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Behavioral Activation as a Mechanism of Change in Residential Treatment for Mood Problems: A Growth Curve Model Analysis

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Research on the efficacy, effectiveness, and dissemination potential of behavioral activation (BA)-focused interventions for depression and comorbid disorders has expanded rapidly. However, research that examines how BA interventions work has seen less growth. A primary purported mechanism of BA is activation, which reflects a person's meaningful (re)engagement in life. BA theory posits that depression will decrease as activation increases, and that changes in the mechanism variable will lead to changes in outcome. The current study aims to investigate activation as a potential mechanism of change in the context of a BA-focused residential treatment intervention for mood problems using repeated measures of self-reported activation and depression from a large comorbid sample ($N = 578$). Growth curve modeling was used to examine between-person differences in within-person change over time. Findings suggest that self-reported activation increases and depression decreases over time. Moreover, results show both linear and quadratic growth and that the rate of change in activation predicts the rate of change of depression. BA-focused residential treatment may facilitate activation, which exerts an effect on depression among residents with diagnostically complex presentations.

Keywords: mechanism of action; behavioral activation; activation; depression

VARIANTS OF BEHAVIORAL ACTIVATION (BA) have been well-established empirically validated treatments for depression for over a decade (e.g., Cuijpers, van Straten, & Warmerdam, 2007; Ekers, Richards, & Gilbody, 2008; Mazzucchelli, Kane, & Rees, 2009). Since then, clinical guidelines put forth by the U.S. Department of Veterans Affairs (Management of MDD Working Group, 2016), the U.K. National Institute for Health and Care Excellence (National Collaborating Centre for Mental Health, 2010), and the World Health Organization (2016) have identified BA as a

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frontline treatment for depression in many cases. Although considerable progress has been made in terms of establishing BA's efficacy and effectiveness, less progress has been made with regard to understanding how BA works. Elucidating the treatment mediators that may function as mechanisms of change in mental health interventions has been a central concern in need of attention for decades (Hyman, 2000). Findings on the processes that lead to change have the potential to inform the development of treatment innovation (Kraemer, Wilson, Fairburn, & Agras, 2002) and more effective psychological practice (APA Presidential Task Force on Evidence-Based Practice, 2006), such as through the development of treatments with greater potency or cost-effectiveness.

According to the BA model, depression results from and is maintained by reductions in BA (i.e., activation), which result from losses of, decreases in, and/or chronically low levels of response-contingent positive reinforcement (RCPR; Lewinsohn, 1974). To reverse the cycle of depression, increases in activation that restore contact with RCPR need to be achieved (see Manos, Kanter, & Busch, 2010, for a more detailed elaboration of the model). BA techniques are therefore ultimately designed to target activation in order to increase contact with RCPR (Kanter et al., 2010). The development of pragmatic and conceptually sound measures of activation and other relevant constructs has facilitated the expansion of the BA mechanism literature. These include the Behavioral Activation for Depression Scale (BADS; Kanter, Mulick, Busch, Berlin, & Martell, 2007) and its variants, which measure self-reported activation.

Research intended to clarify the mechanistic processes in BA has been sparse (Dimidjian, Barrera, Martell, Muñoz, & Lewinsohn, 2011) yet has witnessed some growth over the last several years (Santos, Puspitasari, Nagy, and Kanter, in press). In order to establish a treatment mechanism, a specific temporal relationship must be demonstrated between the treatment mediator and the outcome variable. In particular, change in the mediator variable (e.g., activation) should temporally precede change in the outcome variable (e.g., depression; Gaynor & Harris, 2008; Kazdin, 2007). A review of the available research designed to evaluate whether activation, primarily using the BADS, leads to changes in depression suggests that activation may be a mechanism of change for many, although not all, BA clients successfully treated for depression (see Santos et al., in press, for a more detailed review of research on BA mediators).

The BA mechanism literature has yet to be advanced in key ways. BA treatment mediators must be examined using repeated measures designs with sufficiently large samples to permit the use of state-of the-art and robust statistical techniques for examining processes in psychotherapy, such as growth curve modeling (Laurenceau, Hayes, & Feldman, 2007). A variety of power tools for analyzing repeated measures exist and growth models differ from traditional methods in several key respects. For instance, they are very flexible with regard to including partially missing data, unequally spaced time points, and non-normally distributed repeated measures. In addition, growth models generally show considerably higher levels of statistical power compared to conventional methods when used with the same data (Curran, Obeidat, & Losardo, 2010). An advantage of growth curve modeling is that, in contrast to other methods that examine mean changes and treat individual differences as error variance, it looks at this error variance for information about change (Duncan & Duncan, 2004). Thus, growth curve models permit the examination of between-person differences in within-person change over time.

