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A client-level session-by-session evaluation of behavioral activation's mechanism of action

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ABSTRACT

Background and objectives: Our understanding of how Behavioral Activation (BA) for depression works is limited. BA is theorized to lead to changes in depression through changes in *activation*. While distal support for activation as a mechanism has been obtained, more research is needed before definitive conclusions can be drawn. Research on mechanism should consider the appropriate time-frame for examining changes in the theorized mechanism variable and whether the proposed mechanism is expected to exert causal influence in all BA cases. These issues were considered in the current study in which a post-hoc analysis was conducted to explore BA's mechanism using single-subject data obtained at each session during the course of treatment.

Methods: Activation and depression data were obtained from a randomized-controlled trial of BA for Latinos (BAL) compared to treatment-as-usual (TAU). Cross-lagged correlations were computed to test whether activation changes preceded, co-occurred with, or lagged behind changes in depression in a sample of 21 clients (BAL $n = 14$; TAU $n = 7$). Differences among participants based on activation-depression patterns were examined.

Results: For 79% of the BAL sample, changes in activation preceded or co-occurred with changes in depression, while no clients in the TAU sample evidenced this pattern.

Limitations: Use of more proximal and objective measures of the constructs of interest and a higher dosage of BA may have served as a stronger test of the treatment's mechanism.

Conclusions: More time-sensitive measurement of changes in variables of interest is needed.

Interest in Behavioral Activation (BA) as an intervention for depression has increased over the past decade (Dimidjian, Barrera Jr., Martell, Munoz, & Lewinsohn, 2011). Aggregated findings are supportive of BA's efficacy (e.g., Cuijpers, van Straten, & Warmerdam, 2007; Ekers, Richards, & Gilbody, 2008) and it has been designated a well-established empirically validated treatment (Mazzucchelli, Kane, & Rees, 2009), according to the American Psychological Association's Division 12 Task Force on Promotion and Dissemination of Psychological Procedures (Chambless et al., 1998). BA's strength rests not only in its established efficacy but in its straightforward treatment approach and potential ease of training and dissemination (Dimidjian et al., 2011; Kanter, Puspitasari, Santos, & Nagy, 2012).

With regard to being straightforward, BA offers an inherently flexible treatment model and approach that is well-suited for use in various contexts and with distinct cultural models of illness, an attribute that suggests its effectiveness and efficacy with culturally distinct populations (Kanter et al., 2012). In fact, support for BA has been obtained in the U.S. with diverse populations, such as with Latinos (Collado, Castillo, Maero, Lejuez, & MacPherson, 2014; Kanter, Santiago-Rivera, Rusch, Busch, & West, 2010, 2015), and African-Americans (e.g., Jacob, Keeley, Ritschel, & Craighead, 2013; MacPherson et al., 2010), and in Australia (Wallis, Roeger, Milan,

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Walmsley, & Allison, 2012), Sweden (Freij & Masri, 2008), Iran (Moradveisi, Huibers, Renner, Arasteh, & Arntz, 2013), and the UK (O'Mahen et al., 2014). Empirical support is beginning to accumulate with regard to BA's purported ease of training and dissemination, and findings suggest that BA can be trained using resource sensitive and accessible methods, such as online modular training (Puspitasari, Kanter, Murphy, Crowe, & Koerner, 2013), and can be carried out by paraprofessionals (Ekers, Dawson, & Bailey, 2013; Ekers, Richards, McMillan, Bland, & Gilbody, 2011).

Less progress has been made with regard to understanding how BA works. According to BA's theory, depression is a function of losses of, reductions in, or chronically low levels of positive reinforcement. Decreased positive reinforcement for healthy behavior leads to depressed mood and decreased healthy behavior. In addition, increases in negative reinforcement occur so that the client becomes actively avoidant in an effort to prevent further negative feelings (Martell, Addis, & Jacobson, 2001). When reinforcement (both positive and negative) is altered in these ways, the client becomes inactive and experiences depression symptoms. Additional decreases in positive reinforcement (and increases in negative reinforcement) establish a cycle into deeper depression. (See Manos, Kanter, & Busch, 2010 for a fuller articulation of BA's model of psychopathology.)

