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ScienceDirect

Behavior Therapy 50 (2019) 504–514

Behavior Therapy

www.elsevier.com/locate/bt

Trajectories of Change in a Group Behavioral Activation Treatment for Severe, Recurrent Depression

Heather A. O'Mahen* [1]
Nicholas J. Moberly
Kimberly A. Wright [1]
University of Exeter

Depression is a common and costly problem. Behavioral Activation (BA) is an effective treatment for depression when delivered 1:1, but group treatments often do not perform as well as 1:1 treatments. One way to begin to understand how group treatments perform is to assess the process of change during treatment. This study examined trajectories of change across 10-session group BA for individuals with severe, chronic, or recurrent forms of depression. We also tested whether individuals who had associated sudden gains or depression spikes had better outcomes than those who did not have these change patterns. We examined psychological and sociodemographic predictors of the patterns of change. Participants were 104 individuals who met diagnostic criteria for major depressive disorder and participated in one of 10 BA groups, provided over a 2-year period. A linear, but not quadratic or cubic, rate of change fit the data and the effect size for the change in mood symptoms from baseline to posttreatment was large, Cohen's d = 1.25. Although 34% (26 of the 77 who provided outcome data) of individuals had a sudden gain and 10% (7/77) had a depression spike, neither sudden gains nor depression spikes predicted posttreatment outcomes. None of the demographic or psychological factors (rumination, behavioral activation) predicted the pattern of change. These results suggest that although group BA may help to reduce depressive symptoms in individuals with severe, recurrent, and/or chronic forms of depression, the overall linear pattern of change is different from quadratic patterns of change reported for 1:1 BA.

Keywords: depression; behavioral activation therapy; group; trajectory; sudden gain

Depression is a common mental health problem, affecting up to 15% of individuals (Kessler et al., 2003). It produces considerable individual suffering and disability, impairs social and occupational functioning, and negatively impacts health (Andrews, Henderson, & Hall, 2001). It is also a costly disorder, with profound health and economic costs (Layard, 2006; Murray & Lopez, 1997).

There are effective treatments for depression, and these include Behavioral Activation (BA). Meta-analyses have shown that BA substantially reduces depressive symptoms relative to usual care or waitlist control (Cuijpers, Van Straten, & Warmerdam, 2007; Ekers, Richards, & Gilbody, 2008). The promise of BA is considerable. Studies have demonstrated that BA is effective for severe depression (Coffman, Martell, Dimidjian, Gallop, & Hollon, 2007; Dimidjian et al., 2006) even when delivered by

[1] Heather A. O'Mahen and Kimberly A. Wright equally contributed to the development and conduct of the intervention and to the analysis and writing up of this intervention. *Acknowledgements*: Christopher Martell generously supplied input into the ongoing development and refinement of the group protocol. We would like to thank Edward Watkins for allowing us to use his Rumination-Focused treatment materials in the BA group and for providing peer supervision for the first BA group. As then lead of the ACCEPT clinic, Willem Kuyken gave both intellectual and logistical support for the BA group. We thank Jo Mackenzie, Miriam Cassell, and Rachel Clarke for co-leading the group, and recognize, posthumously, Lyn Carew for her clinical guidance and care in co-leading the group. Lastly, and most importantly, we would like to thank the women and men who participated in the group and, through their feedback, helped us to refine the treatment.

* Address correspondence to Heather A O'Mahen, Ph.D., Mood Disorders Centre, University of Exeter, Washington Singer Building, Exeter, EX4 4QG, UK; e-mail: ho215@ex.ac.uk.

junior mental health professionals (Richards et al., 2016), suggesting that BA may be cost-effective to implement. The cost-effectiveness of BA may be further enhanced if delivered in a group format. Preliminary uncontrolled studies suggest that group BA is feasible, acceptable, and potentially effective in reducing depressive symptoms (Houghton, Curran, & Saxon, 2008; Kellett, Simmonds-Buckley, Bliss, & Waller, 2017; Porter, Spates, & Smitham, 2004). However, in meta-analyses, between-group effect sizes for group-based treatments of cognitive behavioral therapy (CBT) have been smaller than 1:1 delivery formats of these treatments (Huntley, Araya, & Salisbury, 2012). Research is needed to understand processes associated with change in group-format treatments to support opportunities for therapists to optimize client change in existing treatment protocols, including those for group BA. Further, understanding factors that predict these processes of change may help clinicians to identify correlates that promote symptom change.

