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Testing the Effectiveness of Behavioral Activation Therapy in the Treatment of Acute Unipolar Depression

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TESTING THE EFFECTIVENESS OF BEHAVIORAL ACTIVATION THERAPY IN THE TREATMENT OF ACUTE UNIPOLAR DEPRESSION

by

Jenifer M. Cullen

A Dissertation
Submitted to the
Faculty of The Graduate College
in partial fulfillment of the requirements for the
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The present study sought to investigate the clinical effectiveness of Behavioral Activation (BA) Therapy, the behavioral activation component of Beck's Cognitive Therapy (CT; Beck, Rush, Shaw, & Emery, 1979). Seventeen adults seeking mental health services for Unipolar Depression were recruited from the Kalamazoo and Southwestern Michigan regions. All participants were randomly assigned to either (a) an Immediate Treatment Group, or (b) a waitlist control group, while both received 10 weeks of BA therapy. Depressive symptomatology for both conditions were assessed at pretreatment, post-treatment, and 3-month follow-up with the Beck Depression Inventory-II (BDI-II; Beck, Steer, Ball, & Ranieri, 1996), the Structured Clinical Interview for the DSM-IV-Non-Patient Version (SCID; First, Spitzer, Gibbon, & Williams, 1997), and the Revised Hamilton Rating Scale for Depression (RHRSD; Warren, 1996). It was hypothesized that at the completion of treatment, participants in both the immediate treatment and waitlist conditions would be significantly less depressed both on a self-report measure and on clinician ratings of severity of depression. It was further hypothesized that the waitlist participants would show no significant change during the waitlist period.
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CHAPTER I

INTRODUCTION

Unipolar Depression is a serious and debilitating mental disorder that afflicts a large number of human beings worldwide. According to a National Comorbidity Survey in the USA (Blazer, Kessler, McGonagle, & Swartz, 1994), approximately 4.9% of the population suffers from Unipolar Depression. This means that at any given moment approximately 1 person in 20 is significantly depressed. Not only is depression fairly widespread at any given time, but its lifetime prevalence is also high. The National Comorbidity Survey indicates a lifetime prevalence rate of 17% and a 12-month prevalence rate of 10.3% (Blazer et al., 1994; Hammen, 1997). As noted by Lecrubier (2001), these numbers are expected to increase over the coming decades. It has been estimated by the World Health Organization (WHO) Global Burden of Disease Survey that by the year 2020, major depression will be second only to heart disease. Although depression is highly prevalent, it is difficult to know exactly how many individuals suffer from depression at any one time. It seems these numbers may be underestimated since only about 70% of individuals with depression seek treatment (Angst, 1998).

While depression directly affects many people in the USA, even those in society who are not direct victims are impacted. For instance, occupational productivity is directly impacted by depression. According to Mintz, Mintz, Arruda,
& Hwang (1992), the workplace cost of depression in the USA in terms of lost time at
work is estimated at more than 172 million days yearly. When such loss of
productivity is combined with an increase in health care services associated with the
disorder, the costs of depression become astronomical. According to the National
Institute of Mental Health, economic costs reach approximately $27 billion annually.
Today, depression continues to be a challenging mental health problem that is worthy
of much attention (Robinson, Wischman, & Del Vento, 1996).

When Emil Kraeplin first described depression in the early 1800’s, it was
characterized as a disease. Today we know that depression is not something
somebody has, but is more a feeling that one experiences for some period of time.
Some of the common mental states and behaviors that accompany depression include,
but are not limited to: feeling guilty, burdened, and/or dysphoric, problems interacting
with others, low levels of activity, and various physical problems. In order to be
formally diagnosed with Unipolar Depression, as operationalized by the *Diagnostic
and Statistical Manual of Mental Disorders* (4th Edition; DSM-IV; American
Psychiatric Association, 1994), an individual must experience a 2-week period of
dysphoric mood or loss of interest or pleasure, and at least four other symptoms that
may include: (a) significant weight loss or gain; (b) appetite disturbance; (c) insomnia
or hypersomnia; (d) psychomotor agitation or retardation; (e) fatigue or loss of
energy; (f) feelings of worthlessness; (g) inappropriate guilt; (h) impaired
concentration; and (i) recurrent suicidal ideas or a suicide attempt.
These core symptoms of a depressive episode are the same for children, adults, and the elderly, although every individual who suffers from depression does so in their own unique way. Furthermore, there are large individual differences as to which feelings or behaviors accompany the disorder, and the extent to which an individual experiences these symptoms varies widely from person to person (Lewinsohn, Munoz, Youngren, & Zeiss, 1978).

Etiological Components of Unipolar Depression

There have been a number of principles and conceptualizations introduced over the past century to explain the etiology of depression. One theory that precisely explains the etiology of depression, and is backed by numerous years of empirical research, is the behavioral conceptualization. Ferster (1966) has put forth one such theoretical explanation worthy of discussion. He described several hypothetical mechanisms by which depression can occur, the first being the assumption that the major feature of depression is a reduced frequency of adaptive behavior. The environmental events potentially responsible for this reduction are instances when excessively large amounts of adaptive behavior are required before reinforcement is provided or when there is an absence of reinforcement, aversive stimuli or punishment, and finally, a sudden change in the environment (e.g., death of a loved one). Ferster adds that the common denominator of all the above mechanisms is a decrease in the rate of positive reinforcement for adaptive behavior. According to Lewinsohn, Sullivan, & Grosscup (1980), there are three general reasons why a

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person may experience low rates of positive reinforcement. First, the person’s immediate environment may have few available positive reinforcers. Second, the person may lack the skills to obtain available positive reinforcers. Finally, the positive reinforcement potency of events may be reduced.

These changes in the frequency or sources of positive reinforcement can be viewed as the environmental antecedents of depressive symptoms and behaviors. Once these depressive behaviors manifest themselves, they may be maintained by reinforcement from others (the secondary gain phenomenon), and in turn strengthened by their consequences (Lewinsohn, Weinstein, & Alper, 1970). For instance, the individual who mopes around, complains about somatic symptoms, and agonizes over his or her unhappiness generates reactions from the environment that may take the form of sympathy, concern, interest, or suggestions. This positive attention can then inadvertently reinforce depressive behavior.