BA is designed to reverse the cycle of depression through increased behavioral activation (or activation), defined here as the engagement in behavior that restores an environment characterized by diverse and stable sources of positive reinforcement, and decreased avoidance that interferes with activation (Manos et al., 2010). Techniques focus on activating clients to decrease avoidance and re-engage in life, in ways that are specific to the client's values and goals, and to help the client re-establish and sustain contact with positive reinforcement to prevent relapse. The theoretical bases of two major BA variants, BA by Martell et al. (2001) and Brief Behavioral Activation Treatment for Depression (BATD) by Lejuez, Hopko, and Hopko (2001) are similar. They both share the underlying assumption that activation should mediate changes in depression (e.g., Hopko, Lejuez, Ruggiero, & Eifert, 2003; Martell et al., 2001). In other words, the degree to which a client becomes more active and less avoidant over the course of therapy should directly lead to decreased depressive symptoms and improved mood (Kanter, Mulick, Busch, Berlin, & Martell, 2007).

BA's underlying model of depression pathology has been supported, at least partially, in a variety of research studies (e.g., Armento & Hopko, 2007; Carvalho & Hopko, 2011; Hopko, Armento, Cantu, Chambers, & Lejuez, 2003; Lewinsohn & Amenson, 1978; Lewinsohn & Graf, 1973; Lewinsohn & Libet, 1972; MacPhillamy & Lewinsohn, 1974). Less research, however, has demonstrated BA's mechanism over the course of treatment. As discussed by several authors (e.g., Borckardt et al., 2008; Gaynor & Harris, 2008; Hollon, DeRubeis, & Evans, 1987; Kazdin, 2007), to demonstrate a treatment mechanism, the temporal sequence of change must be established, such that there is evidence that change in the mechanism variable (in this case, activation) temporally preceded change in the outcome variable (in this case, depression). Thus, support for BA's mechanism would be provided by studies which show that when clients are less active, they subsequently are more depressed, and when clients are more active, they subsequently are less depressed.

Initial support for behavioral activation as an active component of treatment was observed in research aimed at disentangling the active components of Cognitive Therapy (CT) for depression. In the component analysis of CT conducted by Jacobson et al. (1996), depressed clients were randomly assigned to the full CT package designed to address core schemas, activity scheduling plus cognitive restructuring to address automatic thinking, or activity scheduling (BA) alone. Results demonstrated that the CT package did not produce better outcomes compared to BA at termination (Jacobson et al., 1996) or at 2-year follow-up (Gortner, Gollan, Dobson, & Jacobson, 1998), suggesting the importance of techniques directly targeting activation to produce change in depression.

The influential findings by Jacobson et al. (1996), however, stand in contrast to the early and frequently cited study by DeRubeis and Feeley (1990) which supported not only the CT package but its hypothesized mechanism of change as well. They examined whether CT-concrete methods, in other words specific CT techniques (e.g., examining evidence for and against client thoughts), and CT-abstract techniques, in other words less concrete work (e.g., providing the treatment rationale), predicted treatment outcomes. Aggregating client data obtained early in treatment (Session 2) from 25 CT cases, they found that CT-concrete techniques, but not CT-abstract methods, delivered early in therapy predicted later depression change. However, several authors since then (e.g., Ilardi & Craighead, 1994; Longmore & Worrell, 2007) noted that these early sessions primarily focus on the use of behavioral, as opposed to cognitive, techniques according to the CT manual (Beck, Rush, Shaw, & Emery, 1979). A review of data on the time course of change across a number of CT trials showed that most depression change takes place during early sessions when therapists were primarily expected to implement behavioral techniques (Ilardi & Craighead, 1994), further suggesting that activation may plausibly account for change. Recent BA research has lent additional support. Ryba, Lejuez, and Hopko (2014) conducted a post-hoc analysis of the causal relationship between structured activities (i.e., quantity of activities and proportion of activities completed) and depression. The authors concluded that although the specific quantity of completed activities was not causally related to reductions in depression, participant compliance with assigned activities was causally associated with improvements.

Some research on BA, however, has not been supportive of activation as a treatment mediator. For example, Jacobson et al. (1996), using the methodology of DeRubeis and Feeley (1990), found that early change in cognition predicted later change in depression in BA in the component analysis study, while early change in activity predicted later change in depression in cognitive therapy. Mixed findings suggest that definitive conclusions about activation as a mechanism of change have yet to be reached, but they also raise two issues surrounding mechanism analysis.

