risk/protective factors such as severity or chronicity of depression or comorbid diagnoses. In addition, these factors could be examined in their interaction with BA techniques introduced early on, such as behavioral activity tracking and scheduling, acceptance of the treatment rationale, and ease of application of behavioral techniques. Another behavioral sudden gains trial (Hopko et al., 2009) suggests that early gains may also be due to increased therapeutic alliance, although this concept is yet to be examined since Tang and Derubeis (1999) reported null results. One limiting factor in making conclusions about nonspecific factors is that early gains have been measured only rarely (Busch et al., 2006), so the mechanism of such gains remains to be elucidated. Future research could benefit from measurement of such nonspecific factors in their association with sudden gains.

Although the mechanism involved remains to be determined, the timing of sudden gains may have implications for predicting end-of-treatment outcomes. Data suggests that sudden gains that occurred in the first 1/3rd of CBT group therapy were more predictive of treatment response than sudden gains that occurred later in treatment (Kelly et al., 2005). The implications of very early sudden gains in CT for depression were also explored in a trial examining gains that followed pretreatment and first sessions (Busch et al., 2006), presumably before any cognitive instruction (or change) has occurred. All patients who experienced a "first-session" gain had achieved remission criteria at the end of treatment.

Dysfunctional Attitudes as a Predictor of Sudden Gains

Contrary to hypotheses, baseline DAS scores did not predict sudden gains during BA. One other trial failed to support a relationship between pre-treatment factors and the occurrence of sudden gains (Kelly et al., 2005). In addition, no significant relationships were found between (a) changes in life events (Hardy et al., 2005), (b) self-esteem (Kelly et al., 2005), or (c) change in social-interpersonal functioning and depressive cognitions (Vittengl et al., 2005) and sudden gains.

Whereas the DAS is meant to measure stable depressive cognitions, Tang and DeRubeis observed in-session cognitive *change*, demonstrating an association with the later occurrence of sudden gains. One explanation for our failure to reject the null hypothesis may therefore be that sudden gains are better predicted by within session factors specific to theoretical model rather than pre-treatment patient factors. Accordingly, more BA-specific concepts such as patient agreement to behavior change and patient-reported increase in positive activities were found to be associated with sudden gains in previous BA research (Andrusyna, Unpublished Work). Therefore, it may be more relevant in calculating sudden gains in BA to examine the dynamic process of what occurs in early sessions of treatment rather than baseline variables.

Although measures of behavioral constructs specific to theory of change and BA have been lacking (Manos, Kanter, & Busch, 2010), this is changing. Future trials could use newly

developed measures of behavioral approach and avoidance designed to be administered throughout treatment. Such factors could then be examined in their association with sudden drops in depressive symptoms. It may be possible that behavioral factors predict sudden gains in cognitive therapy as well. Given that CT trials have focused only on cognitive changes and their prediction of sudden gains, it would be beneficial for future CT trials to also examine behavioral changes and their associations with sudden gains.

Course Profiles of Sudden Gain and Non-Sudden Gain Participants

Sudden gain and non-sudden gain participants experienced differing trajectories of change in depression severity across treatment. Specifically, sudden gain participants ended treatment with lower levels of depression severity, and demonstrated significantly greater depressive symptom change across treatment. Although no significant main effects were found for group membership, Chi square analyses comparing outcomes for the two groups indicated that sudden gain participants responded to treatment at significantly higher rates than non-sudden gain participants, similar to previous results (Tang & DeRubeis, 1999).

Sudden gains predict positive outcome in some studies. (Tang & DeRubeis, 1999; Tang et al., 2005; Tang et al., 2007; Tang et al., 2002). However, some do not report a significant difference in remission rate when only treatment responders are considered (Busch et al., 2006; Kelly et al., 2005). Additionally, inconsistent support has been found for the value of sudden gains as a predictor of sustained positive outcomes. Patients who experience sudden gains sometimes display better long-term outcomes (lower risk of relapse; higher response rates) (Tang et al., 2005; Tang et al., 2007); while at other times sudden gain patients do not (Kelly et al., 2007; Vittengl et al., 2005).

Methodological constraints or true variability in the samples collected across different sites may account for varied data on the effects of sudden gains. Although our results are consistent with the outcomes found by Tang and DeRubeis (1999), our analyses were post-hoc and thus should be interpreted with caution. Our results should also be examined at a follow-up phase to determine whether relapse or remission rates differ between groups.