In this study, we were interested in two processes of change that constitute points when clients may make shifts in their behavior and mood: sudden gains and depression spikes, and their associated treatment trajectories. Sudden gains, which are defined as a sudden improvement in depressive symptoms that may be represented by a quadratic pattern of change, have been linked with better depression outcomes at treatment end and follow-up (Tang & DeRubeis, 1999). Likewise, depression spikes, or a sharp increase in negative mood when an individual approaches distressing, previously avoided content that is then followed by a reduction in symptoms, have been associated with better treatment outcomes in an exposure-based CBT for depression (see Figure 1 for visual representation of quadratic and cubic patterns associated with sudden gains and depression spikes; Hayes et al., 2007). Depression spikes are theoretically similar to the increase and then quick decrease in distress that occurs during exposure-based treatments for anxiety. In the exposure-based treatment, individuals who had an overall cubic pattern of change that was characterized by having a sudden gain that was then followed by a depression spike had the best treatment outcome. Although these two patterns of change point to important moments of change during treatment, there has been little research investigating these patterns in group-based treatments. It is important to examine these processes in group formats because it is possible that the intensity of the treatment delivery affects the process of change. For example, sudden gains and depression spikes may be more likely to occur in treatments that are more intensive and individualized, such as in 1:1 therapy. Alternatively, group factors, such as group identity, may promote or inhibit change in individuals within a particular group compared to another group. The presence, or lack, of these processes in a group-based treatment may point to factors associated with the overall performance of group treatments and point to future areas of research for group versus individual delivery modes.

This study builds on a small body of research on sudden gains in group treatment, although the findings are currently mixed. There has been only one study of sudden gains in group CBT for depression. In that study, consistent with the broader literature on sudden gains, 13 out of 30 individuals (41.8%) had a sudden gain, and sudden gains were associated with greater improvement at posttreatment (Kelly, Roberts, & Ciesla, 2005). Although these results are promising, this was a small sample and requires replication with a larger sample. In three other studies of group-based treatments for anxiety and insomnia, rates of sudden gains ranged from 15.2% to 18.7% (Clerkin, Teachman, & Smith-Janik, 2008; Hofmann, Schulz, Meuret, Moscovitch, & Suvak, 2006; Laakso, Tolonen, & Wallin, 2014). These rates compare unfavorably with those in 1:1 treatments, where the average proportion of individuals who have a sudden gain is 36.9%

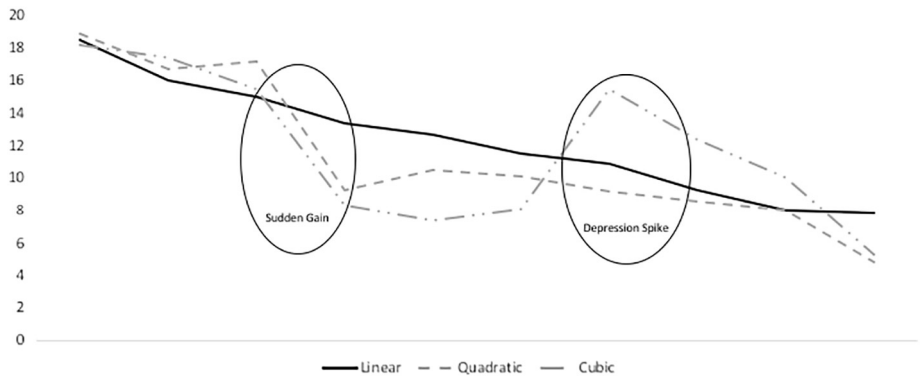

FIGURE 1 Weekly BDI-II Depressive Symptoms and Hypothesized Patterns of Change

(Aderka et al., 2012). There have been no studies we are aware of examining sudden gains in group BA, or depression spikes in group-based treatments for depression, and few studies have examined the overall pattern of change across group treatments. In one exception, Mickelson (2008) found a quadratic pattern of change in which most of the change in individual, group, and conjoint treatment in a university-based clinic happened in the early stages of treatment and levelled off towards the latter stages of treatment.

aims

We aimed to assess patterns of change and associated sudden gains and depression spikes in group BA as delivered in a National Health Service (NHS) offering psychological therapies to individuals with severe or recurrent depression. Because previous research has found evidence of sudden gains, but not depression spikes, in BA (Hunnicutt-Ferguson et al., 2012; Masterson et al., 2014; O'Mahen, Wilkinson, Bagnall, Richards, & Swales, 2017), we predicted that the pattern of change in a group BA treatment would be quadratic, characterized by early sudden gains. We expected that individuals with quadratic patterns of change (i.e., those with early sudden gains) would have better clinical outcomes than individuals who did not have a sudden gain. We also sought to explore whether individuals in a group BA treatment would have depression spikes. We expected that if there were depression spikes, the pattern of change would be cubic in nature and that the depression spikes would be related to better clinical outcomes. We also tested a competing hypothesis. Because group treatments have previously demonstrated weaker outcomes than 1:1 treatments, we explored whether this group BA treatment would be characterized by a more gradual, linear trajectory of change. Lastly, we expected that individuals with a greater number of previous depressive episodes, greater brooding rumination (a repetition mode of thinking that "focuses an individual's attention on their depressive symptoms"; Nolen-Hoeksema & Morrow, 1991, p. 569) or less baseline behavioral activation, because they would theoretically be less likely to engage actively with approach-related behaviors (Watkins & Nolen-Hoeksema, 2014), would exhibit fewer sudden gains and the related patterns of change. We further explored whether age and gender predicted patterns of change.

