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Behavioral Activation for the Treatment of Atypical Depression: A Pilot Open Trial

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Abstract

Psychosocial interventions for atypical depression (AD) have been relatively ignored in the clinical research literature, despite evidence that the atypical subtype of major depression is marked by earlier age of onset, longer duration of mood episode, greater symptom severity, and poorer response to pharmacologic treatment. Given the symptom profile of AD, which is characterized by mood reactivity, psychomotor slowing, and interpersonal withdrawal, we argue that a behavioral activation (BA) intervention may be particularly well suited to this population. As an initial exploration of this hypothesis, the current study presents preliminary outcome data from 10 outpatients with AD who participated in an open pilot trial of BA over a 16-week period. Overall, results provide encouraging preliminary support for the feasibility, acceptability, and efficacy of BA for AD, with significant reductions in depressive symptoms and associated improvements in functional impairment and behavioral activation level. Study results are discussed in the context of existing treatments for AD, and areas for future treatment development are highlighted.

Keywords

depression; atypical depression; behavioral activation

Although first described in 1959 (Quitkin, 2002), atypical depression (AD) was only formally introduced as a major depressive disorder (MDD) features specifier in the 4th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994). A diagnosis of AD requires that individuals meeting current criteria for a major depressive episode also endorse: (1) mood reactivity – the ability to experience at least a 50% improvement in mood following exposure to a positive event, plus a minimum of 2 remaining criteria: (2) hypersomnia – increased sleep beyond 10 hours a day, (3) leaden paralysis – the feeling that ones limbs are weighed down, (4) hyperphagia – increased appetite or weight gain, and (5) interpersonal rejection sensitivity – hypersensitivity to criticism or rejection resulting in functional impairment, such as interpersonal avoidance. This definition was derived from prior research demonstrating that individuals presenting with such “atypical” symptoms of MDD are less responsive than general MDD samples to acute (Quitkin, 2002) and continuation (McGrath et al., 2000) phase trials of certain classes of antidepressant medication.

Indeed, it has been well established that individuals with AD respond preferentially to the monoamine oxidase inhibitors (MAOIs) relative to the tricyclic antidepressants (TCAs)

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(e.g., Quitkin, 2002; Stewart, McGrath, Rabkin, & Quitkin, 1993). Yet even with the advent of the newer antidepressants such as the selective serotonin reuptake inhibitors (SSRIs) and the serotonin-norepinephrine reuptake inhibitors (SNRIs), there nevertheless remains only mixed efficacy data for the pharmacologic treatment of AD (Fava et al., 1997; Henkel et al., 2006; Quitkin, 2002; Stewart et al., 2010). Data further suggest that patients with AD may not respond to continuation trials of these newer medications, as evidenced by greater rates of post-treatment relapse relative to non-atypical forms of MDD (non-AD) (McGrath et al., 2000). A recent review concluded that "in this post-MAOI era, no novel compound or group of drugs has been clearly shown to have good efficacy in atypical depression, leaving the treatment of atypical depression as an unmet need" (Davidson, 2007; p. 10).

Poor pharmacotherapy response in AD is particularly troubling in light of data suggesting that AD may be more prevalent than its name suggests. Research has shown that those with AD may comprise anywhere from 16 – 42% of those seeking treatment for MDD (Posternak & Zimmerman, 2002), and prevalence rates have been reported to be as high as 25% in community samples of depressed persons (Angst, Gamma, Sellaro, Zhang, & Merikangas, 2002). Moreover, there is evidence that AD may actually represent a more severe and chronic form of MDD. Several studies have reported that compared to non-AD, AD is associated with earlier age of disorder onset (Agosti & Stewart, 2001; Angst et al., 2002; Matza, Revicki, Davidson, & Stewart, 2003), longer depressive episode duration (Angst et al., 2002; Posternak & Zimmerman, 2002; Stewart et al., 1993), and greater number of lifetime depressive episodes (Nierenberg, Alpert, Pava, Rosenbaum, & Fava, 1998; Robertson et al., 1996; & Stewart et al., 1993). Compared to those with non-AD, those with AD have also demonstrated greater overall depressive symptom severity (Agosti & Stewart, 2001; Novick et al., 2005), greater rates of both anxiety (Novick et al., 2005) and personality disorder (Singh & Williams, 2006) comorbidity, greater impairments in social adjustment (Agosti & Stewart, 2001; Matza et al., 2003), and greater risk of suicidal behavior (Singh & Williams, 2006).