To date, investigations of activation as a mechanism of change that have used repeated measures designs have been conducted with small samples, which preclude the use of sophisticated statistical methods. Thus, most investigators have relied on single-subject methodology. In an early study, Gaynor and Harris (2008) concluded that increased activation could plausibly explain subsequent improvements in depression in two of four BA clients. In a study of two BA clients, Manos, Kanter, and Luo (2011) showed that change in activation occurred before change in depression for one client and that changes in activation and depression co-occurred within the same evaluation period for another client. Folke et al. (2015) found that changes in activation and avoidance (measured using the Checklist of Unit Behaviors [CUB]; Hanson, Hoxha, Roberts, & Gollan, 2013) preceded or occurred concurrently with changes in depression in an evaluation of six individuals in inpatient psychiatry using a multiple baseline design. Using the same analytic approach with 21 clients who were administered the BADS—Short Form (BADS-SF; Manos et al., 2011), Santos et al. (2017) found that 43% of BA clients, but no treatment-as-usual (TAU) clients, reported increases in activation that temporally preceded decreases in depression. In addition, 71% of BA clients and no TAU clients reported increases in activation that corresponded to decreases in depressive symptomatology during the same window of time. Thus, the results suggested that activation was a plausible mechanism of change for 79% of BA clients and no TAU clients.

Table 1
Participant Characteristics

	N = 578
Female *n* (%)	331 (57.27)
Age	
M (*SD*)	27 (18.7)
Median	23
Marital status, *n* (%)	
Single	455 (78.7)
Married	83 (14.4)
Other [a]	38 (6.6)
Highest level of education, *n* (%)	
Graduate coursework/degree	38 (6.57)
Undergraduate coursework/degree	297 (51.38)
Vocational training	12 (2.08)
High school/equivalent degree	117 (20.24)
Declined to state/unknown	114 (19.72)
Race, *n* (%)	
White	461 (79.76)
American Indian/Alaska Native	87 (15.05)
Black/African American	11 (1.9)
Declined to state	6 (1.04)
Ethnicity, *n* (%)	
Hispanic or Latino	24 (4.15)
Not Hispanic or Latino	551 (95.33)
Declined to state	3 (0.52)
Medication use [b]: yes, *n* (%)	
Upon admission	474 (82.01)
Changed ≤ 2 weeks of admission	485 (83.91)
Mood	454 (78.54)

Note. *M* = mean; *SD* = standard deviation.
[a] Other = divorced, widowed, or separated.
[b] Missing data, *n* = 93 (16.09%) residents.

The BA mechanism of change literature can be meaningfully expanded by including studies conducted with samples that are ecologically valid. Results may help us understand whether BA can manipulate activation and effect subsequent change on depression with and in real-life patients and settings, respectively. Previous examinations of the BA mechanism of change have limited samples to those with depression without select comorbid conditions (e.g., Santos et al., 2017) and have examined this in very small samples (e.g., Folke et al., 2015).

The current study builds on the existing BA literature by evaluating whether activation is a plausible mechanism of change in a large comorbid sample of adults receiving BA-focused treatment for mood problems in a residential treatment facility. The nature of change and relationship between activation and depression symptom severity was examined in the context of a longitudinal nonexperimental design using self-report process measures completed weekly and growth curve modeling. We hypothesized that self-reported activation would increase and depression would decrease over the course of treatment. Furthermore, we hypothesized that changes in self-reported activation would predict changes in depression over the course of residential treatment.

Method

PARTICIPANTS AND TREATMENT SETTING

Data were collected from a residential program specializing in the treatment of mood disorders at

Table 2
Diagnoses of Residents Sampled

	Primary		Secondary	
	Frequency	Percent	Frequency	Percent
Depressive disorder	309	53.5	92	15.9
Depressive disorder NOS	18	3.1	4	0.7
Generalized anxiety disorder	7	1.2	174	30.1
Obsessive-compulsive disorder	6	1.0	25	4.3
Anxiety disorder NOS	2	0.3	36	6.2
Other anxiety disorder	3	0.5	45	7.8
Developmental disorder	1	0.2	6	1.0
Eating disorder	0	0	4	0.7
Other psychological disorder	7	1.2	12	2.1
Substance use disorder	91	15.7	45	7.8
Bipolar disorder	85	14.7	14	2.4
Psychotic disorder	8	1.4	2	0.3
Personality disorder	4	0.7	30	5.2
Posttraumatic stress disorder	3	0.5	22	3.8
No diagnosis	0	0	33	5.8
Not able to be categorized	34	5.9	34	5.9

Note. NOS = not otherwise specified.