Ultimately, as the depressed individual becomes increasingly dysphoric and depressed, when the opportunity arises to come in contact with positive reinforcement, inappropriate behavior may surface (e.g., complaining incessantly about their mood). Close friends and family may then begin to see these individuals as aversive, thus ignoring future contacts and further decreasing the frequency of rewards available in the environment. This in turn aggravates the depressed person’s own self-rejection and self-criticism, and leads to further isolation. This vicious cycle may continue until one is so depressed that he or she is resistant to attempts made by others to help him by showing love and friendship (Bandura, 1977).
Contrary to the behavioral etiological conceptualization for depression is the cognitive etiological theory. Whereas the behavioral view states that a depressed person’s behavior is what causes the following despondent emotion or thought, the cognitive conceptualization would argue that depressive thoughts or emotions precede corresponding depressive behavior, and if one can alter negative thoughts or emotions more positive behavior patterns will follow. Beck’s cognitive theory of depression (Beck, Rush, Shaw, & Emery, 1979) has been the most thoroughly researched approach in the cognitive arena (Taylor & Marshall, 1977). Beck’s theory of depression focuses on the strong relationship between thinking and depression. In Beck’s view, a depressed individual consistently thinks he or she is deficient and inadequate, and life experiences are consistently construed in a negative way. It is these erroneous beliefs and maladaptive information processing that eventually play a role in the onset and maintenance of depressive episodes (Kovacs & Beck, 1978).

More specifically, Beck et al. (1979) proposed that there are three concepts that define the etiological components of depression. These components include: (1) the cognitive triad, (2) schemas, and (3) cognitive errors (faulty information processing). The concept behind the cognitive triad explains that depressed people typically follow three negative cognitive patterns. First, depressed people regard themselves in a negative manner. More specifically, they feel defective, inadequate, diseased, or worthless. Second, depressed people interpret their ongoing life experiences in a negative way. They believe they cannot attain their goals because the world makes exorbitant demands on them. Furthermore, interactions with the
environment are misinterpreted and the person is left feeling defeated or deprived. Finally, depressed people see their future in a negative light. Further difficulties are anticipated, while hardship, frustration, and deprivations are expected to plague them.

The second major element in Beck's cognitive model of depression incorporates the concept of schemas. According to Beck et al. (1979), schemas are stable cognitive patterns that are used to screen out, differentiate, and code the stimuli that confront us daily. Experiences thus become categorized and evaluated through a matrix of schemas. People suffering from depression are said to possess a dormant cognitive schema. This schema may then become active under conditions of stress that are related to experiences initially responsible for embedding the negative attitude. Once activated, this schema influences the way information is processed and people lose voluntary control over thinking processes. Other more appropriate schemas are then unable to be evoked (Kovacs & Beck, 1978; Beck et al., 1979).

The final major element that Beck et al. (1979) recognizes as critical in a discussion of cognitive factors of depression is faulty information processing. The depressed person perceives their present, future, and outside world (the cognitive triad) in a negative light. It is these systematic errors in thinking that maintains their beliefs they are worthless, inadequate, etc. More specifically, depressed people have a tendency to make broad global statements regarding the events in their lives, and the meanings they give to these events are "extreme, negative, categorical, absolute, and judgmental" (Beck et al., 1979, p. 14). The depressed person consequently leads a biased rendition of their life experiences and expects to fail at anything they
undertake, all the while engaging in a tremendous amount of self-criticism (Beck et al., 1979).

Behavior Therapy Treatment Modalities

Because both the behavioral and cognitive conceptualizations of depression have been supported with many years of clinical and empirical evidence (Beck et al., 1979; Steinbrueck, Maxwell, & Howard, 1983; Dobson, 1989; McLean, Ogston, & Grauer, 1973), successful treatment modalities for each have followed. While there are noted similarities between these two modalities of treatment (Beck, 1970), each helps the depressed individual in a unique way. The behavioral treatment of depression is based primarily on learning theory and its therapeutic goal is to change the contingencies that initiate and maintain depressive behaviors. According to Kovacs (1979), the traditional behavioral therapy for depression essentially seeks to increase the frequency of socially desirable behaviors, while at the same time decreasing the rate of undesirable ones. Teaching the client the necessary skills that will enhance the ability to come in contact with positive social reinforcers can do this. Many successful behavioral interventions have therefore been adopted that have allowed the behavioral therapist to institute those that are best suited for the individual client.

Therapeutic techniques that help an individual restore an adequate schedule of reinforcement by increasing activity levels are both common and instrumental in decreasing depressive symptoms (Lewinsohn et al., 1980). "Behavioral activation"
employs the well-known idea that being active leads to rewards that are antidotes to depression (Hammen, 1997, p. 142). As noted by Beck et al. (1979), people who are depressed typically participate in a great deal of social withdrawal and avoidance on the basis that activity and social interaction are meaningless, not interesting, or that others see them as a burden. They criticize themselves for their withdrawal and lack of motivation, which thereby adds to feelings of inadequacy and helplessness. As a result, remaining inactive becomes part of a vicious cycle that is difficult to break out of.

One such intervention that decreases a client’s passivity and inactivity involves the therapist and patient collaborating to construct a daily activity schedule. Essentially, specific hour-by-hour activities are planned throughout the day and the client monitors and records these completed activities on a record form. Constructing specific goal-oriented tasks not only shows the client that he or she is capable of setting and accomplishing goals, but also provides the therapist and client with concrete data from which to base assessments of the patient’s functional capacity (Beck et al., 1979).

To supplement the activity schedule, a “graded task” hierarchy may be incorporated into the daily plan (Beck et al., 1979, p. 121). Graded task assignments are stepwise tasks or activities that help clients progress to completing more difficult assignments as the simpler ones are mastered (Hammen, 1997). After the successful completion of a task, the patient usually feels motivated to move onto the next step.
Repeated successes generally begin to undermine the feelings of inadequacy and worthlessness that fuel one's inactivity (Beck et al., 1979).