First, the results to date lead to the question about the appropriate time-frame during which a theorized behavioral mechanism, such as BA's, is expected to exert its influence. Is change in the mediator variable best captured over the course of 10 weeks or 1 week? The DeRubeis and Feeley methodology (briefly described above) requires that a mechanism be instantiated in a single therapy session (i.e., Session 2), and this single instantiation is required to predict depression change over the remainder of therapy. There is no lee-way for session-by-session variability to occur in the strength of the mechanism. For example, it is reasonable to assume that over the course of successful BA, a client will experience weeks of higher activity and weeks of lower activity. If a week of low activity were to be selected as the data point to predict subsequent depression change over the entire course of therapy, BA's mechanism is unlikely to be supported even if activation was in fact an active ingredient that led to improvements throughout the course of the client's treatment. Thus, the DeRubies and Feeley method implemented by Jacobson et al. (1996) may present an incomplete account of, or lead to a premature conclusion about the nature of activation's relationship to depression change for a given sample. The analyses of more proximal relations may help resolve the measurement time frame issue. For instance, one could ask: In a

one-week time frame, does activation after Session N predict depression change after Session N + 1?

Second, we consider whether we expect BA's theorized mechanism to have causal impact across all BA clients or only some clients. BA's underlying behavioral theory does not require that activation is the *only* source of causal influence over depressive symptoms. Rather, it suggests that activation is one plausible, and easy to implement, mechanism (Kanter & Puspitasari, 2012; Manos, Kanter, & Luo, 2011). Findings to date do suggest that BA's model of psychopathology and treatment fits with the nature of depression for a meaningful number of clients, but perhaps not all. Gaynor and Harris (2008) conducted an early evaluation of BA's theorized mechanism through a single-subject mediation analysis, which required documentation that the client received treatment, improved during treatment, showed change on the mediator variable that occurred at the expected time given the treatment protocol, and that change on this variable temporally preceded a substantial portion of the clinical improvement. Based on these criteria, they concluded that for two of the four clients, increased activation could plausibly explain improvements in depression. Similar findings were obtained with client-level session-by-session analyses of two clients in the validation study of the Behavioral Activation for Depression Scale—Short Form (BADS-SF; Manos et al., 2011), a measure of activation and avoidance. For one client, change in depression lagged change in activation by one week. For another, change in the mediator and outcome variables seemed to occur during the same temporal window. More recently, using group-level methodology, Collado et al. (2014) examined whether depressive symptoms concurred with or lagged behind activation by one session as part of an evaluation of BATD (Lejuez et al., 2001) in a small sample of Latinos ($N = 10$). While increases in activation corresponded to decreases in depression when both measures were obtained concurrently, changes in activation at Session N did not precede changes in depression at Session N + 1.

While it is useful to aggregate client data and look at the degree to which a mechanism is statistically supported at the group-level, another strategy is to look at the degree to which a mechanism is supported at the level of the individual client. Such an analysis may in fact have additive value for clinicians, for whom the likelihood that a treatment mechanism is valid for a specific client is a question of interest. We therefore conducted a post-hoc analysis to explore BA's mechanism using data obtained from an RCT of BA delivered in a Latino community clinic (BA for Latinos or BAL) compared to treatment-as-usual (TAU) in which BAL more successfully retained Latino clients in treatment, and for which BAL may have led to greater improvement among those clients more successfully engaged and retained in treatment (Kanter et al., 2015). In particular, this paper presents findings using weekly session-by-session data.

1. Method

1.1. Participants

Data were obtained from a randomized hybrid efficacy and effectiveness trial in which 43 clients were randomized to either BAL ($n = 21$) or to TAU ($n = 22$) at a community health center located in a large U.S. Midwestern urban center (Kanter et al., 2015). Clients were primarily monolingual Spanish-speakers recruited through the behavioral health clinic of the center. Participants included in the study: a) self-identified as Latino, b) were between 18 and 65 years old, c) obtained a depression severity score of 16 or higher on the first 17 items of the 25-item modified Hamilton Rating Scale for Depression (HRSD; Miller, Bishop, Norman, & Maddever, 1985), and d) met *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev. [DSM-IV-TR]; American Psychiatric Association, 2000) diagnostic criteria for major depressive disorder according to the Mini International Neuropsychiatric Interview version 5.0.0 (MINI; Sheehan et al., 1998). Excluded participants reported: a) a problem that required immediate inpatient hospitalization, b) organic brain syndrome or intellectual or developmental disability according to medical records, c) probable alcohol abuse, d) a lifetime diagnosis of psychosis or bipolar disorder as indicated by the MINI, e) a current diagnosis of panic disorder as indicated by the MINI, or f) use of an antidepressant medication at the time of eligibility assessment. Significant differences were not observed between the treatment groups on randomization or other demographic and clinical variables at the start of treatment.