Despite this emerging profile of AD, psychosocial interventions have been largely ignored in the clinical research literature. Given mixed evidence for efficacy of pharmacologic approaches, psychosocial interventions may be particularly useful to consider in this regard. Although large randomized controlled trials of cognitive (e.g., DeRubeis et al., 2005), behavioral (e.g., Dimidjian et al., 2006), and interpersonal (e.g., Elkin et al., 1989) interventions have not routinely excluded individuals with AD from their samples, their data are rarely reported separately (Holtzheimer & Mayberg, in press). Thus, efficacy of these interventions for the treatment of AD, specifically, is largely unknown, and assumptions of equal efficacy across depression subtypes are similarly unsupported. In the only published trial that we are aware of having evaluated outcomes for AD separately, Stewart and colleagues (1998) conducted a secondary analysis of data collected in the Treatment of Depression Collaborative Research Program (TDCRP; Elkin et al., 1989), which revealed that treatment response following cognitive therapy (CT) in an AD subsample was significantly greater than response following treatment with the TCA imipramine. However, there was no difference between CT and a pill placebo among AD participants. As such, CT outperformed a treatment (i.e., TCA) that was found to be relatively *ineffective* in prior studies of AD (Quitkin, 2002), but did not result in symptom improvement relative to a placebo.

Likewise, there have been only 2 published studies (Jarrett et al., 1999; Mercier, Stewart, & Quitkin, 1992) that have evaluated a psychosocial treatment for AD specifically, only 1 of which used a randomized controlled design (Jarrett et al., 1999). This randomized controlled trial of CT for AD (Jarrett et al., 1999) revealed equivalent rates of patient response to CT (58%) and the MAOI phenelzine (58%), which were superior to response in a pill placebo

(28%) condition. These findings were consistent with preliminary data from a pilot open trial of CT for AD, which resulted in a treatment response rate of 56% (Mercier et al., 1992). In sum, there is some mixed preliminary data to suggest that persons with AD do respond to cognitive therapy, yet response rates have been typically modest and there is a clear need for additional treatment development in this area.

Given the symptom profile of AD, we argue that a psychosocial intervention grounded in behavioral activation (BA) principles may be particularly well suited to this population. BA originated from early behavioral conceptualizations of MDD that stressed the notion that depression is marked by decreased rates of positive reinforcement and increased rates of avoidance behavior (Lewinsohn, 1974). Consistent with this perspective, researchers demonstrated that increased exposure to pleasurable and mastery activities resulted in significant clinical improvement for those suffering from MDD (Lewinsohn & Graf, 1973; Lewinsohn & Libet, 1972). Roughly within this same time period, Beck was developing his CT for MDD (Beck, Rush, Shaw, & Emery, 1979), which also included a behavioral activation component. As part of the larger CT "package," however, BA was conceptualized as a means of contributing to cognitive change necessary for depression reduction. Yet later research revealed that the behavioral component of CT may be an effective treatment for depression on its own. Indeed, in their seminal component analysis of CT for depression, Jacobson and colleagues (1996) found that BA was equally as efficacious as a full package of CT in the reduction of depression symptoms.

Following from these findings, Jacobson and colleagues (2001) suggested that, as its own treatment, BA may impact depression symptoms through the "activation" of the patient by providing exposure to natural reinforcements. In particular, they argued that BA should be expanded to include a particular focus on the consequences and outcomes of behavior for each patient, rather than making *a priori* assumptions about which behaviors should be reinforcing for all patients. This conceptualization led to the development of BA as a stand-alone treatment for MDD, designed to break the cycle of avoidance that maintains depression by engaging the patient in increased goal-directed activity and exposure to positive reinforcement contingencies. However, the activation plan is tailored to each individual based upon a personalized functional analysis, rather than indiscriminant exposure to generally pleasant events, so that access to positive reinforcement can be maximized. Some of the strategies used in this treatment include focused activation through the use of activity logs for monitoring associations between situations, behavior and mood, mental rehearsal of tasks, graded task assignment, and the regulation of basic life routines (Martell, Addis, & Jacobson, 2001).

Emerging research in general MDD samples continues to suggest that this expanded BA may be equally efficacious as a full CT package in the reduction of symptoms, and may even outperform CT in the treatment of severe depression (Dimidjian et al., 2006). Concurrently, several additional variants of BA have been developed since the 1970s (Kanter et al., 2010), including Brief Behavioral Activation Treatment of Depression (BATD; Lejuez, Hopko, & Hopko, 2001; Lejuez, Hopko, LePage, Hopko, & McNeil, 2001), which also relies on a model emphasizing contextual factors that reinforce depression, behavioral goal setting, and activity scheduling. Data reported from clinical trials of these additional variants of BA further support the efficacy of BA treatments for depression (cf. Mazzucchelli, Kane, & Rees, 2009).