Methods

PARTICIPANTS

We evaluated BA group treatment as offered by the Accessing Evidence-Based Psychological Therapies (AccEPT) Clinic, an NHS primary-care mental health clinic housed within the Mood Disorders Centre at the University of Exeter, specializing in the treatment of severe or recurrent depression. Clients in this service were referred from NHS general practitioners or mental health practitioners. Between September 2008 and August 2011 we offered 10 iterations of the BA group treatment; 101 patients participated over this period. Individuals who were 18 years or older, met DSM-IV diagnostic criteria for major depressive disorder, scored above 15 on the Patient Health Questionnaire (Kroenke, Spitzer, & Williams, 2001), or had significant clinical symptoms of depression and who were able to engage in psychological therapy were eligible for the BA group (see Table 1). Patients were not eligible to take part if they had a history of psychosis, bipolar disorder, or organic brain damage, they were currently engaging in antisocial behavior or persistent self-harm, they were already receiving psychological therapy, or they had significant longstanding interpersonal difficulties requiring specialist longer-term psychological therapy (see Figure 2 for flow of referrals).

MEASURES

Depression

Diagnostic status was assessed with the Structured Clinical Interview for DSM-IV Axis 1 Disorders—Patient Edition (SCID-I; First et al., 2002).

Depressive Symptoms

Baseline, weekly, and posttreatment mood was assessed with the Beck Depression Inventory-II (BDI-II; Beck et al., 1996). The BDI-II is a reliable measure of mood, is sensitive to change, and is frequently used in CBT trials. Cronbach's alpha in this study was .88.

Depressive Rumination

Depressive rumination was assessed with the 22-item Response Styles Questionnaire–Ruminative Response Scale (RRS; Treynor et al., 2003). Previous studies reported acceptable convergent and predictive validity for the RRS (e.g., Nolen-Hoeksema & Morrow, 1991). Cronbach's alpha in this study was .71.

BA

We used the 25-item Behavioral Activation for Depression Scale (BADS; Kanter et al., 2007) to assess behavioral activation over the past week. The BADS has been validated in a community sample with elevated depressive mood and has good test–retest reliability (Kanter et al., 2009). Higher scores on the scale are indicative of more behavioral activation and less behavioral avoidance. Cronbach's alpha in this study was .73.

Table 1
Baseline Demographic Information for Group Behavioral Activation Participants and Differences in Treatment Dropout

Variable	*n*	%
Recurrent depression	96/99	94.1
Number of episodes		
1	7/92[2]	7.6
2	13/92	14.1
3	23/92	25
4	7/92	7.6
5	42/92	45.7
BDI-II depression severity		
"Normal" (0-10)	0	
Mild (11-16)	3/100	3
Borderline depression (17-20)	4/100	4
Moderate depression (21-30)	28/100	28
Severe depression (31-40)	34/100	34
Extreme depression (40+)	31/100	31.0
Previous suicide attempt	41/101	40.1
Previous treatment		
Antidepressant	82/96	85.4
Psychological treatment	85 /100	85.0
Inpatient treatment	21/101	20.2
Number of Axis 1 disorders		
1	47/87	54.1
2	18/87	22.1
3+	22/87	25.2
Age of onset of depression		
	M = 21.96	SD = 12.16
Childhood onset		
Pre-puberty	12/96	15.1
Adolescence	38/96	38.7
Adult onset	46/96	47.92
Ethnicity		
White[1]	96/98	98
In a relationship	37/102	36.3
Gender	97/102	63.4
Age	M = 41.38	SD = 12.82
Number of children		
0	1/100	1
1	5/100	5
2	30/100	30
3	33/100	33
4+	31/100	31

BDI-II, Beck Depression Inventory-II

ASSESSMENT AND BA GROUP TREATMENT

Assessments were conducted by AccEPT clinic assessors and therapists, although the assessor was not the treating therapist. Assessors had graduate degrees in psychology and/or therapy (i.e., master's in CBT) or were currently completing a qualification in clinical psychology, and had completed the Mood Disorders Centre SCID training protocol. This training involved watching the SCID tapes, one day of didactic training with a clinical psychologist, and ongoing SCID group supervision to ensure diagnostic reliability.

Treatment

The 10-session weekly group BA treatment followed the BA functional analytical framework (Addis & Martell, 2004). Each session was 120 minutes in duration, including a mid-session break. Therapists followed the Group BA manual developed by the lead authors, KW and HAO. Group participants used a participant workbook. Groups were between 7–14 individuals in size. To help ensure that participants individualized the content from the group, all individuals were first invited to an orientation group where they discussed the purpose of the group, and their goals for treatment. A mid-treatment individualized "review" session was also offered, to support participants to continue adapting the content of the group to their own goals. A final, individual session was offered at the end of treatment to discuss individual relapse-prevention goals. The group sessions (described in Table 2) also included visualization of individual use of skills, and significant use of pair-work to develop individual activation goals, and conduct role-plays. Group themes focused on approaching the self and behavioral tasks with compassion, curiosity, and effectiveness and using specific, concrete methods to change.