While it is important that a depressed individual remain behaviorally activated, it is also important for a client to derive some pleasure from these activities and tasks. According to Beck et al. (1979), some depressed patients engage in activities, but procure very little pleasure from them. One reason for this may be their "selective inattention to sensations of pleasure" (Beck et al., 1979, p. 128). Mastery and Pleasure techniques have thus been designed to drive a patient to undertake a particular pleasurable activity for a specified number of minutes each day. Changes in mood and reductions in depressive symptoms associated with the pleasurable activity are recorded by the patient. More specifically, it is helpful if the patient rates their degree of Pleasure (P) and Mastery (M), which refers to their sense of accomplishment when performing a particular task or activity. As a result, these ratings rivet one's attention to the enjoyment they are deriving from the participation in and completion of activities (Beck et al., 1979).

Interventions that have been aimed at improving depressed patients' social and assertive skills are also common in the behavioral therapeutic process. As mentioned previously, depressed individuals typically have a tendency to slip into social isolation, becoming increasingly passive as their opportunities to interact socially become fewer and fewer (Beck et al., 1979). Social skills training seeks to increase the frequency of adaptive behaviors associated with positive social reinforcement. Furthermore, so that depressed individuals can extract more positive...
reinforcers from their lives, assertiveness skills (e.g., role-playing) may also be taught (Lewinsohn, Antonuccio, Steinmetz, & Teri, 1984).

Cognitive Therapy Treatment Modalities

Along with BT, Cognitive therapy (CT) has also established itself as an effective short-term therapy for clinical depression (Dobson, 1989). The cognitive therapy of depression, developed by Beck and his associates (Beck et al., 1979), is a specific type of the broad class of therapies called cognitive behavioral (Dobson & Block, 1988). Contrary to the emphasis in behavioral treatment for depression, CT is based on the premise that introspective data (i.e., thoughts, feelings, wishes, daydreams, attitudes) provide a wealth of information that the therapist can use as the principal target for therapeutic work (Beck, 1970). Unlike behavior therapy which focuses on the overt behaviors of its clients, CT focuses on a set of operations that center around “a client’s cognitions (verbal or pictorial) and on the premises, assumptions, and attitudes underlying these cognitions” (Beck, 1970, p. 187). Because a cognitive psychologist believes that dysfunctional thought patterns are what fuels one’s depressive symptoms, CT focuses to change the client’s misinterpretations, self-defeating behavior, and dysfunctional attitudes. The client is taught to identify his or her faulty cognitions and recognize the crucial link between negative antecedent thoughts and the subsequent negative feelings that follow (Kovacs & Beck, 1978).
According to Beck et al. (1979), because this relationship between negative cognitions and unpleasant emotions is so strong, CT relies heavily on "emotional techniques" as part of its therapeutic repertoire (p. 36). The following is a list of operations that may be included in CT. While not exhaustive, this slate of interventions is designed to delineate and test the client's specific misconceptions and maladaptive assumptions.

As Beck et al. (1979) notes, the first and most critical intervention involves the self-monitoring of "automatic thoughts." Automatic thoughts come out of the blue, unprompted by events and are not necessarily the result of 'directed' thinking. These thoughts are typically immediate and are valid in the sense that the individual accepts them as true without further analyzing them. Automatic thoughts spawn further thoughts and images to emerge and cause a "downward spiral of despair" (Clark & Fairburn, 1997, p. 263). To target these automatic thoughts, the therapist trains the client to observe, define, and record the negative cognitions. Through in-between session assignments, the relationship between cognition and affect are demonstrated, using specific, real-life examples. Once a client learns to detect as many negative automatic thoughts as possible, the client and therapist can work together to examine the evidence for and against these distorted automatic thoughts. With the help of the therapist, more pragmatic and reality-oriented interpretations for these biased cognitions can then be substituted. While the depressed person characteristically views the world in a negative light, the goal here is to encourage a more accurate description of the way things truly are. Finally, as the client learns to
identify, challenge, and alter these dysfunctional beliefs, a decrease in depressive symptoms may follow (Beck et al., 1979).

While there are obvious differences between the behavioral and cognitive treatments for depression, these two systems of psychotherapy share many similarities and are often blurred. First, in both the behavioral and cognitive paradigms, clients are trained to initiate, conduct and evaluate their own treatment, with the guidance of their therapist. In essence, both are "action" therapies in which clients do something about their difficulties, rather than just talk about them. Furthermore, clients engage in specific tasks to alleviate their depressive symptoms. These therapeutic tasks (i.e., homework assignments) are an integral part of both BT and CT and are commonly used to designate therapy procedures in the client's natural environment (Spiegler & Guevremont, 1998). In addition, the goals set for both therapies are specific, as opposed to open-ended, and they guide the treatment process that will be implemented for that particular therapy (Beck, 1970).

Another similarity is the fact that both cognitive and behavior therapists focus at least some of their techniques at public, overt symptoms or behavior problems. While the targets of therapeutic change differ for both behavior and cognitive therapists, both systems conceptualize symptom formation in terms of constructs that are accessible to either behavioral observation or to introspection (Beck, 1970).

Moreover, in contrast to psychoanalytic therapy, neither behavior nor cognitive therapies focus their sessions on recollections or reconstructions of the client's childhood experiences and early family relationships. Causal relationships
between childhood events and current psychological functioning are rarely made, while the focus of treatment is on the “here and now,” rather than on historical determinants of behavior. Techniques are employed to change the relevant current factors that are influencing the depressive symptoms (Beck, 1970; Franks & Barbrack, 1983; Spiegler & Guevremont, 1998).

A final commonality between behavior and cognitive therapies is that both paradigms exclude most traditional psychoanalytic assumptions (e.g., infantile sexuality, the unconscious, defense mechanisms, fixations) from clouding the therapeutic process. More specifically, both systems essentially restrict the high-level abstractions that are characteristic of psychoanalytic therapy (Beck, 1970).