Given this evidence base, and the specific symptom profile of AD, extension of BA treatments to those suffering from AD may be particularly indicated. For example, reversed vegetative symptoms of hypersomnia, hyperphagia, and leaden paralysis may all contribute to behavioral and experiential avoidance in depression. Moreover, interpersonal rejection

sensitivity, as defined in the diagnostic criteria for AD, is marked by significant avoidance of the familial, social, and occupational sphere in order to reduce perceived risk of rejection (Stewart et al., 1993). Consequently, a distinct focus on routine regulation, avoidance pattern modification, and alternative coping may be particularly useful in the treatment of AD. Furthermore, people with AD, by definition, meet criteria for mood reactivity. As such, BA may be especially well suited to the treatment of AD given that patients are more likely to respond to positive contingencies. An additional benefit of BA is that it was designed to be parsimonious, idiographic, and flexible (Jacobson et al., 1996; Martell et al., 2001). Thus, BA is arguably well matched and allows for tailoring of treatment to this unique subsample of patients who have been assigned an otherwise heterogeneous diagnosis of MDD, which is consistent with recent efforts at greater "treatment personalization" (Insel, 2007).

The current study was designed to evaluate the feasibility and acceptability of BA for AD. The aim of this research was not to develop a new variant of BA, but rather to extend the application of an existing BA intervention (Martell et al., 2001) to the treatment of AD and to evaluate this "proof of concept." Using a pre-post design, we report preliminary outcome data from 10 outpatients with AD who participated in an open pilot trial of BA over a 16 week period.

Method

Participants

Participants were 10 outpatients with AD who were referred by outpatient psychiatrists (n = 2) or were self-referred (n = 8) in response to internet advertisements. Inclusion criteria specified that eligible participants: 1) meet criteria for a diagnosis of MDD with atypical features as assessed by the Structured Clinical Interview for DSM-IV-TR axis I disorders – Patient Version (First, Spitzer, Gibbon, & Williams, 2001), 2) score greater than or equal to 30 on the Inventory of Depressive Symptomatology –Clinician version (IDS-C; Rush, Guillon, Basco, Jarrett, & Trivedi, 1996), 3) be 18 years or older, and 4) have an ability to speak, read, and understand English sufficiently well to complete study procedures. Exclusion criteria were: 1) a primary psychiatric diagnosis other than MDD with atypical features, 2) a diagnosis of bipolar I or II disorder, 3) current psychotic symptoms, 4) alcohol or substance dependence, and 5) a diagnosis of borderline or antisocial personality disorder as assessed by the Structured Clinical Interview for DSM-IV-TR axis II disorders (SCID-II; First, Spitzer, Gibbon, & Williams, 1997).

Demographic characteristics of study participants are presented in Table 1. Participants had on average 1.8 (SD = 1.75) current and lifetime comorbid axis I diagnoses, with secondary diagnoses of social phobia (n = 4; 40%), panic disorder with agoraphobia (n = 2; 20%), generalized anxiety disorder (n = 1; 10%), posttraumatic stress disorder (n = 1; 10%), specific phobia (n = 1; 10%), and binge eating disorder (n = 2; 20%). One participant (10%) met criteria for lifetime (not current) binge eating disorder, 3 (30%) met criteria for lifetime alcohol abuse, 1 (10%) met criteria for lifetime alcohol dependence, and 1 (10%) met criteria for lifetime substance dependence. Five out of the 10 participants received concurrent antidepressant treatment from their community psychiatrists; however, in order to participate, we required that patients be on a stable pharmacotherapy regimen for 6 weeks prior to study treatment entry.

Measures

Structured Clinical Interview for DSM-IV Axis I Disorders – Patient Version (SCID-I/P; First et al., 2001)—The SCID-I/P was administered at baseline to assess axis I diagnoses, conducted by the study first author or by a trained bachelors-level research

assistant. All diagnostic decisions were based on a review of the SCID-I/P between the rater and the clinical team, and diagnostic decisions were based upon consensus. Prior to administering the SCID-I/P, interviewers were required to achieve at least 90% agreement with supervisors' ratings.

Structured Clinical Interview for DSM-IV Axis II Disorders (SCID-II; First et al., 1997)—The SCID-II was administered at baseline to assess for the presence of borderline or antisocial personality disorders, for purposes of determining eligibility for participation. Only the 2 SCID-II modules relevant to those disorders were administered. As with the SCID-I/P, all SCID-II ratings were conducted by the study first author or by a trained bachelors-level research assistant. Diagnostic decisions were based on a review of the SCID-II between the rater and the clinical team, and diagnostic decisions were based upon consensus. Prior to administering the SCID-II, interviewers were required to achieve at least 90% agreement with the supervisors' ratings.