Therapists

Six different therapists conducted the group in pairs. Therapists had postgraduate training in therapy (or were in their qualifying year on such a program), backgrounds in conducting CBT, and received additional BA-specific training and weekly supervision from the two lead authors, KW and HAO. Both KW and HAO have completed BA training and have extensive research and clinical experience in CBT and BA. Training consisted of reading Dimidjian, Martell, Addis, Herman-Dunn, and Barlow's (2008) BA book, Addis and Martell's (2004) workbook and the BA group treatment manual, in addition to didactics and role-play training with either KW or HAO. Therapists co-led at least one group with an established BA group therapist before undertaking independent group leadership. Supervision used videotaped footage of the group to ensure ongoing adherence to the BA manual.

PROCEDURE

GPs or mental health practitioners in the local community were informed about the AccEPT clinic and its remit. GPs and mental health practitioners referred individuals whom they felt were appropriate for the clinic, and who agreed to the referral. Referred individuals were first asked to complete a telephone screen to determine if they had either current or residual symptoms of depression. Individuals who met these criteria were mailed an assessment packet, which

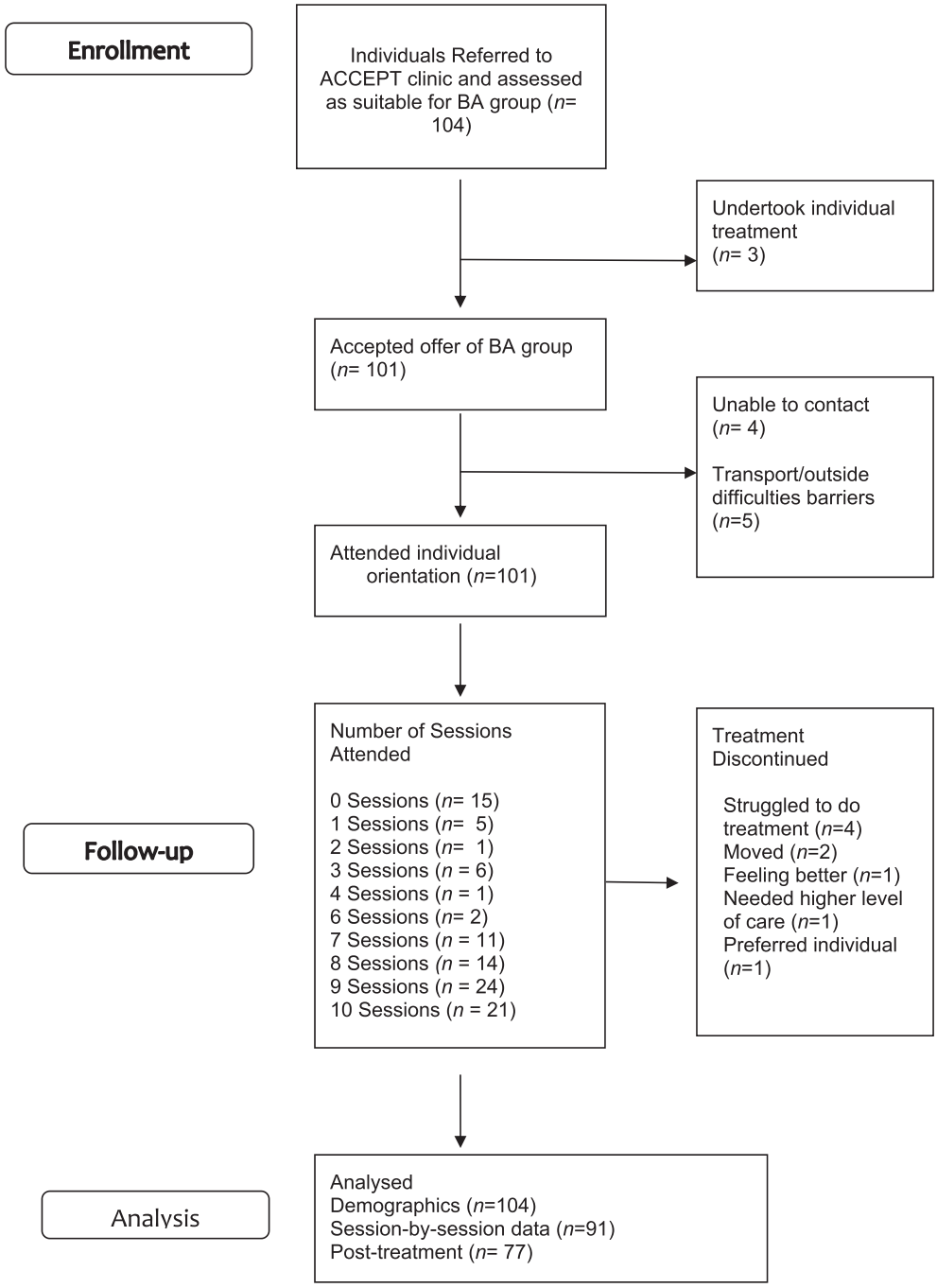

FIGURE 2 CONSORT Flow Diagram for BA group for Depression

included the BDI-II, a screening measure for Axis II personality disorder features, and a rumination and BA measure, and were invited to an in-person interview. At the interview, written consent for treatment was obtained. As part of the consent process, individuals were informed that the clinical service would use routine data to evaluate treatments, and this may include publications resulting from these evaluations. Following this, the clinic assessor conducted a full SCID for Axis I. Those persons who met the criteria for the BA group were invited to join the group, which was offered on a termly basis; or another suitable treatment available at that time (including individual CBT) where individuals requested those treatments. Individuals who agreed to join the BA group were then asked to come for an individual orientation prior to the beginning of the first group. At each weekly group, participants were

Table 2
BA Group Treatment Module Description

Session Number	Session Title	Session Content
1	Learning Your Patterns	Group Introduction, Introducing the concept of avoidance and relating it to depression, Beginning self-monitoring
2	Getting Out of Traps	Relating self-monitoring to avoidance. Introducing functional analysis of avoidance
3	Taking ACTION	Translating avoidance behavior into approach related behavior. Setting specific plans
4	Breaking Down Challenges	Functional analysis of barriers to change
5	Rumination	Understanding rumination as a barrier to approach related behavior. Discriminating between productive and unproductive forms of thinking. Functional analysis of when individual ruminates. Identifying alternative behaviors to rumination. Learning to counter "why" forms of thinking with "how" forms of thinking to get individuals to be more concrete/specific
6	Connecting to the Present	Combating rumination by introducing strategies to help individual become absorbed in everyday activities
7	Self Compassion	Identifying critical ways of "speaking" to oneself that are associated with rumination. Introducing strategies to identify ways to "speak" to oneself with a compassionate voice. Discussion of justification to use self-compassion.