The Advent of Cognitive-Behavioral Therapy

While the theoretical frameworks of behavioral and cognitive therapies are noticeably congruent on many levels, researchers have merged the two to form “cognitive-behavioral” therapy (CBT) for depression (e.g., Beck’s Cognitive Therapy). Essentially, CBT represents an extension of traditional behavior therapy and encompasses the modification of cognitive events as actual behaviors (Kovacs, 1979). While behavioral and cognitive approaches differ in their emphasis on the etiological role of overt and covert behaviors, when the two systems are united to form the cognitive-behavioral approach, it is indicated that both covert and overt behaviors play important roles in maintaining and modifying depression (Taylor & Marshall, 1977).
According to Dobson & Block (1988), the actual outcomes of CBT will vary from client to client, but in general, the two main indices targeted for change are both cognition and behavior. At its core, CBT shares three key positions: (1) cognitive activity affects behavior, (2) cognitive activity may be monitored and altered, and (3) desired behavior change may be affected through cognitive change (Dobson & Block, 1988). Both cognitive and behavioral techniques are exercised in CBT, as the therapeutic goal is twofold. Therapist and client set out to alter faulty information processing systems, while simultaneously modifying the environmental contingencies that maintain depressive symptoms.

While CBT places emphasis on the cognitive mechanisms that create behavioral effects, Dobson & Block (1988) add that elaborate cognitive mechanisms are not ultimately required for behavioral change to occur. In fact, some CBT interventions may have little to do with cognitive appraisals and evaluations, but may instead be heavily dependent and focused upon client actions and behavior.
CHAPTER II

REVIEW OF THE LITERATURE

Evidence Supporting Behavior Therapy

While CBT has been widely established as an effective treatment modality, its components in isolation have also proven to be clinically effective. Numerous studies have investigated depression treatments with an emphasis on behavioral principles and interventions and have produced findings that support the use of these techniques. For instance, in a study conducted by Wilson, Goldin, & Charbonneau-Powis (1983), a behavioral treatment for depression was found to be clearly superior to no treatment. Twenty-five depressed, non-psychotic participants were randomly assigned to either a behavior therapy or a waitlist condition. Behavior therapy was implemented for eight weeks and included interventions such as activity schedules, graded task assignments, daily self-monitoring of mood, and social reinforcement for attempted and completed activities and tasks. Measures of depressive-related symptomatology and treatment-related target areas were administered prior to treatment, at mid-treatment, at the termination of treatment, and at a 5-month follow-up. In comparison to the waitlist condition, participants in the BT condition significantly improved on self-report and clinician rated measures such as the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), with
$F(1, 22) = 55.04, p < 0.01$, and the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960), with $F(1, 22) = 20.04, p < 0.01$. Additionally, treatment effects were maintained at the 5-month follow-up [$F(1, 14) = 56.0, p < 0.01$]. The results of this study provided support for the use of short-term behavioral therapy for depression (Wilson et al., 1983).

Another study that provided support for BT as a viable treatment for depression was conducted by McLean, et al. (1980). The authors compared social-skills behavior therapy for couples to a “doctor’s choice” treatment for 20 depressed outpatients. According to their findings, the group that received social-skills behavior therapy demonstrated significant improvement in original target problems as compared to a “doctor’s choice” of treatment group. The “doctor’s choice” treatment varied as a function of the treating agency and involved diverse psychological interventions (e.g., office consultations with a social worker) or pharmacological interventions (e.g., antidepressant medication). Ten patients received an 8-week behavioral treatment and 10 were returned to their referral sources (“doctor’s choice”). Pretreatment assessment consisted of self-ratings of mood and the construction of a list of five problematic target behaviors (e.g., decrease use of negative verbal interactions). Results showed that at the end of treatment, the behavioral treatment had a significant positive impact on the target behaviors ($p < 0.001$), whereas those in the comparison group did not ($p < 0.23$). Also, compared to pretreatment levels of depression, the experimental group showed a significant decrease in depression levels ($t = 4.07, p < 0.001$), as measured by the Depression scales.
Adjective Checklist (DACL; Lubin, 1965). The significant difference in depression levels were also maintained at the time of follow-up ($t = 3.42, p < 0.01$). This study yielded further support for the effectiveness of behavioral techniques in the management of depression.

Evidence Supporting Cognitive Therapy

Cognitive therapy has also proven itself efficacious in the literature as a potent treatment for depression. For instance, Shaw (1977) demonstrated that Beck's CT was clearly superior to a no-treatment control group in decreasing depressive symptomatology. The sample consisted of 32 mildly to moderately depressed persons who were treated at a university student health service. A group treatment format was utilized over four weekly 2-hour sessions. Results indicated that the cognitive modification group, as compared to the waitlist control group, demonstrated a significantly greater decrease in self-report depressive symptomatology on the BDI ($t = 4.47, p < 0.01$) and on clinical ratings of the HRSD ($t = 2.79, p < 0.01$). These results provided favorable and supporting results for a cognitive treatment program for depression.

In a study conducted by Rush, Beck, Kovacs, and Hollon (1977) CT was again established as a promising treatment for depression, as 79.8% of patients showed marked improvement or complete remission of symptoms. Nineteen moderate to severely depressed outpatients received a maximum of 20 sessions of CT, while 22 participants (also experiencing moderate to severe depression) received

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weekly 20-minute medication reviews and nonspecific supportive therapy. Both psychological and pharmacological treatments (i.e., Tricyclics) were administered for 12 weeks and assessments included a variety of self-rating scales including the Beck Depression Inventory and independent clinical ratings such as the HRSD and the Raskin Depression Scale (Raskin, Schulterbrandt, Reating, & McKeon, 1970). Results showed that not only was CT associated with a significant decrease in depressive symptomatology on the BDI \( t (17) = 11.76, p < 0.001 \), but it resulted in greater clinical improvement on the HRSD \( t (14) = 7.78, p < 0.001 \) and the Raskin Depression Scale \( t (13) = 9.50, p < 0.001 \). Additionally, a one-way analysis of covariance for treatment effects unveiled cognitive therapy as significantly more effective than pharmacotherapy in reducing depressive symptomatology, with \( F (1,29) = 4.43, p < 0.05 \) (Kovacs, 1979; Rush et al., 1977).