Inventory of Depressive Symptomatology – Clinician Rated (IDS-C; Rush et al., 1996)—The IDS-C is a 30-item interviewer rated measure of depressive symptom severity. Scores range from 0 to 84, with the following severity ranges: none (0 – 11), mild (12 – 23), moderate (24 36), severe (37– 46), and very severe (47 – 84). In addition to its strong psychometric performance in several large-scale clinical trials (e.g., Fava et al., 2003), one benefit of the IDS-C is that it reliably assesses all 5 symptoms of AD. IDS-C ratings were conducted by bachelors-level research assistants who were trained and supervised by the study authors. All post-treatment assessments were conducted by research assistants who were blind to study aims and procedures. Reliability of IDS-C ratings was high, with ICCs = .94 – 1.00 for agreement between expert faculty and research assistant ratings.

Behavioral Activation for Depression Scale (BADS; Kanter, Mulick, Busch, Berlin, & Martell, 2007)—The BADS is a 25-item self-report scale that was developed to assess avoidance and activation behaviors that represent specific treatment targets in BA interventions for depression. Items are rated on a scale from 0 (not at all) to 7 (completely), with total scores ranging from 0 to 175. Higher scores reflect greater overall levels of "activation." The BADS also consists of 4 subscales: activation, avoidance/rumination, work/school impairment, and social impairment (Kanter et al., 2007). Of note, scoring is reversed for 3 of the 4 subscales (avoidance/rumination, work/school impairment, and social impairment) so that lower ratings are considered to reflect better functioning in these areas. Demonstrating strong psychometric performance in both analogue and community samples of people with elevated depressive symptoms (Kanter et al., 2007; Kanter, Rusch, Busch, & Sedivy, 2009), the BADS has also been shown to correlate with depressive symptom levels over course of treatment in two case studies evaluating BA for depression (Kanter, Dieguez-Hurtado, Rusch, Busch, & Santiago-Rivera, 2008; Manos et al., 2009) and one study evaluating CT in a partial hospitalization program (Christopher, Jacob, Neuhaus, Neary, & Fiola, 2009). Internal consistency reliability of the BADS was high in the current study (Cronbach's $\alpha = .88$).

Sheehan Disability Scale (SDS; American Psychiatric Association, 2000)—The SDS is a brief, 3-item questionnaire that assesses overall level of functional impairment. Using a scale from 0 (not at all) to 10 (extremely), patients are asked to rate the degree to which their symptoms of depression affected their functioning in the areas of: 1) work/school, 2) social life, and 3) family life/home responsibilities. The 3 items are summed into a single dimensional measure of global functional impairment, with total scores ranging

from 0 (unimpaired) to 30 (highly impaired). The SDS demonstrated good internal consistency reliability in the current study (Cronbach's α = .78).

Client Satisfaction Questionnaire-8 (CSQ-8; Larsen, Attkisson, Hargreaves, & Nguyen, 1979)—The CSQ-8 was administered at post-treatment to assess overall patient satisfaction with treatment received. This brief questionnaire contains 8 Likert-type items, with total scores ranging from 1 to 32. Higher scores reflect greater satisfaction with treatment received. The CSQ-8 has been used extensively in psychosocial treatment research, and data from previous studies support its reliability and validity (Larsen et al., 1979; Nguyen, Attkisson, & Stegner, 1983). In the current study, internal consistency reliability of the CSQ-8 was high (Cronbach's α = .96).

Procedure

As reported above, patients were self-referred or referred by outpatient psychiatrists to participate in this IRB-approved open pilot trial. Prior to baseline assessment, patients provided written informed consent to participate. The SCID-I/P and SCID-II were administered at baseline for eligibility purposes. The IDS-C, BADS, and SDS were administered at pre- and post-treatment, and were used as the main outcome measures for the current study. The study treatment was delivered by the study first author (LMW), who was supervised by the study's coauthor (IWM) on a weekly basis. Treatment was provided over the course of 16 weeks (weekly for the first 12 weeks and biweekly for the last 4 weeks). If clinically indicated, patients could receive an additional 2 scheduled sessions, for a total of 14 – 16 individual treatment sessions. Along with post-treatment outcome measures, the CSQ-8 was administered immediately post-treatment. Supplemental open-ended questions were included with the CSQ-8, asking participants to share their experiences and overall satisfaction with the treatment program. Assessment and treatment were conducted in an outpatient research clinic located in an academically-affiliated private psychiatric hospital.