Rogers Memorial Hospital (RMH) in Oconomowoc, Wisconsin. Participant characteristics are presented in Table 1. Diagnoses for the residents sampled are presented in Table 2. In all cases, a problem with mood was deemed a primary intervention target. Treatment plans were grounded in the BA model of change (e.g., Manos et al., 2010) and intervention was guided by a manual that consolidates the theories put forth by BA variants and clarifies the clinical techniques that comprise them (Kanter, Busch, & Rusch, 2009). The treatment duration (i.e., length of stay in the residential program) was based on clinical and financial considerations and varied by resident. With rare exception, residents took psychotropic medication upon admission and throughout residential treatment. Medication use data are presented in Table 1. Participants were eligible to participate in the current study if they were admitted into the residential treatment program, consented to having their responses to the measures used for research purposes, and completed a posttreatment assessment. Research procedures were approved by the RMH Human Subjects and Clinical Effectiveness Steering Committees.

PROCEDURE

Assessment

Prior to admission, individuals were telephone screened by a trained program intake coordinator. This screening was evaluated by a psychologist or psychiatrist to determine appropriate program placement. At admission, individuals were evaluated for DSM-IV-TR (American Psychiatric Association, 2000) depression and comorbid diagnoses by a board-certified adult psychiatrist. Within 72 hours of admission, residents were asked to complete a variety of self-report measures (baseline). A subset of these were also given each week over the course of treatment, with the larger self-report assessment battery given again at discharge (posttreatment). These measures were used to inform treatment, and residents were also provided the option to consent to their data being used for research purposes. Participants in this sample provided consent for their demographic and self-report assessment data over the course of treatment to be used for research purposes.

Treatment

The BA model (Kanter et al., 2010; Manos et al., 2010) served as the foundation for treatment, which was thus designed to increase residents' engagement in healthy, nondepressed behaviors and decrease engagement in avoidance behaviors. The activation-focused treatment protocol is grounded in three characteristics of BA. The first characteristic has to do with its fundamental objective. BA's ultimate goal is to increase the client's activation and level of engagement with his or her world (Martell, Dimidjian, & Herman-Dunn, 2010). Thus, all techniques in BA are implemented in the service of this paramount objective. The second characteristic has to do with BA's primary intervention target. In their review of the empirical literature and interventions spanning nearly three decades, Kanter et al. (2010) concluded that BA's core technique is activity scheduling. The vast majority of approaches reviewed shared the same goal of increasing contact with available sources of positive reinforcement in the environment and set out to accomplish this goal via activity scheduling. The third characteristic relates to BA's idiographic approach to treatment. Relative to other approaches, BA is highly individualized. The specific techniques used are therefore determined by the particular needs of the client. These include techniques traditionally associated with activation-focused protocols (e.g., skills training) and techniques traditionally associated with other approaches (e.g., dialectical behavior therapy [DBT]). In BA, techniques are appropriate (e.g., use of wise mind) if they get the client activated in the ways he or she needs to get engaged in life (Martell et al., 2010). The BA protocols used in the residential treatment are grounded in these BA characteristics.

The BA treatment and training protocols used to instruct behavioral specialists was developed by a BA expert (R.C.L.). Behavioral activation interventions were led by a residents' behavioral specialist. Behavioral specialists completed a BA training protocol that was implemented by a team of licensed doctoral-level psychologists with expertise in BA and other cognitive-behavioral therapy approaches. Training consisted of a 2-week didactic course and shadowing of experienced behavioral specialists in various treatment contexts. Psychologists provided behavioral specialists with individual supervision for 1 hour per week from the point of starting their position onward with additional group supervision and clinical direction provided on a weekly basis (see Farrell, Leonard, and Riemann, 2019, for more on the training model and use of paraprofessional clinicians).