8	Recap and Planning Ahead	Reviewing progress to this point. Identifying strategies that are working for individual and barriers to implementing other strategies. Identifying "next steps" goals and specific ways to achieve these goals.
9	Values	Helping individuals to identify life values and process ways of living versus outcome focused ways of living (e.g., "learning" goal, versus, "achievement" goal). Linking depression to disconnection with values.
10	Resilience	Thinking ahead to managing future times of emotional struggle using strategies from the group. Thinking forwards about how to enjoy a rich and fulfilling life. Reflecting on group process.

asked to complete the BDI-II to track their mood. Mood and functioning measures were collected again at the end of treatment.

ANALYTICAL STRATEGY

Mood Improvement

We report means and standard deviations for BDI-II mood at baseline and posttreatment and Cohen's *d* effect size for within-person comparisons. To quantify clinical improvement, we report response, defined as an improvement of 50% in scores and Jacobson and Truax's (1991) formula for calculating reliable and clinically significant change in depressive symptoms. We populated the formula using data from the BDI-II manual (Beck et al., 1996) to estimate reliable change and data from Dozois, Dobson, and Ahnberg (1998) to define clinical change. On the basis of these data, a participant had to improve by 10 points or more from pre- to posttreatment to show reliable change. To demonstrate clinical change the individual had to score below 20 on the BDI-II (see Jacobson & Truax, 1991, for details of calculations). We report rates of individuals who experienced improvement, stayed the same, or experienced decline. We provide data for both completers and for noncompleters using last observation carried forward.

Trajectories of Change

We examined the shape of change in depressive symptoms (BDI-II) using individual growth curve (IGC) modeling (Singer & Willett, 2003). There are problems with standard repeated-measures (i.e., ANCOVA, repeated measures ANOVA) approaches when using data with multiple assessment points across time. Standard repeated measures approaches assume that the timing between assessments will be equivalent and assume independence of observations. However, with multiple assessments there is the possibility that there will be missing data at different time points for different participants. This violates the assumption of equal intervals between assessments. Further, with multiple repeated assessments (e.g., weekly mood measures), it is possible that each piece of information at each time point is not independent. This results in biased standard errors. An IGC approach is preferred to traditional repeated measures approaches because it reduces standard errors of within-subject change in the growth parameter estimates (Singer & Willett) and can more flexibly manage missing data and the associated problems this produces for the repeated measures assumption of equal intervals between assessments (Shek & Ma, 2011). Further, IGC approaches can consider the shape of change. It is also possible to examine

whether the shape of change varies based on different predictors.

An IGC approach is ideal to use with group treatments, because it can test for both within group and between group change, and can be used to determine if there is significant clustering of data within groups.

Using IGC, we examined whether the shape of symptom change followed a linear, quadratic, or cubic pattern. We also examined which pattern of change predicted outcome. Because depression severity is frequently a predictor of response to treatment, in the IGC analyses we examined whether depression severity affected the rate of change across individuals across each of the three patterns of change.