Finally, in a meta-analysis conducted by Dobson (1989), an exhaustive review of 28 studies was completed to analyze the effect of Beck's CT on depressed clients. The clinical efficacy of CT was compared against a waitlist or no-treatment control, pharmacotherapy, behavior therapy, and other various psychotherapies (e.g., psychoanalysis, interpersonal therapy). The results of this meta-analysis showed that CT was more effective than nothing at all, behavior therapy, and pharmacotherapy. More specifically, the average CT client did better than 98% of control subjects, 67% better than behavior therapy clients, and 70% better than drug therapy or other psychotherapy clients.
Evidence for Combined Treatment-Medication Plus Cognitive Therapy

As noted by Hollon, et al. (1992), numerous questions remain regarding the efficacy of CT relative to pharmacotherapy in the treatment of depression. Consequently, numerous studies have been conducted that examined the combined effects of CT and pharmacotherapy in comparison to each modality in isolation (for a comprehensive review, see Hollon, Shelton, & Loosen, 1991). The literature seems to be divided in that some studies favor the combined modality of cognitive pharmacology over either treatment alone (Bowers, 1990), while others have found no significant differences between combined treatment and CT or pharmacotherapy alone (Murphy, Simons, Wetzel, & Lustman, 1984; Hollon, Shelton, & Davis, 1993; Oei & Yeoh, 1999).

A recent study reported a significant benefit for combined treatment in a multicenter study of patients suffering from chronic, nonpsychotic major depression (Keller, et al., 2000). The sample consisted of 681 adult outpatients who were randomly assigned to (a) cognitive-behavioral therapy, (b) nefazodone, or (c) a combination of the two. All treatments were administered for 12–16 weeks and the primary outcome measure was the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960). Remission was defined a priori as an HRSD score of no more than 8 at weeks 10 and 12. Although participants in all three groups significantly improved over the 12 weeks ($p < 0.001$), an analysis of endpoint HRSD scores revealed that from week 4 through 12, the average rate of improvement in the combined-treatment group was significantly larger than the rate of improvement in
the nefazodone and psychotherapy groups ($p < 0.001$). More specifically, 85% of participants in the combined-treatment group had a positive response to treatment by week 12, as compared with 55% of participants in the nefazodone group and 52% in the psychotherapy group ($p < 0.001$ for both comparisons). Because the combined treatment group achieved early benefits from medication and then additive benefits from psychotherapy later in the trial, the authors suggested that the combination of two independent effects rather than a synergistic mode of action, increased patients response to treatment. In sum, the authors determined that the degree of superiority of combination therapy provided a clinically meaningful advantage over both CT and medication alone.

One study that attested to the additive effects of combined cognitive pharmacotherapy was conducted by Blackburn, et al. (1981). Sixty-four clinically depressed patients, drawn from a general practice setting and a hospital outpatient clinic, were randomly assigned to 20 weeks of treatment in one of three conditions: (a) cognitive therapy, (b) tricyclic pharmacotherapy, or (c) a combination of the two. Depressive symptomatology was assessed via the BDI, HRSD, and the Snaith’s Irritability, Depression, and Anxiety Scale (IDA; Snaith, Constantopoulos, Jardine, & McGuffin, 1978). For the outcome results, Snaith’s scale displayed few overall changes, but the BDI and HRSD both revealed clear cut effects. Results revealed that in the hospital outpatient sample, the combined treatment of CT and tricyclics was significantly superior to CT or pharmacotherapy alone ($p < 0.01$). In the general practice setting however, the combination treatment and CT alone were equally
effective and considerably better than drugs alone (Blackburn et al., 1981). Not only did this study have implications for establishing appropriate treatments for depression, but it also was the first outcome study to use a population that was not from North America. Previous to this study, the generalizability of the effectiveness of CBT across continents was in question, but using a European sample served to decrease existing suspicions (Williams, 1992).

In contrast, studies have not found any significant advantage for the combined modality relative to either modality alone. Murphy et al. (1984) compared the differential effects of CT, pharmacotherapy, CT plus a placebo, and CT plus pharmacotherapy by randomly assigning eighty-seven moderately to severely depressed psychiatric outpatients to one of four conditions. Each participant completed 12 weeks of treatment and was assessed with a self-report measure (i.e., the BDI) and an independent clinical interview (i.e., the HRSD). Results prompted the authors to conclude that over time, while all three treatment groups led to significant decreases in depressive symptomatology \(F(2, 65) = 236.50, p < 0.001\), improvement did not differ as a function of the different treatment modalities \(F(6, 130) = 0.32, p = 0.92\). In essence, combining treatments did not lead either to additive effects or negative interactions (Murphy et al., 1984).

In a more recent randomized clinical trial, Blackburn & Moore (1997) also found that combining CT and medication was not significantly more effective in lowering depression levels than either treatment alone. Seventy-five outpatients with recurrent major depression were allocated to three groups, each including 16 weeks of
acute treatment and two years' maintenance therapy, in the following manner: (a) antidepressants and maintenance antidepressants, (b) CT and maintenance CT, or (c) a combination of antidepressants and maintenance CT. Ratings on outcome measures (i.e., BDI-II and HRSD) were repeated every four weeks during acute treatment and every four months during the 2-year follow-up phase. Results for acute treatment showed that over the 16-week treatment period, all three treatment groups continued to improve over time and there was no significant difference among treatments on both the HRSD and the BDI-II ($p < 0.0001$ for both measures). Results of longer term outcomes indicated that patients in all groups continued to improve significantly over time [$F(6, 329) = 4.51, p < 0.001$] and there were no significant differences between treatments [$F(2, 55) = 0.31, p = ns$] at any point in time. Thus, this result supported the idea that combining acute medication and maintenance CT treatments was not more effective in lowering depression levels than either treatment modality alone. Further analyses did show one trend for a difference appearing at 20 months on the BDI [$F(12, 329) = 1.61, p < 0.08$], reflecting a more steady improvement in the two maintenance CT groups. This allowed Blackburn and Moore (1997) to conclude that "maintenance cognitive therapy has a similar prophylactic effect to maintenance medication and is a viable option for maintenance after acute treatment with medication in recurrent depression" (p. 328).
Combined Cognitive and Behavior Therapy