Within approximately the first 24–48 hours following admission, residents were assigned to start tracking clinically relevant problem behaviors, such as avoidance, isolation, and rumination. They were asked to mark the number of times they engaged in these behaviors (and, at times, additional behaviors) and the number of times they had

wanted to engage in these behaviors but resisted doing so each day. Residents were also asked to read psychoeducational material about depression and BA at this time. Soon after admission (i.e., within approximately 72 hours), residents met with their behavioral specialist to create an activity hierarchy, a list of diverse activation targets generated from an assessment of current activity, enjoyable activities, values, and basic activities that the client may have stopped doing (e.g., hygiene). As in exposure treatments for anxiety disorders (e.g., Franklin & Foa, 2008), activities were ranked in order of difficulty. The resident then steadily moved up the hierarchy by gradually completing more difficult activities (Kanter et al., 2009).

Residents typically start with activity assignments related to routine activities (e.g., showering, doing laundry), then identify and add enjoyable activities (e.g., drawing, playing a board game) to the hierarchy. Valued activities (e.g., work on college admissions essay, talk each week with sibling due to a desire to improve their relationship) are typically added last as these tend to take more time for residents to identify. Residents were assigned various BA activities from their hierarchies to complete at a specified frequency and duration (e.g., call sister twice weekly and talk with her for 20 minutes each time). All residents also participated in psychoeducational groups on weekdays that included BA topics (e.g., rationale for treatment, routine and enjoyable activities, rumination, values) in addition to general cognitive-behavioral therapy topics not specific to depression (e.g., communication styles, psychoeducation about anxiety, goal setting). Recreational activities during weekends afforded residents additional opportunities to engage in BA assignments within the program building and in the community.

Residents also participated in art and recreational therapy several times per week, which may function as informal (not on their hierarchy) or formal opportunities for further BA. The primary therapist, who was trained as a master's-level social worker or therapist, provided individual therapy sessions, family sessions, and discharge planning. This often included educating family members on BA and how they can best support their loved one in implementing BA strategies among other topics. The therapist also provided DBT-informed skills groups on weekdays. Residential counselors were on site 24 hours per day 7 days per week to assist in monitoring safety and engagement in programming, including completion of BA assignments. Residents typically met with a psychiatrist (or rarely an advanced practice nurse prescriber) twice weekly for medication management.

The time between admission and discharge was on average 6.07 weeks (SD = 2.63, median = 6). We opted to conduct our analysis up to 4 weeks as after this time point missing data were in excess of 50%.

MEASURES

For purposes of the current study, ratings provided at baseline, weekly during the first 4 weeks of treatment, and at posttreatment were examined.

Behavioral Activation for Depression Scale—Short Form (BADS-SF)

The nine-item self-report BADS-SF (Manos et al., 2011) measures BA and avoidance (e.g., "I engaged in many different activities") during the previous week. Participants rate the extent to which each statement applies on a 0 (*not at all*) to 6 (*completely*) scale. Total scores obtained range from 0 to 54, with higher scores reflecting greater levels of self-reported activation. Per recommendations by Manos et al. (2011), the total score was used rather than the subscale activation and avoidance scores. The BADS-SF has shown good reliability, construct validity, and predictive validity (Manos et al., 2011). In addition, the BADS has been shown to discriminate between individuals based on depression diagnosis status (Raes, Hoes, Van Gucht, Kanter, & Hermans, 2010). Self-reported activation will be referred to as activation henceforth.

Quick Inventory of Depressive Symptomatology—Self-Report (QIDS-SR)

The 16-item self-report QIDS-SR (Rush et al., 2003) measures the severity of depression as operationalized by the DSM-IV-TR (American Psychiatric Association, 2000), which assessed depressed mood, loss of interest or pleasure, concentration problems, negative self-perceptions, changes in weight/appetite, changes in sleep patterns, decreases in energy, psychomotor functioning, and suicidality. Participants were asked to rate the degree to which they experienced each symptom during the past week using a scale that ranged from 0 to 3. Scoring is based on symptom clusters such that the highest rated item pertaining to a symptom cluster is scored. Total scores range from 0 to 27, with higher scores indicating greater depression severity. The QIDS-SR has demonstrated acceptable psychometric properties, including sensitivity to changes in symptom severity (Trivedi et al., 2004).

