J. Behav. Ther. & Exp. Psychiat. 54 (2017) 93–100

Contents lists available at ScienceDirect

Journal of Behavior Therapy and Experimental Psychiatry

journal homepage: www.elsevier.com/locate/jbtep

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

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ARTICLE INFO

Article history:
Received 30 July 2015
Received in revised form
27 June 2016
Accepted 4 July 2016
Available online 7 July 2016

Keywords:
Behavioral activation
Depression
Mechanism of action
Mediators
Activation

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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http://dx.doi.org/10.1016/j.jbtep.2016.07.003
0005-7916/

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