The current study's sample is comprised of 21 clients, 14 of whom were randomized to BA and 7 to TAU during the trial. These participants provided at least 5 data-points for the analyses (described below). These clients tended to be female ($n = 17$, 81%) with an average age of 39.7 years ($SD = 10.11$), born abroad ($n = 18$, 85.7%), and of Mexican origin ($n = 15$, 71.4%). The rest of the sample was of Puerto Rican origin ($n = 4$, 19%), or identified with another ($n = 1$, 4.8%) or did not endorse a country of origin ($n = 1$, 4.8%). Over half of the sample was married or in a common law relationship ($n = 13$, 61.9%) and unemployed ($n = 11$, 52.4%), with an annual income of $20,000 or less ($n = 12$, 57.1%). The average Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) score was 29.71 ($SD = 8.89$), with under half of the sample reporting high depression severity on the BDI-II ($n = 10$, 47.6%).

1.2. Measures

1.2.1. Diagnoses

The MINI v. 5.0.0 (Sheehan et al., 1998) is a brief structured interview designed to evaluate whether diagnostic criteria for Axis I disorders are met. The MINI is comparable to the gold standard Structured Clinical Interview for DSM Disorders with regard to depression diagnosis sensitivity (0.96) and specificity (0.88), has demonstrated good inter-rater reliability ($k = 1.0$) and test-retest reliability ($k = 0.87$; Sheehan et al., 1998). The sensitivity and specificity of the Spanish version of the MINI for major depression when compared to the gold standard psychiatrist is 94.1 and 62.2, respectively (Bobes, 1998).

1.2.2. Depression severity

As recommended by Dimidjian et al. (2006) and Bobes et al. (2003), depression severity was assessed using the first 17-items of the 25-item modified HRSD (Miller et al., 1985). Good internal consistency (0.72); inter-rater reliability (0.99); content, concurrent, and discriminant validity, and a good fit with the English-language factor structure has been observed for the Spanish version (Ramos-Brieva & Cordero-Villafáfila, 1988; Ramos-Brieva, Cordero Villafáfila, & Yañez Sáez, 1994). The scale's internal consistency (α) was 0.7 in the current study. The self-report 21-item BDI-II (Beck et al., 1996) was used to measure depression severity throughout the course of treatment. Translated and validated with bilingual clinical and college student samples (Novy, Stanley, Averill, & Daza, 2001; Wiebe & Penley, 2005; respectively), the Spanish version has demonstrated good internal consistency (0.91–0.95), no significant language effect (Wiebe & Penley, 2005), and a high correlation between responses obtained using an English and Spanish language version (Novy et al., 2001). The scale's internal consistency (α) in the current study was 0.90. Participants were asked to rate items based on their experiences over the past two weeks, including the day during which the items were rated.

1.2.3. Activation

Given that the 9-item English version of the BADS-SF has shown stronger psychometric properties relative to the original 25-item BADS (Kanter et al., 2007; Manos et al., 2011), we aimed to achieve a Spanish translation of the BADS-SF as part of this project. Original BADS items were translated into Spanish using back translation (using an independent translator), a recommended method for producing a different language version of an English language instrument (Berkanovic, 1980) following procedures consistent with the World Health Organization's process of adaptation and translation of instruments (World Health Organization, 2016). Participants were asked to rate BADS-SF items based on their experiences over the previous week, including the day during which the items were rated.