DATA ANALYTIC PLAN

The hypotheses were tested using growth modeling techniques (Duncan, Duncan, & Strycker, 2006) using EQS software. Statistical assumptions were

Table 3
Descriptive Statistics of Process Variables

Measures	Mean	*SD*	Median	Min.	Max.
BADS-SF					
Baseline	23.74	7.75	24	0	48
Week 1	27.96	7.61	28	1	54
Week 2	28.55	7.05	28	6	48
Week 3	28.89	6.71	29	8	48
Week 4	28.34	7.75	28	0	53
Posttreatment	30.72	7.79	30	10	52
QIDS-SF					
Baseline	16.18	5.09	17	1	26
Week 1	12.87	5.33	13	0	27
Week 2	11.85	5.24	12	0	26
Week 3	11.16	5.11	11	0	24
Week 4	10.79	5.39	11	0	24
Posttreatment	7.6	5.32	7	0	27

Note. SD = standard deviation; BADS-SF = Behavioral Activation for Depression—Short Form; QIDS-SR = Quick Inventory of Depressive Symptomatology—Self-Report.

evaluated and missing data estimated and imputed through maximum likelihood (ML) estimation (Allison, 2003). All residents had BADS-SF and QIDS-SR posttest data and all other variables had some missing data. There were 332 complete cases. The percentage of missing data for each variable increased as the number of weeks increased. At Week 1, 19% (n = 111) of cases were missing BADS-SF data and 18.7% (n = 108) were missing QID-SR data. By Week 4, 38.2% (n = 221) and 37.9% (n = 219) of cases were missing BADS-SF and QID-SR data, respectively. As expected, some residents completed treatment sooner than others. The data met the assumption of missing at random (MAR) and missing values were imputed using ML estimation (Schafer & Graham, 2002). The data violated the assumption of multivariate normality;

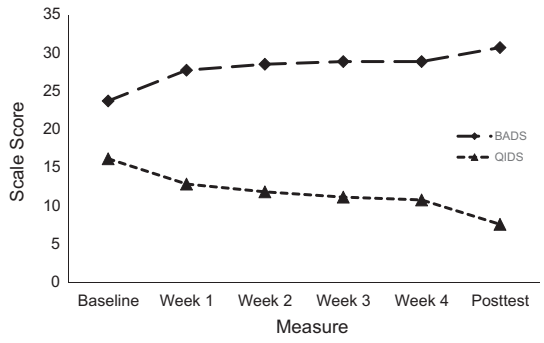

FIG. 1 Change in behavioral activation (BADS-SF) and depression (QIDS-SR) over time. *Note.* BADS-SF = Behavioral Activation for Depression—Short Form; QIDS-SR = Quick Inventory of Depressive Symptomatology—Self-Report.

Yuan, Lambert, and Fouladi's (2004) coefficient = 23.29, $z = 17.72$, $p \ll .001$. Therefore, models were evaluated with Yuan and Bentler's (2000) scaled chi square and the standard errors of parameters estimates were also adjusted to the extent of the non-normality. There was no evidence of univariate nor multivariate outliers. The data analytic plan included an examination of medication use as a moderator in the growth models to isolate the effect of medication use on activation and depression. However, the extent of medication use by the residents sampled (see Table 1) precluded the examination of medication use.

We began by estimating models for linear and quadratic growth across six time periods: baseline, 4 weeks of treatment, and posttreatment. After establishing both linear and quadratic growth for the BADS-SF and QIDS-SR, we estimated models to test whether the rate of change (both linear and quadratic) in QIDS-SR could be predicted from the rate of change in BADS-SF. Data from a total of 578 participants were used in the analyses.

Results

PRELIMINARY ANALYSES

Descriptive statistics are presented in Table 3. At baseline, residents reported BADS-SF measured activation levels comparable to other adult samples with depression symptoms in the clinical range (Dimidjian et al., 2017) and lower than levels reported by a sample of college students with subthreshold depression symptoms (Manos et al., 2011). They reported depression symptoms in the moderate to severe range (Rush et al., 2003). Paired samples t tests were conducted to examine differences in repeated measures. Residents evidenced significant increases in activation between baseline and Week 1, Week 1 to 2, Week 2 to 3, and Week 4 to posttreatment (ps $\ll$ .001–.05) but not Week 3 to 4. The greatest increases in activation occurred between baseline and Week 1 and between Week 4 and posttreatment. Moreover, paired samples t tests showed significant decreases in depression between all pairs of repeated measures (ps $\leq$ .001). The greatest decreases in depression occurred between baseline and Week 1 and between Week 4 and posttreatment. By posttreatment, residents reported levels of activation that were higher than a depressed sample treated with BA at 5-week follow-up (Dimidjian et al., 2017) and depression symptoms in the mild range (Rush et al., 2003).