Our participants were nested within groups, so it was possible that there would be clustering in patterns of change over time depending on group membership. Although one analytic approach would be to model group as a random effect, the small number of groups and small number of participants within groups led to model convergence problems. Therefore, we modeled the group structure using fixed effects by including dummy variables to represent group differences in the intercept and interacted these with slope parameters to represent group differences in the slopes.

Lastly, we examined psychological and sociodemographic predictors of the pattern of change using IGC. These included gender, age, number of previous episodes of depression, baseline behavioral activation, and brooding rumination.

Calculating Sudden Gains

We conducted analyses that examined patterns of response within individuals. Using ANCOVA, we tested whether there were significant differences between people who did or did not show sudden gains on BDI-II depression scores at posttreatment, controlling for baseline (pretreatment) depression.

To calculate sudden gains, we used Tang and DeRubeis' (1999) approach. They defined a sudden gain using three criteria. First, the gain should be large in absolute terms. Tang and DeRubeis used a change of 7 points of more on the BDI. Second, the change should be large relative to the change in the previous session. We used Tang and DeRubeis' criterion that the improvement was at least 25% of the score in the pregain session. Lastly, the sudden gain should represent a shift in scores that is larger than the stability of scores preceding and following the gain. Although Tang and DeRubeis originally defined this as a *t*-test comparing the three scores preceding the gain to the three scores following the gain, subsequent tests of sudden gains have conducted *t*-tests on the two scores preceding and following the sudden gain to allow examination of earlier sudden gains. We used this definition in this study.

Calculating Depression Spikes

The depression spike is a temporary increase in depressive symptoms that conceptually results from approaching emotionally distressing and previously avoided content. We adapted Hayes et al.'s (2007) definition of a depression spike to capture an absolute increase in depression scores that was consistent with the reliable change of 10 or greater on the BDI-II, followed by a decrease of 10 points of more. In their study, Hayes et al. examined depression spikes using 7-point shifts on the Hamilton Rating Scale for Depression, and the increase and decrease in symptoms had to occur within a specific phase of treatment. We expected that the spike would be most likely to occur when the individual was engaged in approach-related behaviors that countered avoidance, and in BA these can occur at several points across the treatment. We therefore did not constrain the spike to a particular part of treatment. We did, however, examine only those spikes that occurred after Session 3, when participants started to engage in approach-related behavior, and we specified that the spike should decrease by 4 points within three sessions. We also did not include spikes that were on the "down-side" of a sudden gain, as these may simply represent a return to a previous depression level following a sudden gain. Thus, this operationalization of spikes is different from the Hayes et al. method, but is conceptually similar.

Results

PARTICIPANTS

Individuals in our group BA treatment had severe, recurrent, treatment-resistant depression (see Table 1). Over 94% of the sample suffered from recurrent depression, with 78% of those individuals having suffered from three or more episodes of previous depression, and despite the fact that 85% of the sample had received either antidepressant medication or had a psychological therapy during their most recent episode of depression, 65% still met criteria for "severe" or "extreme" depression on the BDI-II. Of the 101 individuals who accepted the offer of the BA group, 76% (n = 78) provided posttreatment data.

DESCRIBING OUTCOMES

Mean depressive symptoms reduced from assessment (M = 35.89, SD = 9.80) to posttreatment (M = 23.63, SD = 13.11; Cohen's d = 1.25). Of those

individuals who provided posttreatment scores, 48% (n = 36/75) had a response (defined as a 50% reduction in scores), 52% (n = 39/75) had a reliable change, 27% (20/75) had no reliable change, and 21% (16/75) deteriorated. Further, 42% (n = 29/69) met criteria for remission, or clinical change, defined as scores that were below 20 on the BDI at posttreatment (in those who had scores of 20 or above at baseline). Lastly, 42% (n = 29/69) had both clinical and reliable change.

Within the last observation carried forward sample, 30% (n = 29/97) of clients had a response, 45% (n = 44/97) had a reliable change, 39% (38/97) had no reliable change, and 16% (15/97) deteriorated. Further, 37% (n = 33/90) met criteria for remission, or clinical change, and 32% (n = 31/97) had both clinically and reliable change.

TRAJECTORY OF CHANGE

Using the intraclass correlation coefficient (ICC), we first assessed the proportion of outcome variation that was related to interindividual differences. The ICC was 95.83/(95.83 + 59.97) = .61, suggesting that approximately 61% of the total variation in BDI scores throughout treatment was due to interindividual differences. An IGC approach is recommended when ICC values are .25 or greater (Shek & Ma, 2011).

When we added the fixed effects of group, these did not explain significant variance in mean levels of depressive symptoms, $\Delta\chi^2(9) = 8.12$, $p = .52$. Gender was also not a significant predictor of mean levels of depressive symptoms, b = -0.84, $SE(b)$ = 2.23, $p = .71$.