Not only have the behavioral and cognitive therapies been tested for clinical efficacy, but a variety of studies have also supported cognitive-behavioral treatments for depression. One such study (Taylor and Marshall, 1977) noted that, because both covert and overt events play important roles in maintaining and modifying depression, the additive effects of behavior and cognitive therapies (i.e., CBT) may be therapeutically more effective in decreasing depressive symptomatology than either modality alone. The authors tested this hypothesis by randomly assigning 28 mild to moderately depressed college student volunteers to one of four groups: (1) cognitive treatment, (2) behavior treatment, (3) cognitive and behavioral treatments combined, or (4) waitlist control group. Behavior therapy encompassed the approaches of Ferster, Lazarus, and Lewinsohn, while CT encompassed a combination of modalities offered by Beck, Ellis, Bandura, and Marston (Kovacs, 1979). Each experimental participant received six 40-minute treatment sessions. Depression outcome measures included the BDI, the D-30 scale (Dempsey, 1964), and the Visual Analogue Scale (VAS; Aitken, 1969). Results showed that while there were significant improvements in self-rated symptomatology for all three treatment groups, the combined cognitive-behavioral treatment was more effective in decreasing depressive symptoms \( F(1, 18) = 6.71, p < 0.03 \) than either behavioral or cognitive treatments alone. These effects were maintained at the 5-week follow-up point \( F(1, 18) = 9.64, p < 0.01 \). In their attempt to examine the utility of CBT, Taylor and Marshall (1977)
concluded that for maximum benefit, the integration of BT and CT (i.e., CBT) should be performed when treating depression.

Dismantling of Cognitive Behavior Treatment

While the clinical effectiveness of CBT has been well-documented (Dobson, 1989; Miller & Berman, 1983; Taylor & Marshall, 1977; Williams, 1992), a question remains as to which component of CBT is most responsible for therapeutic change and improvement. Is it the behavioral component, the cognitive component, or the additive effects of the two that make CBT so clinically effective? There is an evident need to dismantle CBT in order to identify the components that are either sufficient or vital ingredients for improving depressive symptomatology.

An ingenious study conducted by Jacobson, Traux, Addis, Koerner, Gollan, Gortner, & Prince (1996) set out to answer these questions by dissecting Beck’s CT for depression (Beck et al., 1979). Jacobson and his colleagues randomly assigned 152 depressed outpatients to one of three treatments based on components of CBT: (1) behavioral activation (BA), which is the behavioral component of CBT; (2) automatic thoughts (AT), which includes behavioral activation along with skills to modify automatic thoughts or; (3) “full” CBT, which consists of behavioral activation, modification of negative thoughts, plus changing core dysfunctional schemas. It was hypothesized that “CT should work significantly better than AT, which in turn, should work significantly better than BA” (p. 296). An additional purpose for this study was to investigate whether the various treatments differentially
affected the process that they were supposed to affect. For instance, was the “full” CBT more successful at modifying dysfunctional schemas than BA? Would the BA condition be more successful at activating people to participate in previously enjoyable activities than the full CBT?

Participants in each condition received treatment for 16 weeks with a maximum of 24 sessions. The depressive symptomatology was measured for all participants with the Longitudinal Interval Follow-up Evaluation-II (LIFE; Keller, et al., 1987), the BDI and the HRSD. Measurements were taken before therapy, at the time of termination, and at 6, 12, and 24-month follow-ups. Unexpectedly, results showed that after 20 sessions, as well as at the 6-month follow-up, there were no significant differences in self-reported depression levels between the three groups. Additionally, there were no differences found between the treatments one or two years after treatment (Gortner, Gollan, Jacobson, & Dobson, 1998).

In opposition to their hypothesis, those who received BA alone fared as well as those who were taught additional coping skills to counter depressive thinking. Essentially, the BA component was as successful in reducing depression and altering negative thinking and attributional styles as was the AT and “full” CBT conditions. Most importantly, this study identified the BA condition as an active ingredient in CBT, capable of producing clinically significant antidepressant effects. In sum, Jacobson and his colleagues found no evidence that CBT was any more effective than either of its components alone (Jacobson et al., 1996). In other words, as noted by
Martell, Addis, & Jacobson (2001), helping to activate these participants was just as effective to treat their depression as helping them to change their thinking.

These results have important implications for the treatment outcome of major depression, as they run contrary to the cognitive model of depression put forth by Beck and his colleagues (Beck et al., 1979), that states that the modification of negative cognitions are necessary to maximize treatment outcome. The Jacobson et al. (1996) study calls into question some of the assumptions of the cognitive-behavioral model by suggesting that altering thinking and dysfunctional schemas may not be necessary to counter depressive symptomatology. Alternatively, it is suggested that the participation in and exposure to a variety of pleasurable activities served not only to change dysfunctional thinking, but to act as an antidote for depression (Hammen, 1997).

In the search for empirically validated treatments that are short-term, cost-effective and simpler to learn and administer, the Jacobson et al. (1996) findings may have great economic and clinical value. Having to implement only the BA portion of CBT makes behavioral activation appear much more parsimonious and “user friendly” (Robinson et al., 1996). As noted by Chambless and Hollon (1998), the behavioral approaches to depression are typically easier to master than the more complex cognitive interventions. In turn, this could make BA more accessible to less experienced or paraprofessional therapists. Furthermore, BA represents a less expensive alternative to CBT in that the intervention choices are fewer and therapists do not have to implement the full CBT treatment plan (Jacobson et al., 1996).
Problem Statement

Previous research suggests that cognitive behavioral treatment of depression stands as the psychotherapeutic intervention of choice in the treatment of clinical depression (Hammen, 1997). Recent work by Jacobson et al. (1996) further suggests that the active ingredient in cognitive behavioral treatment of depression might entail behavioral activation. This ingredient is found in the overall cognitive behavioral treatment protocol as one of three clearly identifiable components. According to the Jacobson investigation behavioral activation may be simpler to administer and yet achieve outcomes that equal those found with the full cognitive behavioral treatment protocol. If this observation can be verified it would serve to render a more efficient treatment for clinical depression. It would also give rise to a conceptual challenge to the prevailing theory of cognitive behavioral intervention; a theory that suggests that direct modification of dysfunctional cognitions is essential for successful treatment outcome.