An initial evaluation of the psychometric properties of the Spanish version of the BADS-SF with 173 Spanish-speaking U.S. Latinos supported the measure's internal consistency ($\alpha = 0.79$) and construct validity. In particular, the measure correlated in the expected directions with a measure of depression (Center for Epidemiologic Studies Depression Scale [CESD]; Radloff, 1977; $r = -0.67$, $p < 0.001$) and a measure of physical health (Short Form 36-item Health Survey, Version 2, Physical Health Component [SF-36v2 PCS]; Ware, 2000; $r = 0.56$, $p < 0.001$) and distinguished between clinical ($n = 121$) and non-clinical ($n = 64$) samples, $t(171) = 6.58$, $p < 0.01$, $\eta 2 = 0.2$, with greater levels of activation observed among participants in the non-clinical range. Higher scores are indicative of higher levels of activation. The internal consistency (α) of the scale in the current study was 0.6. Although the behavioral activation scale is comprised of two subscales, activation and avoidance, the current study evaluates BA's mechanism using the total scale scores. Consistent with English-language BADS-SF findings, evidence supports the use of the Spanish BADS-SF total scale without scoring the two factors of activation and avoidance as separate subscales. The scale's two factors help with understanding the total scale score as a combination of increased activation and decreased avoidance.

1.3. Procedures

1.3.1. Assessment and treatment

The Institutional Review Boards of the University of Wisconsin-Milwaukee and the community clinic approved the study protocol. Clinic providers referred clients to the study assessor, who informed potential clients about the nature of the larger study, conducted a brief eligibility screening, and scheduled a clinical evaluation with potentially qualified clients to determine fit based on inclusion and exclusion criteria contingent on obtaining informed consent. Eligible clients were randomized to condition, which was balanced on gender, marital status, and depression severity, and subsequently assigned to a study therapist.

The BAL treatment protocol was based on the BA model by Martell et al. (2001). Treatment consisted primarily of scheduling and structuring activities to increase client contact with positive reinforcement and helping the client effectively address challenges for activity completion. On-going assessment of values was emphasized to guide the selection and scheduling of activities, which was one strategy for providing treatment sensitive to the client's cultural needs over the course of therapy. TAU therapists provided treatment as they would outside of the study context, and the treatment implemented consisted of a diverse set of techniques grounded in therapists' respective theoretical orientations and training. Treatment for all clients consisted of up to 12 sessions which were typically 50 minutes long. However, scheduling challenges commonly observed in the population were accommodated by extending treatment completion time on a case-by-case basis.

1.3.2. Data analyses

Following the procedures detailed in Borckardt et al. (2008), we investigated the real time covariance of behavioral activation and depression scores using a sample of participants who received depression treatment in a trial in which BAL was compared to TAU. Specifically, we used cross-lagged correlations to test whether changes in behavioral activation were better considered as predictors or outcomes of depression scores. All participants who reported activation and depression on at least 5 occasions over the course of treatment were included in the current study's final sample, as per Borckardt et al.'s recommendation. As such, the final sample consisted of 21 of the 43 trial clients. We then used simulation modeling analysis for each participant by first computing the cross-lagged correlations at Lag -1, Lag 0, and Lag 1 (where Lag 1 is where an observation at Time N predicts the next observation at Time N + 1). A significant correlation at Lag 1 would indicate that activation significantly predicts depression in the next session, a significant correlation at Lag 0 would indicate a significant concurrent relationship between activation and depression, and a significant correlation at Lag -1 would indicate that activation is predicted by depression in the previous session.

After computing the cross-lagged correlation estimates, for each participant, we calculated the autocorrelation estimates for both the BADS-SF total score and BDI-II and used these parameters to simulate and compute the cross-lagged correlations for 10,000 pairs of data streams (Borckardt et al., 2008). For each simulated data stream, we compared the observed cross-lagged correlations to the original cross-lagged correlation values to determine the empirical p-value. Finally, we corrected for multiple comparisons using the Benjamini-Hochberg procedure (Benjamini & Hochberg, 1995).

Key to supporting BA's theory were findings of significant negative correlations at Lag 1. Significant correlations showing that activation predicts depression in the next session indicate that increased activation significantly predicted decreased depression or that decreased activation predicted increased depression at the next session ("BA-positive"). In contrast, significant positive correlations at Lag 1 would represent data contradicting BA's theory ("BA-negative"). These would suggest that increases in activation predicted increases in depression or that decreases in activation predicted decreases in depression at the next session.

Likewise, significant Lag -1 negative correlations indicate that decreased depression at one session predicted increased activation at the next session or increased depression at one session predicted decreased activation at the next session ("BA-negative"). Significant positive Lag -1 correlations suggested that increases in depression predicted increases in activation, findings that are difficult to interpret, requiring some speculation, so they are left uninterpreted.