After adding a fixed linear growth parameter of week, we found that there was a significant linear decrease in BDI-II depressive symptoms from a baseline adjusted mean of 25.28 (SE = 3.13) over time, b = -0.87, $SE(b)$ = 0.07, $p < .001$. Allowing the linear growth parameter to vary randomly across persons significantly improved the model, $\Delta\chi^2(2) = 71.78$, $p < .001$. The covariance ($\tau = 4.17$, $SE = 1.18$, $\chi^2 = 12.58$, $p < .001$) between the intercept and the linear growth parameter at the person level was significant, indicating that people with higher BDI-II scores at baseline tended to show a reduced rate of improvement over the course of the treatment. When we added interactions between the dummy variables representing groups and the linear growth effect, the model did not improve significantly, $\Delta\chi^2(9) = 6.72$, $p = .67$, suggesting an absence of between group differences in linear trajectory of symptoms.

When we tested a quadratic pattern of change by adding a fixed effect of week squared, this was not significant, $b < 0.01$, $SE(b) = 0.02$, $p = .42$, suggesting that there was no significant acceleration or deceleration in depressive symptoms over time. However, allowing the quadratic growth parameter to vary randomly between persons significantly improved the model, $\Delta\chi^2(3) = 28.71$, $p < .001$, suggesting that there were significant between person differences in the acceleration or deceleration of depressive symptoms over time. Adding interactions between the set of dummy variables representing groups and the quadratic growth effect did not significantly improve the model, $\Delta\chi^2(9) = 10.83$, $p = .29$, suggesting that groups did not differ significantly in the extent to which depressive symptoms accelerated or decelerated over time.

We also tested a cubic model of change by entering a fixed effect of week cubed, but this was not significant, b = –0.01, $SE(b)$ = 0.01, $p = .08$, suggesting that there was no significant rate of change of acceleration in depressive symptoms over time. Allowing the cubic effect to vary randomly across persons significantly improved the model, $\Delta\chi^2(4) = 15.08$, $p = .004$, suggesting that there were between-person differences in cubic trajectory. When we added the interactions between the cubic effect of week and the set of dummy variables representing groups, the model fit did not improve significantly, $\Delta\chi^2(9) = 5.61$, $p = .78$, indicating that there were no significant group differences in the rate of change in acceleration of depressive symptoms.

Overall, despite individual differences in the trajectory of depressive symptoms over time, the sample exhibited a significant linear decrease in depressive symptoms over time representing an average improvement of 0.9 BDI-II points per week with no evidence for quadratic or cubic change over time. Groups did not differ significantly in trajectory.

Predictors of Trajectory of Change

We subsequently examined psychological and demographic predictors of linear change. None of the psychological or demographic factors we examined were significant predictors of change in BDI-I score, including gender, b = –0.22, $SE(b)$ = .86, $p = .80$, previous number of episodes of depression, b = –0.04, $SE(b)$ = 0.02, $p = .07$, brooding rumination, b = –0.05, $SE(b)$ = 0.12, p = .66, age, b = –0.01, $SE(b)$ = 0.01, $p = .37$, or baseline behavioral activation scores b = –0.01, $SE(b)$ = 0.02, $p = .36$.

SUDDEN GAINS

Twenty-six individuals (34%) had a sudden gain; 4 of these individuals had more than one sudden gain. Although individuals who had a sudden gain had lower posttreatment depression scores (M = 20, SD = 2.34) than those who did not have a sudden

gain (M = 25.88, SD = 12.90), this difference was not significant, $F(1, 73) = 2.70$, $p = .03$, nor did sudden gains predict reliable change, $\chi^2(1) = .39$, $p = .53$. However, individuals who had a sudden gain were more likely to have clinically significant improvement (n = 13/22, 59%) than those who did not have a sudden gain, n = 18/53, 34%; $\chi^2(1) = 4.24$, $p = .03$. Sudden gains did not predict reliable and clinical change, $\chi^2(1) = 3.22$, $p = .07$.

Predictors of Sudden Gains

None of the baseline demographic or psychological factors we measured predicted sudden gains, including brooding rumination, $F(1, 73) = 3.22$, $p = .08$, behavioral activation, $F(1, 73) = .13$, $p = .72$, number of previous major depressive disorders, $F(1, 73) = .34$, $p = .56$, or gender, $\chi^2(1) = .03$, $p = .87$.

Depression Spikes

Only 10% (n = 7/77) of individuals had a depression spike. Most spikes occurred at Session 3 or 4 (71%, n = 5/7). Although individuals who had a spike had lower depression scores posttreatment (M = 18.29, SD = 8.83) than individuals who did not have a spike (M = 23.99, SD = 13.40), this difference was not significant, $F(1, 75) = 2.31$, $p = .13$. Because of the low number of spikes, we were not able to conduct chi-square analyses examining differences in response, or reliable and clinical change.