The present investigation aims to conduct a quasi-replication of the basic findings noted in the Jacobson study. It will utilize a sample that differs, however, from that treated by Jacobson. In the practice setting in which most psychologists operate, clients present for treatment even while still consuming psychotropic medications. Of all the people who are prescribed a psychotropic medication by a psychiatrist, as many as one-quarter to one third (30.4%) are treated with antidepressant medication (Olfson & Klerman, 1993). It is policy in many
community agencies that clients who are diagnosed as clinically depressed and who receive antidepressant medication must also be seen by an additional psychosocial intervention service. These other psychosocial interventions may include psychotherapy, case management, or psychoeducational services (personal communication 1998, Venture Behavioral Health).

Because of this prevailing reality, it is important to test the effectiveness of behavioral activation with clients who realistically present while consuming medication, in addition to testing clients who are unmedicated, as was done in the Jacobson study. Yet this must be done in a way that allows for reasonably valid comparisons between persons who were and were not medicated. This arrangement is made difficult in the present study by the absence of a psychiatrist who would simultaneously administer medication or no medication within the context of the proposed research design. However, the present investigation will attempt to achieve quasi-experimental control that permits reasonable inferences regarding the effects of behavioral activation treatment of both samples. The primary research question is “What are the effects of behavioral activation on a mixed sample of medicated and unmedicated depressed adults?” In answering this question, this study will provide further clarification on the role of behavioral activation protocol in the treatment of a more “real world” sample of depressed clients.

In this investigation, rather than random assignment to one or the other medication condition, all subjects, irrespective of medication condition will be randomly assigned to either a waitlist (delayed treatment) control or an immediate
treatment condition. Those subjects who are medicated, however, will have to have initiated medication at least four weeks prior to entering the study and must remain on the same medication for the duration of the study period. Changes in doses will be allowed. This arrangement is essential in order to provide a fair test of whether any treatment outcomes are attributable to the medication condition alone. If subjects in the delayed treatment condition continue to qualify for admission into the study even though they continue to take medications for a period of at least six weeks, then it is unlikely that observed treatment outcomes can be easily attributable to medications alone. This would suggest that behavioral activation was at least a contributing factor to treatment outcome.

On the other hand, if unmedicated subjects who receive immediate treatment demonstrate positive outcomes at post-treatment, when compared to the unmedicated subjects in the delayed treatment condition, then it must be concluded that behavioral activation was likely responsible for the observed outcomes. However, because of the quasi-experimental nature of this investigation, no firm conclusions regarding causality can be drawn. That level of conclusion will be left to a future true randomized controlled trial.
CHAPTER III

METHOD

Sample

Seventeen adult participants seeking mental health services for Unipolar Depression were recruited from the Kalamazoo and Southwestern Michigan regions through public service announcements, newspaper advertisement, solicitations from community professionals, and other healthcare agencies.

All participants met criteria for Major Depressive Disorder (MDD) according to the Diagnostic and Statistical Manual of Mental Disorders (4th edition; DSM-IV; American Psychiatric Association, 1994). The DSM-IV diagnosis was based on the Structured Clinical Interview for DSM-IV-Non Patient (SCID-NP; First, Spitzer, Gibon, & Williams, 1997). Participants scored at least 20 on the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996), and 14 or greater on the Revised Hamilton Rating Scale for Depression (RHRSD; Warren, 1996). The author and a licensed clinical psychologist who had utilized both outcomes in several other investigations provided training and supervision of the RHRSD and SCID-NP.

Exclusion criteria included a number of coexistent psychiatric disorders including bipolar or psychotic subtypes of depression, panic disorder, current alcohol or other substance abuse, past or present schizophrenia or schizophreniform disorder,
organic brain syndrome, obsessive compulsive disorder and mental retardation. Additionally, the comorbid presence of personality disorders were limited inasmuch as the percentage of patients with a personality disorder in the sample approximated the percentage of patients in the population of Psychology Clinic patients having the same personality disorder.

Furthermore, suicidality was assessed and participants who presented a considerable risk were referred for service outside of the study for further assessment and determination of appropriateness for participation in the study. A licensed clinical psychologist, along with the author, made final decisions regarding the eligibility of the participants. Suicide risk was determined by an individual’s responses to the suicide items on the BDI-II, the RHRSD, and the SCID-NP, as well as by any verbalizations of suicidal ideations made by the patient during the assessment phase of the study. Finally, participants who were in some concurrent form of psychotherapy or who needed to be hospitalized because of imminent suicide potential or psychosis were deemed ineligible for the study and referred for alternative treatment.

Setting

All assessment and treatment sessions were conducted in private therapy rooms at Western Michigan University’s Psychology Clinic, located at 1000 Oakland Drive, Kalamazoo, MI. Each room was equipped with a one-way mirror so that the integrity of the treatment could be monitored with video cameras.
Assessors and Therapists

Second and third year doctoral students in a clinical or counseling psychology graduate program conducted all assessment interviews. Training and supervision of the assessment tools were provided by the author and included SCID training videos and the BDI-II and RHRSD manuals.

Therapists were selected and trained from among third year doctoral students in clinical and counseling psychology graduate programs. All therapists had at least two years practicum experience. Therapists received basic training in the cognitive behavioral treatment of depression and for purposes of the present investigation received an additional 12 hours of training in the use of Behavioral Activation (BA) therapy. All therapy training was conducted by the principal investigator, Richard Spates, Ph.D., who is a licensed clinical psychologist and has vast experience in cognitive behavioral therapy, along with the author, who at the time of training had three years practicum experience and held a temporary license to practice in the state of Michigan.