Treatment

Following the BA intervention developed by Martell et al. (2001), we provided 14 – 16 sessions of individual BA in an open pilot trial format. As specified in the manual, treatment targeted: 1) psychoeducation and behavioral monitoring, 2) avoidance modification and behavioral scheduling, and 3) behavioral maintenance and relapse prevention. Although already compatible with BA as manualized, we further focused the content of the treatment to directly address some of the unique needs associated with AD. The treatment program is described in more detail below. For ease of description, session content is provided topically; however, consistent with the flexible nature of this treatment, it should be noted that content typically overlapped (e.g., behavioral scheduling was assigned concurrently with behavioral monitoring; activity monitoring and functional analysis were utilized throughout treatment, etc.).

Psychoeducation and behavioral monitoring—The initial treatment focus was on familiarizing the patient with the treatment rationale, behavioral assessment and graded task assignment and routine regulation. To address these targets, treatment focused on: a) Rapport building and orientation to the behavioral model. The therapist provided psychoeducation concerning the associations between withdrawal/avoidance and depression, and the overall rationale for a targeted behavioral treatment. In particular, by eliciting examples from the patient, the therapist emphasized the importance of decreasing problematic mood-dependent behaviors and of committing to behavioral change regardless of internal mood state. Discussion also highlighted the importance of graded exposure to natural reinforcement for purposes of promoting behavioral activation. b) Activity

monitoring homework. Activity monitoring was introduced to patients at the end of the first session, and continued throughout the course of treatment. The purpose of this activity was to help patients identify the associations between situations, behaviors, and mood. Information gathered from activity charts was reviewed in each session, with a particular focus on the idiographic nature of these data. c) Functional analysis of behavior. Information gathered from activity charts was also used in the functional analysis of behavior. In particular, the therapist and patient worked together to identify antecedents and consequences of specific behaviors in relation to depression symptoms. A particular emphasis was placed on the identification of avoidance behaviors that contribute to mood symptom maintenance, and identification of potential alternative coping behaviors that may break the cycle of avoidance and depression. To identify behaviors of interest, we used the Trigger-Response-Avoidance-Pattern and Trigger-Response-Alternative-Coping (TRAP/TRAC) worksheets developed by Martell et al. (2001).

Avoidance modification and behavioral scheduling—Treatment also focused on skill development and implementation of a behavioral plan. These treatment aims were targeted through: a) Behavioral goal setting. The therapist and patient identified appropriate short-term and long-term behavioral goals within several life areas, and selected a limited number of short-term goals (e.g., 3–4) to target in treatment. b) Avoidance modification via graded task assignment. The therapist and patient worked together to develop a behavioral hierarchy to promote a graded, step-wise approach to increased activity and regulation of daily routines. An emphasis was placed on developing a behavioral plan that reduces, or eliminates, mood-dependent behavior and introduces naturally reinforcing alternative behaviors (as identified using the TRAP/TRAC exercise). As recommended by Martell et al. (2001), strategies used to help implement the behavioral plan included mental and/or verbal rehearsal of assigned tasks, role playing, problem-solving around obstacles to behavioral activation, and management of situational contingencies to help overcome potential obstacles. c) Homework practice. A critical element of treatment was the application of graded task assignments and strategies learned as homework between sessions, as well as activity monitoring throughout.

Behavioral maintenance and relapse prevention—Following successful implementation of the skills above, treatment sessions focused on maintenance of treatment gains and development of a relapse prevention plan. To achieve these goals, treatment included: a) Progress review. The therapist and patient reviewed progress through the behavioral hierarchy, and identified problems and continued to problem-solve around potential obstacles to maintaining the behavioral plan. b) Return to long-term goals. Once the patient had used the behavioral plan to master short-term goals, the therapist and patient discussed ways to use behavioral skills and new knowledge to gradually work longer-term goals identified earlier in treatment. c) Relapse prevention plan. The patient and therapist worked together to identify prodromal behavioral indicators and symptoms of depression relapse. They then developed a relapse prevention plan focused on monitoring such early warning signs, applying behavioral strategies to aid in symptom management, and identifying when and how to seek help.

Specific Treatment Considerations for Atypical Depression

Specific content of the treatment was further tailored to address some of the unique needs of this patient population, when appropriate. As noted above, these strategies are not incompatible with existing BA (cf. Martell et al., 2001), but rather were especially attended to given the symptom profile of AD. In particular, the therapist directly addressed: 1) oversleeping and overeating as avoidance behaviors, 2) interpersonal rejection sensitivity as an obstacle to behavioral activation, and 3) harnessing mood reactivity.