ACTIVATION AND DEPRESSION SYMPTOMS OVER TIME

A growth model fit with a linear growth for both activation (BADS-SF) and depression (QID-

SR) fit the data well, $\chi^2(N = 578, df = 60) = 382.67$, $p \ll .05$, robust CFI = .95, robust RMSEA = .07, 90% confidence interval (CI) [.06, .08]. The slope for linear growth for both BADS-SF and QID-SR was significant, BADS-SF unstandardized (b) coefficient = .30, $z = 6.86$, $p \ll .001$, QIDS-SR $b = -.62$, $z = -12.74$, $p \ll .001$. From baseline through 4 weeks of treatment and posttreatment, BADS-SF significantly increased, and QIDS-SR significantly decreased. As seems reasonable, all participants did not change at the same rate and there was significant variability for both slope coefficients (BADS-SF variance = .364, $z = 5.13$, $p \ll .01$, QIDS-SR variance = .117, $z = 5.75$, $p \ll .01$).

A second model was estimated that added a quadratic growth component. This model fit the data well, $\chi^2(N = 578, df = 52) = 241.31$, $p \ll .05$, robust CFI = .97, robust RMSEA = .06, 90% CI [.05, .07], and significantly improved the model fit to the data. The change over time in BADS-SF and QIDS-SR had both significant linear and quadratic growth components, BADS-SF linear $b = .21$, $z = 3.65$, $p \ll .01$, BADS-SF quadratic $b = .11$, $z = 2.61$, $p \ll .05$, QIDS-SR linear $b = -.20$, $z = -2.53$, $p \ll .05$, QIDS-SR quadratic $b = -.29$, $z = -7.04$, $p \ll .05$. This can be observed in the graph depicting change in Fig. 1. While BADS-SF increases and QIDS-SR decreases, there is a change in the rate of the change indicating a significant quadratic component. These findings show that the slope of the change in BADS-SF and QIDS-SR changed over time. With the exception of the quadratic change for BADS-SF, participants changed at different rates across time. There was significant variability for all slope coefficients with the exception of the variance in the quadratic slope parameter for BADS-SF (BADS-SF linear slope variance = .36, $z = 4.67$, $p \ll .01$, BADS-SF quadratic slope variance = .01, $z = 0.35$, $p \gg .05$, QIDS-SR linear slope variance = .16, $z = 7.13$, $p \ll .01$, QIDS-SR quadratic slope variance = .05, $z = 3.81$, $p \ll .05$).

RELATIONSHIP BETWEEN ACTIVATION AND DEPRESSION ACROSS TIME

The model testing the prediction hypotheses fit the data well, $\chi^2(N = 578, df = 51) = 211.68$, $p \ll .05$, robust CFI = .98, robust RMSEA = .05, 90% CI [.04, .06]. The trajectory of the rate of change in BADS-SF, both the linear and the quadratic components, predicted the trajectory of change, both linear and quadratic, in QIDS-SR. The linear change in depression was significantly predicted by activation, BADS-SF linear $b = 3.94$, $z = 3.22$, $p \ll .01$, BADS-SF quadratic $b = 16.06$, $z = 3.52$, $p \ll .05$. The quadratic change in depression was significantly predicted by activation, BADS-SF linear $b = 5.11$, $z = 8.52$, $p \ll .01$, BADS-SF quadratic $b = 19.11$, $z = 7.89$, $p \ll .05$.

Discussion

The aim of the current study was to examine how a BA intervention impacted improvements in depression within a large clinically diverse sample. We examined the nature of change in activation and depression across time and whether the process variable exerted an effect on the outcome variable. Consistent with BA theory, results from growth curve modeling analyses supported our hypothesis with respect to the trajectory of activation and depression symptomatology. Over the course of BA-focused treatment, residents generally evidenced an increase in activation and a decrease in depression from baseline through posttreatment. Moreover, our hypothesis that changes in activation would predict changes in depression was likewise supported. Specifically, the rate of change in activation predicted the rate of change in depression.

Results showed that the trajectory of activation and depression growth across treatment was accurately and parsimoniously characterized as both linear and quadratic in our comorbid sample. As stated above, overall, residents experienced a steady increase in activation and a steady decrease in depression between baseline and posttreatment. Visual inspection of the trajectory of activation suggested a consistent rate of change between Weeks 1 and 4 for both variables. However, the group's changes in activation suggested a different rate of change between baseline to Week 1 and Week 4 to posttreatment. Visual inspection of the course of depression is consistent with the trend in repeated measures of activation.