Discussion

We found that participants in a 10-session BA group had significant decreases in their depressive symptoms, and the overall pattern of change was characterized by a linear, but not a quadratic or cubic pattern of change. Importantly, we did not find any evidence that changes in individual depressive symptoms clustered by the group that they were in. Although these results are consistent with other studies of individual BA (O'Mahen et al., 2017), they were contrary to our predictions that the BA group would be characterized by a quadratic pattern of change that might represent early sudden gains, followed by a more gradual pattern of improvement. Alternatively, we hypothesized that this early pattern of sudden gains might be followed by sudden increases in depression associated with approaching previously avoided domains—represented as a cubic pattern. However, we did find that the model fit improved when we allowed the quadratic and cubic effects to vary randomly, suggesting that there were between individual differences in the types of patterns of change individuals had. Although we did not have the sample size to formally test these subgroup differences with methods such as latent growth curve analyses, we expect that the variability in trajectories may be in part due to the fact that some subgroups of individuals had either sudden gains or depression spikes.

Consistent with this notion, we found that a significant minority (34%) of individuals in the BA group had a sudden gain. Although this rate is lower than the 42.5-51.0% of sudden gains reported in other studies of one-to-one BA with mild to severe depression (Masterson et al., 2014; O'Mahen et al., 2017), it is similar to rates of sudden gains reported in meta-analyses of the broader depression treatment literature (36%; Aderka et al., 2012). Unlike other studies of sudden gains in BA, however, there were mixed results about whether sudden gains were related to symptom outcome at treatment end. Sudden gains did not predict overall symptom reductions, but did predict the likelihood of having clinically significant improvements. Importantly, this difference was not attributable to severity of symptoms at baseline.

We also found that a small minority of individuals (10%) experienced a depression spike and most experienced it in either Session 3 or 4, when the content of the group focused on beginning to approach previously avoided behaviors. These results suggest that the timing of the depression spikes may have been theoretically consistent with the concept of temporary increases in depressive symptoms occurring when individuals begin to reengage with avoided content (Hayes et al., 2007). Although individuals who had depression spikes had lower posttreatment depression scores than those who did not have a depression spike, this was not significant and the study was underpowered to test whether depression spikes were clinically beneficial.

Together, these results suggest that subgroups of individuals demonstrated different moments of change in treatment (i.e., sudden gains, depression spikes), although we found that the number of individuals who had these changes was smaller than in other studies of BA and CBT, and we did not find that these patterns were consistently related to outcome. Further, we did not find that trajectories that we expected would be related to these moments of change were a good fit with the data. It is unclear why sudden gains and depression spikes occurred with less frequency than in other studies of treatments for depression and why they were not related to posttreatment depressive symptoms in this study. Tang and DeRubeis (1999) suggest that sudden gains provide an important shift that starts an upwards positive trajectory during treatment, and Hayes et al. (2007) posit that depression spikes

represent shifts in mood that reflect a reordering of understanding about events when an individual approaches a previously avoided topic. However, it is possible that the severe, recurrent nature of depression in this sample of individuals required a larger shift or greater ongoing, individualized support of the changes that happen with sudden gains and depression spikes. It is also possible that individual attitudes and behaviors coalesced around group attitudes (Forgas, Fiedler, & Sedikides, 2012), and this process could have reduced the subsequent individual impact of a sudden gain or depression spike. This process may have also dampened overall patterns that may have otherwise emerged as quadratic or cubic patterns of change. Future research gathering more in-depth session-by-session data, such as coding therapy tapes and conducting individual process interviews, may help to further delineate these processes.

Lastly, in this study none of the predictors we measured at baseline were related to either the shape of the trajectory or presence of a sudden gain. This may have been due in part to our sample size. Methods such as latent growth curve analyses may help to better delineate subsamples of individuals with particular trajectories of change, but these methods typically require larger sample sizes, ranging upwards from 300–500 (Finch & Bronk, 2011). Future analyses of large clinical datasets that include weekly measures of mood, such as the UK's Improving Access to Psychological Therapies (IAPT) data, may have sufficient sample size to answer these questions. It may then be possible to predict latent class membership with greater accuracy. It may also be useful to measure life events and specific therapist and client factors session-by-session in order to more accurately assess factors the promote or inhibit sudden gains and depression spikes.

CONCLUSION

The findings of this evaluation suggest that group BA for individuals with SCID-diagnosed, primarily severe, treatment-resistant depression can help to reduce depressive symptoms, and that for most individuals, this change pattern is primarily linear in nature. Importantly, we did not find that individual outcomes were determined by the group they were in. Although some individuals experienced sudden gains or depression spikes, and sudden gains were associated with better clinical improvement, these moments of change were otherwise not significantly related to outcome at group end. In all, these results to key factors that might help to explain differences in the effectiveness of group interventions when compared to 1:1 treatments. Further research exploring subgroup differences in trajectories and change, and factors associated with these differences may provide important insights into how to optimize group-based interventions.

Conflict of Interest Statement

The authors declare there are no conflicts of interest.

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RECEIVED: October 15, 2017
ACCEPTED: August 28, 2018
AVAILABLE ONLINE: 31 August 2018