Strengths and Limitations

Our study adds specificity to the timing of sudden gains within the context of BA therapy, extending beyond previous research that discounted sudden gains during the early course of treatment (Tang & DeRubeis, 1999). From a clinical perspective, sudden gains can transform anguished and impaired patients into functioning adults. Importantly, when they do occur, sudden gains are equitable to reliable clinical improvement. The ways in which sudden gains are cultivated remains unknown. However, the gains coincide with early introduction to BA principles of context, mood, and function of avoidance, as well as connecting increased goal-driven activities with increased sense of mastery and pleasure. This early engagement with goal-driven activities may then cause restoration of health, adaptive routines, better coping, and decreased depression severity. Alternatively, these early changes coincide with potential nonspecific therapy factors that were not assessed in this study. Although we have speculated as to potential mechanisms here, these factors remain to be addressed in future studies.

Our sample size as a whole is comparable with other research studies (Busch et al., 2006; Kelly et al., 2005), though the small sample, particularly small number of individuals who experienced sudden gains, could generate null results regarding group differences. In addition, sudden gains and outcome data are limited to self-report. Finally, without a control group we cannot fully determine which factors of BA treatment contribute to sudden gains.

Future research would benefit from comparing the occurrence of sudden gains using behavioral treatments of differing lengths (e.g., 6–8 sessions of BATD; Hopko et al., 2011) to evaluate the extent to which dose of treatment invokes sudden gains. The advantage of behavioral techniques is parsimony and ease of comprehension to patients compared to a full Cognitive Behavioral treatment (Cuijpers, van Straten, & Warmerdam, 2007; Hopko et al., 2011; Jacobson et al., 1996). Additionally, measuring BA-specific techniques in pre-sudden gains sessions to identify mechanisms of sudden clinical change would assist in determining key ingredients of treatment. Finally, generalizability of sudden gains to primary and psychiatric clinic settings, where patients may have limited opportunities for clinical care, may also benefit from research examining mechanisms that spark sudden gains, particularly given recent results in outpatient cancer centers (Hopko et al., 2011; Hopko et al., 2009).

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Article Highlights

- This study examines sudden gains in BA outpatient therapy for Major Depressive Disorder.
- We examined the timing and cognitive predictors of sudden gains in BA for MDD.
- We found that sudden gains in BA occur earlier than in previous CBT studies.
- Dysfunctional cognitions did not predict the occurrence of sudden gains.
- Sudden gains in BA may be better predicted by treatment process factors rather than baseline patient variables.

Figure 1.

Course profiles of sudden gain and non-sudden gain participants.

^aThe flatness test was significant, indicating a main effect of time, $p < .001$.

^bThe parallelism test was significant, indicating a significant interaction effect between group membership and time, $p < .05$.

Table 1

Summary of participant characteristics

	Mean	Freq.	%
<i>Age</i>	35.38	--	--
<i>Gender</i>			
Male		13	31.0%
Female		29	69.0%
<i>Employment status</i>			
Unemployed		15	35.7%
Employed		22	52.4%
Full-Time		4	9.5%
Student			
Disabled		0	.0%
Retired		1	2.4%
<i>Ethnicity</i>			
White		28	66.7%
Black		10	23.8%
Asian		1	2.4%
Hispanic		3	7.1%
Native American		0	.0%
Other		0	.0%

Table 2

Ms and SDs of clinical characteristics for Sudden Gain and NonSudden Gain participants across time points.¹

	SG		NSG	
	Pre	Post	Pre	Post
QIDS-SR	16.6 (4.05)	5.27 (3.35) **	14.3 (5.41)	8.15 (7.08) **
QIDS-C	14.0 (2.42)	5.83 (4.13)	13.07 (3.06)	--
DAS	145.73 (37.64)	--	155.11 (19.27)	--

Note: SG = Sudden Gain Participants; NSG = Non-Sudden Gain Participants; QIDS-SR = Quick Inventory of Depressive Symptomatology, Self Rated; QIDS-C = Quick Inventory of Depressive Symptomatology, Clinician Rated; DAS = Dysfunctional Attitudes Scale.

¹ Unless noted, differences between groups are not significant.

** SG showed significantly larger change in QIDS-SR scores across treatment, $p < .01$. SG also showed significantly higher response rates across treatment (change in QIDS-SR score $\geq 50\%$ from pre-to-post treatment), $p < .05$.