A change in the rate of change between baseline and Week 2 may be attributable to differences in residents' environments as a result of initiating residential services. Residential treatment represents a higher level of care compared to services provided in the community (e.g., outpatient care), and is typically recommended for individuals with greater severity of symptoms and poorer functioning (Management of MDD Working Group, 2016). Given their clinical presentation (i.e., levels of activation and depression), BA theory suggests that residents' environments were not characterized by diverse and stable sources of RCPR that could pull for and maintain healthy, nondepressed behaviors. Upon entering residential care, residents at all levels of severity began the activation-focused intervention and daily treatment activities that were designed to support the work of reengaging residents in their lives. As described above, for individuals with poorer functioning, the work of

activating residents centered upon reestablishing basic routines (e.g., hygiene). Prior research has demonstrated that behavioral interventions targeting activation can produce improvement among severely depressed adults (Dimidjian et al., 2006). Thus, a move from environments not characterized by contingencies to support behavior change to an environment designed to foster behavior change may account for the relatively rapid rate of change in activation observed between baseline and Week 1. Rapid improvements may also be due, at least in part, to the sudden-gain phenomenon observed in the context of cognitive-behavioral treatment for depression. Some individuals show sudden, large improvements that account for a substantial portion of total improvement and are associated with more favorable outcomes (e.g., Kelly, Roberts, & Ciesla, 2005).

Treatment plans were informed by individually tailored activation hierarchies. As such, activities lower on lists were targeted at the outset of treatment. As treatment progressed, activities higher on individual hierarchies, which are by definition more difficult to complete, become the focus of intervention. Thus, a slowdown in the rate of change in activation may have been due to the natural progression of hierarchy-driven activation-focused treatment. The changes observed in depression during the baseline to Week 2 period is consistent with BA theory as an increase in activation is expected to correspond with a subsequent decrease in depression. With respect to the observed trend in activation and depression between Week 4 and posttreatment, it is important to note that the rate of change reflected is based on change that occurred during varied periods of time. The average resident had a length of stay of about 6 weeks. Thus, it is possible that inclusion of repeated measures between Week 4 and posttreatment may have reflected linear as opposed to quadratic change.

Evaluation of random effects (in this case, variances of the slopes) suggested significant differences in the rates of change across residents sampled with the exception of quadratic change for the BADS-SF. In other words, individual residents varied considerably with respect to the rate of linear activation, linear depression, and quadratic depression change they experienced over time, with some residents demonstrating a relatively slower (less-steep slope) or more rapid (steeper slope) rate of change compared to others. The BA model does not assume that change in activation and depression will occur at the same or even a similar rate across individuals. Given that it is grounded in the philosophy of contextualism (Kanter et al., 2009), BA assumes that the effectiveness of change efforts will be determined by the specific needs, context, and history of the individual receiving treatment. A provider and resident may collaboratively decide on an activation plan and find that it does not result in the desired behavior change because it does not meet the needs of the resident. The process of finding appropriate individually tailored interventions can contribute to the slowdown in the rate of change in activation.

Future research should examine factors that may predict individual differences in the rate of change in activation and depression over time. This sample includes residents who were at different developmental stages. It may be useful to examine whether younger and older adults differ with respect to the rate at which they become activated over the course of treatment. This sample also includes individuals with varied clinical presentations. Examining whether factors such as co-occurring diagnoses predicts individual differences in the rate of change may also be useful. Examining predictors of the rate of change, as well as of higher or lower starting points in process and outcome variables, may guide efforts to increase the effectiveness of activation-focused interventions.

Findings that the rate of change in activation predict the rate of change in depression lends further support to the theory that activation may exert an effect on depression in the context of interventions designed to increase BA. These results build on prior case-based single-subject design research (e.g., Folke et al., 2015; Santos et al., 2017) that suggested that activation led to changes in depression in BA treatment. Activation is thought to be engaged in treatment directly through activity scheduling and structuring techniques. Other techniques that comprise BA packages are conceptualized to help support the work of activity scheduling and structuring. From a clinical standpoint, these results suggest the utility of remaining focused on activity scheduling/structuring and generally implementing BA techniques in a concerted manner from one clinical encounter to another.

This study further builds on the extant literature by examining activation as a mechanism of change in a real-world clinical setting and sample. Activation-focused treatment was implemented by clinicians who are likely not as highly trained as providers in randomized controlled trials. Moreover, this sample is clinically more complex than samples included in effectiveness studies designed to examine the generalizability of treatment study findings.

An alternative view of the results of this study is that treatment techniques and mechanisms of change other than BA and the activation process may have driven symptom improvement.