Oversleeping and overeating as avoidance behaviors—Identified through initial clinical assessment and through activity charting, we paid close attention to associations between fluctuations in mood and the specific AD symptoms of hypersomnia and overeating. In particular, the therapist and patient worked together to identify the mood-dependent nature of these behaviors, and the short- versus long-term consequences of engaging in such. Through functional analysis, patients often concluded that such behaviors, while rewarding in the short-term, functioned as a means of (experiential and behavioral) avoidance, thereby perpetuating depression. In this way, overeating and oversleeping were treated not as symptoms that "happened" to patients, but rather as behaviors that they actively engaged in. Using the TRAP/TRAC exercise, the therapist and patient identified alternatives to oversleeping and overeating, and included these alternatives as behavioral goals. In fact, "getting out of bed" at an agreed-upon time was typically the first behavioral goal identified in treatment, as progress toward other behavioral goals was dependent upon this step. Often, this goal involved management of situational contingencies, such as scheduling an activity with another person. Of note, this emphasis of treatment is in keeping with the emphasis on routine regulation in standard BA (Martell et al., 2001).

Interpersonal rejection sensitivity as an obstacle to behavioral activation—Second to mood reactivity, which is required for the diagnosis of AD, interpersonal rejection sensitivity is the most commonly reported symptom of AD, with endorsement rates among 71 – 86% of patients (Angst et al., 2002; Derecho, Wetzler, McGinn, Sanderson, & Asnis, 1996; McGrath et al., 1992; Posternak & Zimmerman, 2002). Given that rejection sensitivity is associated with behavioral avoidance (Stewart et al., 1993) and ruminative thinking (Pearson, Watkins, Mullan, & Moberly, 2010), it is important that the therapist address this particular feature of AD as a potential obstacle to implementing the larger BA plan. For example, a patient may have a behavioral goal of "increased exercise," but has avoided the gym secondary to fear of evaluation from other gym goers. In this case, the graded task assignment was adjusted to address this potential obstacle, and to include graded exposure to feared interpersonal scenarios, similar to behavioral treatments for social phobia (e.g., Butler, 1985). In this way, exposure to situations that trigger rejection sensitivity was seen not only as a potential obstacle to behavioral activation, but also as an ultimate behavioral goal. With respect to rumination, we applied the perspective, set forth by Martell and colleagues (2001), that treats rumination as an "avoidance behavior." Through functional analysis, the therapist and patient worked together to identify the function and consequences of ruminative thinking. Through the TRAP/TRAC exercise, alternatives to rumination were identified, often with the aim of reorienting the patient to the present moment and how chosen alternative behaviors are linked to short- and long-term goals.

Harnessing mood reactivity—Given presence of mood reactivity in this population, as noted previously, BA may be especially well suited to the treatment of AD given that patients are more likely to respond to positive contingencies. Early in treatment, the therapist helped guide the patient to identify situations and behaviors not only associated with worsened mood, but also with more improved mood states. Following from assessment of patterns of mood reactivity for each patient, the therapist and patient used the functional analysis of behavior to not only understand behaviors that perpetuate depression, but also to better understand behaviors that may result in more positive outcomes. Similar to the idea that hypersomnia and hyperphagia may not simply be symptoms that befall the patient, but rather behaviors that the patient can or cannot choose to engage in, we emphasized that mood reactivity may not necessarily be limited to external positive stimuli outside of the patient's control (e.g., a compliment from a supervisor at work). Rather, the therapist and patient problem-solved around ways the patient could actively engage in activities that may

result in a similar positive outcome (e.g., completing a work task after much procrastination, resulting in self-satisfaction), and built those into the behavioral plan.

Results

Nine of the 10 participants completed the intervention. The one study drop-out discontinued treatment after 7 sessions, but completed post-treatment assessments at that time. Consistent with an intent-to-treat approach, these data are included in all outcome analyses. For all 10 study participants, average session attendance was 13.3 (*SD* = 3.6) out of the 14 – 16 sessions offered.

Means and standard deviations for all study variables are presented in Table 2. IDS-C scores revealed that, on average, participants fell into the "severe" category of depressive symptoms upon study entry and the "mild" category upon study completion. Results from paired samples t tests evaluating change from pre- to post-treatment indicated a significant decrease in depressive symptoms ($t(9) = 6.8$, $p = .000$, $d = 2.8$) and functional impairment ($t(9) = 3.9$, $p = .004$, $d = 2.2$) over the course of study treatment, with large effect sizes. Along with improvements in clinical outcomes, there was a concurrent increase in overall behavioral activation level, as assessed with the BADS ($t(9) = -.24$, $p = .042$, $d = -1.2$). Evaluation of BADS subscales revealed significant improvements in the areas of: activation ($t(9) = -2.37$, $p = .04$, $d = -1.2$), avoidance/rumination ($t(9) = 2.58$, $p = .03$, $d = 1.1$), and social functioning ($t(9) = 3.08$, $p = .01$, $d = 1.3$). There was a trend toward improvement in work/school functioning, ($t(8) = 1.92$, $p = .09$, $d = 1.0$), although this change did not reach statistical significance.