A significant negative correlation at Lag 0 (a concurrent relationship between activation and depression) was interpreted as "BA-consistent" but not "BA-positive." Although there may have been unmeasured temporal precedence of one variable over the other within these one week windows, suggesting either "BA-positive" or "BA-negative" findings, interpreting significant negative concurrent correlations as "BA-consistent" rather than inconclusive is consistent with much previous literature on BA's theory which involved cross-sectional, concurrent correlations interpreted as supportive of the theory (Manos et al., 2010). Significant negative Lag 0 correlations could represent increases in activation occurring concurrently with decreases in depression or decreases in activation occurring concurrently with increases in depression. Significant positive correlations at Lag 0 were BA-negative as these show that activation and depression increased during the same period of time.

Additional analyses were carried out to explore potential differences between participants based on patterns of activation and depression observed. Chi-square tests of independence were conducted to examine potential associations between patterns of change and participant demographic and clinical characteristics, namely gender, marital status, country of origin, employment status, depression severity (HRSD and BDI-II), and response/remission status. Dichotomous variables for the patterns of interest were generated for this purpose. Participants with change patterns that were BA-positive or BA-consistent were distinguished from participants whose patterns were BA-negative, or whose data did not reveal an interpretable or a significant pattern of change.

2. Results

2.1. Baseline and treatment characteristics

Significant differences between the treatment groups were not observed for most demographic and clinical characteristics. Consistent with the report in Kanter et al. (2015), a difference was observed between the treatment groups on number of sessions attended, t (23) = 3.109, $p < 0.01$, where BA clients (M = 10.23, SD = 1.98) were found to attend a greater number of sessions than TAU clients (M = 7.27, SD = 2.87). The average number of days between sessions did not differ between conditions (TAU days between sessions: M = 13.57, SD = 3.90; BAL days between sessions: M = 14.03, SD = 3.93).

2.2. Cross-lagged correlations

Table 1 presents the results of the cross-lagged correlations between BADS-SF total scores and BDI-II scores for individual clients. Table 2 presents a summary of the results, specifically the frequency of significant correlations for each condition. At Lag 1, 6 of 14 (43%) BAL clients and 0 of 7 TAU clients demonstrated significant "BA-positive" correlations in which increased activation temporally preceded decreased depression. No BAL clients and 1 of 7 TAU clients (14%) showed a significant "BA-negative" Lag 1 correlation, in which activation increases at a given session temporally led depression increases at the next session.

Table 1
Cross-lagged correlations by treatment condition.

Client	Data points	Lag 1[a]	Lag 0[b]	Lag -1[c]
BA				
1	12	−0.59	−0.88**	−0.58
2	12	−0.60*	−0.76**	−0.79
3	11	−0.70*	−0.79**	−0.54
4	8	−0.64*	−0.66	−0.72
5	6	0.26	−0.56	0.64*
6	11	−0.27	−0.18	−0.19
7	12	−0.59	−0.79**	−0.83*
8	9	−0.97**	−0.97**	−0.93**
9	11	0.09	−0.67*	0.21
10	12	0.57	−0.63**	−0.05
11	10	−0.81**	−0.75**	−0.56
12	11	0.03	−0.10	0.17
13	8	−0.68*	−0.66*	−0.08
14	8	−0.50	−0.90**	−0.67
TAU				
15	11	0.46	−0.09	0.32
16	8	−0.56	−0.43	−0.65
17	8	−0.52	−0.48	−0.33
18	5	0.70*	0.31	0.81**
19	9	0.10	0.20	0.06
20	9	−0.19	−0.54	−0.43
21	12	−0.42	−0.19	−0.32

*$p < 0.05$.
**$p < 0.01$.
[a] BADS-SF predicts BDI-II.
[b] BADS-SF concurrent with BDI-II.
[c] BDI-II predicts BADS-SF.

Table 2
Frequency of BA-positive, consistent, and negative cross-correlations for each type of activation-depression pattern by condition.