Treatment techniques traditionally associated with other treatment packages were utilized in the residential program. For instance, DBT-informed interventions were implemented. It is therefore possible that mechanisms targeted by DBT-informed approaches (e.g., emotion regulation; Rudge, Feigenbaum, & Fonagy, 2017) may at least in part account for changes in depression severity. Given that measures of other viable theorized mechanisms of change were not included in this study, it is not possible to empirically examine this possibility. However, it is important to note that all therapeutic techniques implemented were carried out in the service of activating residents such that they increase their level of engagement in their worlds. In other words, although the specific function varied from one treatment component to another, the unifying function and consistent focus of treatment across components was activation. Given this focus, the treatment package is conceptualized as BA.

Medication use is a potential confound in the current study. It is possible that the effects of residents' medication use led to changes in their levels of activation, rather than or in addition to the effects of BA intervention. Unfortunately, an examination of the effect of medication use was unachievable in the current study. Future studies examining the effect of activation, or other purported psychotherapeutic mechanisms, on outcome must account for medication effects.

Future examinations of activation and other purported BA mechanisms of change will need to be conducted with diverse samples and in treatment contexts that may impact the course of treatment and outcome. Regarding the former, this includes samples that vary with regard to socioeconomic status and ethnic and racial background. The current sample was largely well educated and non-Hispanic White. In terms of the latter, it may be useful to examine whether activation changes over time and predicts outcomes in settings where treatment engagement and continuation is a challenge, such as community mental health settings (Stein et al., 2014). A limitation of the current study is that provider treatment protocol adherence data are not available. A drawback is that it is not possible to determine whether this study represents a fair evaluation of a BA-grounded intervention.

The lack of a control condition restricts the conclusions that can be drawn from the current study. For instance, given the study's within-program design, the possibility that the observed changes are due to the effect of time or the structure and activity that can typify residential treatment cannot be ruled out. Nevertheless, the observation that the rate of activation predicted the rate of depression change in the context of an intervention explicitly designed to target activation suggests the need for further examination of the impact of the intervention. A future study should include a control condition, such as standard residential care without targeted BA, to build on the current study. This would permit an examination of the extent to which activation is manipulated by the BA intervention relative to a comparison intervention and differential impact on outcomes. Moreover, it would allow inferences with regard to generalizability of study findings. For instance, whether the results stem from treatment characteristics unique to the treatment setting under study or are attributable to the BA protocol could be examined. Such a study would represent an important contribution for understanding the effect and process of residential treatment more broadly, as this literature has been (Curry, 1991) and continues to be hampered by the absence of controlled studies. A subsequent controlled study should also include follow-up assessment of activation and depression. Follow-up data would permit an evaluation of whether, in contrast to a comparison condition, BA results in lower relapse rates or sustained depression gains and whether these outcomes are associated with levels of activation.

An additional study limitation is that it does not include an objective measure of activation, such as one that is a direct product of treatment (e.g., activity hierarchy or activation homework completion; Busch, Uebelacker, Kalibatseva, & Miller, 2010). An advantage of using an objective measure of activation includes greater confidence that this important aspect of the BA change process is in fact being measured and examined. In addition, such alternative measures may result in more proximal measures of the effects of BA. That said, the BADS has been recognized as an established and validated measure of overall level of activation (Busch et al., 2010). This is reflected in the widespread use of BADS variants in studies designed to empirically evaluate BA's mechanism by independent research groups (e.g., Dimidjian et al., 2017; Folke et al., 2015). In fact, the original BADS was developed in response to the need for measures that capture BA's mechanism of change (Kanter et al., 2007) and was specifically designed to reflect changes in client activation that are the effect of in-session BA techniques.

Albeit based on self-report and distal, data suggest that the BADS is a valid measure of activation for examining BA process in the absence of alternative instruments with conceptual and methodological relative advantages. For instance,

the BADS-SF (Manos et al., 2011) was related to other measures of activation in Folke et al.'s (2015) multiple baseline study of psychiatry inpatients, in which the BA mechanism was examined. Primary measures of activation were the CUB (Hanson et al., 2013), which includes items that describe specific activation and avoidance behaviors (e.g., talked with others, attended assigned groups, attempted to pay attention in groups, stayed in room to avoid), and daily diaries of engagement in activation. Results of these measures were confirmed using the BADS-SF. Changes in BADS-SF scores for most participants were consistent with CUB scores and activation daily diaries. Together, these findings speak to the validity of the BADS as a measure of activation. Future studies should examine BA's purported mechanism using objective and proximal measures of activation.

Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

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