Following study completion, 6 of the 10 participants (60%) met criteria for treatment response, as defined by at least a 50% reduction in symptoms from pre- to post-treatment. Using an IDS-C cut-off of < 12, corresponding to a depression severity of "none," the same 60% of participants also met criteria for depression recovery. In an effort to evaluate the clinical significance of the study findings, we calculated a reliable change index (RCI; Jacobson & Truax, 1991) score for the IDS-C. Using a calculated RCI = 11.3, overall change in depression severity (23.9 points) across participants was determined to be clinically significant. Moreover, although 60% met criteria for depression response/recovery, 9 out of the 10 (90%) participants demonstrated clinically significant improvement in depression symptoms from pre- to post-treatment, as determined by the RCI.

Overall, patients reported high levels of satisfaction with the treatment, as assessed by the CSQ-8 ($M = 30.6$, $SD = 3.3$). When asked what they found to be helpful about the study treatment, participants wrote in: "homework assignments, because they helped me set goals for the week," "the weekly [activity chart] helped me recognize patterns and address them," "learning about avoidance," "the structured behavioral approach," "[learning] about how ruminative thoughts are an avoidance technique," and "taking little steps towards changing behaviors which led to depression." When asked what we could do to improve the treatment, 5 participants wrote, "nothing" or left this section blank. Others suggested that we make the activity chart "more user friendly," and that the "activity chart was a bit superfluous because I was already in tune to what influences my mood." Another participant expressed difficulty with "the idea of doing the opposite of what was making me unhappy." Finally, one participant wrote in, "I wish it were longer."

Discussion

Results from this pilot study provide encouraging, preliminary data that support feasibility and acceptability of BA as a treatment for atypical depression. In general, participants

attended therapy sessions regularly, were accepting of the treatment rationale, and reported high levels of satisfaction with the treatment. Consistent with the theoretical rationale for the treatment, participants specifically identified several direct targets of BA (e.g., functional analysis, avoidance modification, and graded exposure) as elements of the treatment that they found to be particularly helpful.

Also encouraging were preliminary efficacy data for BA in the treatment of AD. On average, there was a significant reduction in depressive symptoms from pre- to post-treatment, which is particularly notable given the rather high severity of depression and level of comorbidity in this community-referred sample. This clinical presentation is consistent with other published reports of the phenomenology of AD (Quitkin, 2002; Stewart et al., 2010), and suggests that clinicians should be prepared to address complex comorbidity, heightened depression severity, and associated clinical features (e.g., social avoidance; Alpert et al., 1997) when treating individuals with AD. Yet despite this complex clinical picture, 60% of participants met criteria for depression response/recovery following treatment, and 90% evidenced clinically significant improvement following BA for AD. Corresponding to these changes in depression, there was a significant improvement in overall functioning. Although preliminary in nature, these outcomes are quite similar to those reported in trials of CT for AD (Jarrett et al., 1999; Mercier et al., 1992) and are comparable to outcomes reported for BA in general outpatient samples (Mazzucchelli et al., 2009). For example, in the largest trial of BA for MDD to date, Dimidjian et al. (2006) reported a 60% response rate and a 56% remission rate in a subset of participants identified as “high severity” at study entry, which parallels outcomes reported from our study sample.

Although this study was not designed to examine potential mechanisms of change, it should also be noted that there was significant change in overall behavioral activation level that corresponded to changes in depression and functioning from pre- to post-treatment. Consequently, there is some preliminary evidence that the study treatment was associated with change in the constructs that are purported to be the “active” ingredients of BA (Kanter et al., 2010). Despite the existence of BA interventions for depression since the 1970s, albeit in slightly different iterations (Kanter et al., 2010), only recently have investigators attempted to develop more targeted measures that more closely assess the theoretical mechanisms of action of BA (Manos, Kanter, & Busch, 2010). Current study data, although preliminary, add to a growing literature supporting the use of the BADS as an assessment tool in studies of BA for depression. However, it is recommended that future research employ repeated measures designs, allowing for the evaluation of temporal ordering of effects to more directly assess change in “behavioral activation” as a mediator of depression outcome.