	BAL (n = 14)	TAU (n = 7)
Lag 1 correlations[a]		
Significant negative ("BA-positive")	6	0
Significant positive ("BA-negative")	0	1
Non-significant	8	6
Lag 0 correlations[b]		
Significant negative ("BA-consistent")	10	0
Significant positive ("BA-negative")	0	0
Non-significant	4	7
Lag -1 correlations[c]		
Significant negative ("BA-negative")	2	0
Significant positive ("BA-negative")	1	1
Non-significant	11	6

[a] BADS-SF predicts BDI-II.
[b] BADS-SF concurrent with BDI-II. [c]BDI-II predicts BADS-SF.

At Lag 0, 10 of 14 (71%) BAL clients and 0 of 7 TAU clients demonstrated significant negative "BA-consistent" correlations. For these clients, strong (large effect) inverse relationships were observed between activation and depression, in which the former increased as the latter decreased. Five of these 10 clients were the same clients that also showed "BA-positive" correlations at Lag 1.

At Lag -1, 3 of 14 (21%) BAL clients and 1 of 7 (14%) TAU clients showed significant "BA-negative" correlations. Two of these clients experienced reductions in depression symptom severity that temporally led increases in activation (both BAL clients), while the other two experienced increases in depression that temporally led increases in activation (one in each condition).

2.3. Associations with specific change patterns

Overall, within BAL, 11 clients (79%) showed significant BA positive/consistent change patterns. Of these BAL clients, 5 (45%) showed positive and consistent change patterns, 5 (45%) showed only the pattern consistent with BA, and 1 (10%) showed only the BA-positive pattern. However, no clients showed these change patterns within TAU. This difference between conditions was significant, $\chi^2(1) = 9.55$, p = 0.004. Regarding participant demographic and clinical characteristics, no trends or significant findings were obtained.

3. Discussion

In the current study, BA's theorized mechanism of change was examined at the level of the individual client using weekly session-by-session data obtained from an RCT comparing BA for Latinos (BAL) to treatment-as-usual (TAU). Results suggested BA's model as a plausible mechanism of change for 79% of the BAL sample, and for none of the TAU sample. For these participants, analyses of change patterns suggested the temporal precedence of change in activation before change in depression and/or concurrent change in activation and depression, both patterns that are consistent with BA's theory of change. In other words, when these clients were more active, they were less depressed, and when they were less active, they were more depressed. They showed a clear temporal precedence of change in activation preceding change in depression ("BA-positive"), concurrent change in activation and depression that was consistent with BA but inconclusive with respect to temporal precedence within the one week window ("BA-consistent"), or both

patterns of change.

These findings are consistent with the behavioral theory of depression – the notion that change in activation may produce change in depression – and this pattern may explain both depression onset (decreased activation produces increased depression) and its treatment (increased activation produces decreased depression). Our findings also suggest that BA as an intervention may make this pattern of change in an anti-depressant direction more likely. The findings also are consistent with the notion that this model is applicable for some but not all clients and with the existence of various etiologies underlying depression (Manos et al., 2011). As such, activation may function as a mechanism of change when other treatment approaches are implemented, but it is also important to note that this mechanism may not be applicable for all clients, and other viable pathways to successful treatment exist.

Factors not related to the therapy may have influenced our findings. For instance, our measure of activation, the BADS-SF total score, may have captured changes in activation and avoidance that did not result from BA interventions. It is conceivable that a client experienced changes in activation due to extraneous changes in life events (e.g., acquisition of work that requires greater activity) or interventions outside of mental health (e.g., restrictions on activity due to a health condition). These types of extraneous factors and natural variations in mood could have also impacted depression severity ratings throughout the course of treatment. The finding, however, that BA-positive/consistent correlations were only observed with BAL clients suggests that treatment exerts a significant effect as well.

The BA model of depression is flexible with respect to the nature of the relationship between activation and depression and emphasizes the idiographic rather than one-size-fits-all nature of the phenomenon. Thus, while we explored a session-by-session approach to the mechanism, it could be the case that, for some clients, consistent activation over several weeks is required to produce change in depression. Alternately, even smaller windows of time, rather than larger, could be explored. It could be the case, for example, that activation predicts depression for a larger number of participants in the current study than the study methods allowed us to detect. Seventy-one percent of the BAL clients experienced increases in activation and decreases in depression that occurred during the same one-week period. Our measurement window of weekly assessments at each session made it impossible to observe BA-positive (or BA-negative) changes that might have occurred within a smaller time frame. For instance, given predictions for BAL clients, would activation at Day 1 predict reduced depression at Day 2 for the current sample? Likewise, perhaps activation in the morning of Day 1 would predict reduced depression at the end of the day. Alternately, it may be the case that the concurrent correlations would reduce to BA-negative findings if the time frame was tighter. For example, perhaps waking up in the morning feeling better, due to biological or cognitive shifts, leads to more activation by the end of the day.