Indeed, the current study was designed primarily as an initial pilot to assess feasibility and acceptability of BA for AD, and future research will need to address several additional limitations of this research. Primarily, studies employing larger samples, a randomized controlled design, and post-treatment follow-up data will be imperative to properly evaluate the efficacy of BA for AD. Further, the current study sample was largely Caucasian and female. As such, it is unclear to what extent study results generalize to more racially and ethnically diverse patient populations and to men with AD. Although it should also be noted that, in contrast to the roughly 2:1 ratio of women to men represented in cases of non-AD, this ratio is more likely to approximate 3:1 in AD (Posternak & Zimmerman, 2002). Our research was further limited by use of only one therapist, and future research should incorporate formal evaluations of therapist competence and adherence to the manual, to ensure fidelity of the intervention. Finally, given the pilot nature of this study, it is important to interpret treatment outcomes with caution as multiple comparisons may have increased family-wise error and effect sizes derived from small samples may be unstable. As

recommended in the literature (Kraemer & Kupfer, 2006), indices of clinical significance were provided to complement more traditional inferential statistical methods, so as to partly address this limitation associated with pilot studies, with the aim of guiding and advancing future research on behavioral treatments for AD.

As part of the process of extending BA to the treatment of AD, future research will also incorporate refinements to the treatment following from therapist experience and participant feedback. Future treatment development goals include expanding the treatment to more directly address obstacles to behavioral activation frequently reported by our patients with AD, such as low motivation and rumination. In particular, we plan to incorporate a more direct focus on patient values into the treatment, in an effort to promote behavior change that is consistent not only with specified goals, but also with what is inherently important to the patient (e.g., family, health, occupation). Such an approach is consistent with recent conceptualizations of BA (Wilson & Murrell, 2004) as well as existing motivational enhancement interventions (Wagner & Sanchez, 2002). Although there was no direct assessment of rumination in the current study, the study therapist noted that rumination was quite frequently identified as an "avoidance behavior" by study participants, and often times difficult to overcome. Consistent with the existing BA model (Martell et al., 2001), work in this area will focus on developing tools to more directly address the process (vs. content) of ruminative thinking in a behavioral context, such as potentially expanding the functional analysis procedures and/or integrating techniques consistent with the acceptance and mindfulness approaches (e.g., Martell et al., 2001; Wilson & Murrell, 2004). Finally, given the high rates of social phobia in AD (Posternak & Zimmerman, 2002), we will continue to develop the treatment in an effort to address these frequent comorbid conditions, with a focus on how to more directly integrate behavioral activation for AD with exposure for social anxiety.

In conclusion, there is mounting evidence that AD is frequently encountered in clinical settings (Posternak & Zimmerman, 2002), may represent a more severe and chronic form of MDD (Agosti & Stewart, 2001; Angst et al., 2002), and is associated with poorer response to pharmacologic treatment (Davidson, 2007). Although there is some evidence supporting the efficacy of CT for AD (Jarrett et al., 1999; Mercier et al., 1992), the evaluation of psychosocial interventions for AD has been largely ignored in the clinical research literature. Current study data provide encouraging preliminary support for the notion that BA may be a feasible, acceptable, and efficacious treatment for AD, and add to a small but growing literature focused on psychosocial treatments for this depression subtype. Future research is necessary to replicate study findings, and more carefully evaluate BA for AD using more rigorous, randomized controlled designs.

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Table 1

Demographic Characteristics of Study Participants (N = 10)

	M	SD	n	%
Age	36.1	12.7		
Sex (female)			9	90
Race (Caucasian)			10	100
Ethnicity (Non-Hispanic)			9	90
Marital Status				
Married/Cohabiting			5	50
Divorced			1	10
Single, Never Married			4	40
Household Income				
< $20,000			5	50
$20,000 – $39,999			2	20
$40,000 – $59,999			2	20
$60,000 +			1	10
Employment Status				
Employed, Full Time			2	20
Employed, Part Time			2	20
Student, Full Time			3	30
Unemployed			2	20
Disability			1	10

Table 2

Means, Standard Deviations, and Differences from Pre- to Post-Treatment for Study Outcomes

	Pre-Treatment		Post-Treatment				
Measure	***M***	***SD***	***M***	***SD***	***t*(9)**	***p***	***d***
IDS-C	39.0	6.8	15.1	10.1	6.8	.00	2.8
BADS	105.0	22.2	131.6	21.0	−2.4	.04	−1.2
Activation	23.6	5.3	31.6	8.2	−2.3	.04	−1.2
Avoidance/Rumination	29.8	10.0	19.9	7.3	2.6	.03	1.1
Work/School Impairment	21.1	6.8	14.2	6.9	1.9	.09	1.0
Social Impairment	15.2	4.8	9.7	3.3	3.1	.01	1.3
SDS	22.0	3.8	9.9	6.9	3.9	.00	2.2
CSQ-8			30.6	3.3			

Note. IDS-C = Inventory of Depressive Symptomatology – Clinician Rated version; BADS = Behavioral Activation for Depression Scale; SDS = Sheehan Disability Scale; CSQ-8 = Client Satisfaction Questionnaire-8.