Future studies aiming to examine BA's mechanism of action should be designed to include alternative methods of data collection to improve upon the measurement limitations of the current study. These include techniques that allow participants to provide reports on variables of interest with proximity to the experience under study and that would also allow greater sampling, such as ecological momentary assessment (Moskowitz & Young, 2006). The use of idiographic activation measures would represent more proximal measures of activation as a result of BA treatment. Self-reported activation obtained through the BADS-SF likely captures activation caused by other factors in a client's life (e.g., increased activity due to non-mental health intervention). Previous research has used the proportion or percent completion of ideographically designed homework assignments as a measure of personalized activation in BA treatment (Ryba et al., 2014).

Complimenting subjective data collection techniques with objective measures of activation and depression severity would strengthen the design of future BA mechanism of change research. Objective measurement would help circumvent problems associated with self-report. Physical activity or social activity, which are sensitive to affective illness manifestation (Faurholt-Jepsen et al., 2012), may serve as less biased measures of activation. Daily activity that capture these domains and others may be monitored using cell phones with ease and at limited expense (Faurholt-Jepsen, Frost, et al., 2014). Physical activity may also be accurately measured using global positioning system (GPS) technology, which may also provide data to understand the context of the activity (e.g., location; Maddison & Ni Mhurchu, 2009). Objective measures of depression symptom severity should also be considered. Support has been obtained for the use of sleeping heart rate (beats/min) as a supplemental objective measure of depression severity as it has been found to correlate with the HRSD-17 in a patient sample with unipolar depression (Faurholt-Jepsen, Brage, et al., 2014). These methods may be more sensitive to moment-to-moment or subtle changes in activation and depression throughout the course of treatment.

An important limitation of this study is the sample size and results require replication in larger samples. We are hopeful, however, that the current demonstration of the methodology will allow for replications in future studies and existing datasets. As weekly data on BA accumulate, it will be possible to attempt to replicate the current findings with larger samples and more fully explore moderators of these effects. We believe it is important to conduct analyses at the single subject level, thereby detailing, rather than simply controlling for, variation in what is undoubtedly a complex phenomenon, and giving clinicians important information not just on if BA is effective, but on how many and which clients can be predicted to respond to BA, and why they respond if they do.

Another limitation of this study is that different time frames were measured by the key instruments of activation and depression. While the BADS-SF asked clients to rate items based on the past week, including the current day, the BDI-II instructed clients to rate items based on the past two weeks, including the current day. Although the average between-session interval was 2 weeks for study clients, this complicates interpretation of the findings somewhat when clients had weekly sessions. We believe, however, that most clients were socialized by the session-by-session administration of measures to focus on the time frame from the previous session as the primary source of experience regardless of the BDI-II instructions. This assumption about how client's responded on the BDI-II is consistent with the previously published analyses of the relation between activation and BDI-II scores, both of which used the same methodology as in the current report (Collado et al., 2014; Gaynor & Harris, 2008), with recommendations for the session-by-session interpretation of BDI-II scores (Dozois & Covin, 2004) and with many published analyses of weekly, session-by-session BDI-II data (e.g., Hardy et al., 2005; Lemmens, DeRubeis, Arntz, Peeters, & Huibers, 2016; Webb et al., 2011). Nonetheless, future studies may consider changing the BDI-II instructions to explicitly encourage reporting on weekly experience.

It is both a weakness and a strength of this study that it was conducted with therapists who received relatively brief training and a primarily mono-lingual, Spanish-speaking Latino sample. The weakness is that the therapists who provided BA in this study were not established as competent in BA before engaging the protocol

with study clients, and it is possible that expert BA therapists would provide a stronger dose of BA and function as a better test of hypotheses about BA's mechanism. The strength is that the study demonstrates that BA is a plausible treatment for this group, and that the mechanism can be instantiated without extensive training and applied across distinct cultural groups with measurable effects on a substantial number of clients. This dovetails with one of the primary strengths of BA in general in that its greatest value may be its parsimony, ease of training, and broad applicability.

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