Treatments

The treatment implemented in this study was the “behavioral activation” component of Beck’s Cognitive Therapy (CT; Beck et al., 1979). Behavioral Activation therapy is based on the behavioral conceptualization that depression is best understood as “a series of actions and events rather than some sort of internal object
or mechanism” (Martell et al., 2001, p. 12) and results from changes in client’s life circumstances (e.g., death of significant other, losing a job). The loss of reinforcement resulting from these life changes is what precipitates the depression. Once an individual is depressed, the negative way in which he or she responds to their environment (e.g., avoidance) often exacerbates their dysphoric mood by depriving them of further reinforcement. Thus the purpose of BA is to activate clients so that they may break a passive approach to life and maximize their opportunity to make contact with natural, positive reinforcers in their environment (Martell et al., 2001).

The emphasis of BA is on “focused activation,” as opposed to simply activity at random. This includes not only finding behaviors and activities that will be positively reinforcing, but paying close attention to the activities with which one is participating (e.g., noticing colors, noises, and smells associated with the activity). This attention to the experience intervention is very similar to the mindfulness training taught by Marsha Linehan in Dialectical Behavior Therapy (Jacobson, Martell, & Dimidjian, 2001; Linehan, 1993).

Behavioral Activation treatment is a therapeutic tool whose goals are to (a) determine the life circumstances that precipitate the depression, (b) determine the coping patterns that maintain and exacerbate the depression (e.g., chronic negativity, social withdrawal), and (c) develop a treatment plan for improving the coping patterns and provide access to more reinforcing life circumstances (Jacobson, et al., 1997). The BA therapist helps the client to achieve these goals by working with them as a so-called “personal trainer” who helps them to learn and implement a set of skills
that are likely to be effective, just as a supportive coach might do. The therapy is delivered in a directive manner, but the client and therapist choose the direction in concert. It is important to note that the therapist coaches the client to learn a core set of BA skills, but because the skills’ form varies from client to client, the BA therapist is required to be flexible, proficient, and able to coach a wide range of unique clients. Without such a supportive, collaborative working relationship, it is unlikely clients will change ingrained patterns of maladaptive behavior (Martell et al., 2001) and meet the BA treatment goals.

What BA is not about is teaching clients simple maneuvers that increase pleasant activities. Many people engage in regular, positive activity and are still depressed. Therefore, pleasant events in and of themselves cannot be assumed as antidepressants (Dobson & Joffe, 1986). As noted by Martell et al. (2001), BA is not about getting people to do nice or fun things. It is about incorporating directed activity into a client’s narrow repertoire of behavior, regardless of how one feels internally, so that they break avoidance patterns and increase the possibility of coming in contact with reinforcers in their environment.

Behavioral Activation is also not a “psychotherapy from the neck down” (Martell et al., 2001, p. 64). Therapists who treat clients with BA recognize that not only are inactivity, withdrawal, and avoidance commonplace depressive behaviors, but according to Nolen-Hoeksema, Morrow, & Fredrickson (1993), a great deal of time is also spent thinking about, or ruminating, the misery of their lives. Thus the context of client thinking is assessed and acknowledged, rather than the actual content.
of the thought. For instance, if a client presents with the ruminative thought, “everyone at school hates me,” the BA therapist would ask the client under what conditions does this sort of thinking occur, what is he doing when thinking that way, and what is he avoiding by spending time ruminating? The client is then encouraged to identify antecedents and consequences of such ruminations. Moreover, the veracity of that thought would not be challenged or evaluated, but rather the impact of the behavior “thinking that people hate me” would be addressed (Martell et al., 2001).

The theory and practice of BA is based primarily upon a manual called *Cognitive & Behavioral Treatment of Depression: A Research Treatment Manual*, developed by Jacobson and colleagues (Jacobson et al., 1997). This training manual was derived from Beck’s original cognitive therapy manual, called *Cognitive Therapy of Depression* (Beck, et al., 1979). The BA treatment manual used in this study contains a total of 25 prescribed assessment and intervention techniques that therapists can use with their clients. Specific assessment techniques include, but are not limited to, conducting functional analyses, symptom reports from the BDI-II, and daily activity schedules. Specific intervention techniques that BA therapists are permitted to use include, but are not limited to, assigning activities to increase a sense of mastery or pleasure, graded task assignments, examining alternative behaviors in different situations, therapist modeling of activation strategies, and teaching clients to give themselves rewards for behavioral achievements. The manual also lists those interventions that should not be included in treatment (i.e., cognitive interventions).
In sum, over the course of treatment, therapists administering BA teach clients a series of interventions that not only help determine the life circumstances that may have precipitated the depression, but also identify the coping patterns that most likely exacerbated the depression. Ultimately the therapist helps the client decrease avoidance behaviors that maintain the disorder, while teaching the client new coping patterns that provide access to more rewarding life circumstances.

**Treatment Integrity**

In order to insure that BA was administered properly, protocol outlines were supplied to each therapist for each session after initial training. These outlines described essential steps in the procedure. The particular treatment adherence measure used in this study (Appendix A) was a modified version of the National Institute of Mental Health Collaborative Study Psychotherapy Rating Scale (CSPRS; Hollon, Evans, Elkin, & Lowery, 1984) that included the procedural steps in BA, along with a checklist of prescribed BA techniques. Also included was a list of proscribed cognitive therapy techniques.

Trained observers then viewed a random sample of the video taped treatment sessions (11%), checking off the presence of each step in the outline, along with the specific treatment interventions used that session. Furthermore, inter-rater reliability checks were performed on this same sample of videotapes. It is also important to note that the therapists participation in ongoing research team meetings specific to this investigation likely reduced therapist drift.
Outcome Measures

Depression levels were evaluated for each participant before treatment began, weekly during the course of treatment, at the termination of treatment, and at three months following the cessation of treatment. The BDI-II (Beck et al., 1996), the RHRSD (Warren, 1996) and the SCID-NP (First et al., 1997) were the three inventories used to measure depression levels in all participants. The BDI-II was completed at the intake session for both the waitlist and treatment conditions. Those in the waitlist condition only were assessed with the BDI-II at the beginning, middle, and end (i.e., every other week) of their six-week delay period. Once the wait period ended, participants in both conditions completed the BDI-II at the same weekly intervals over the course of treatment to monitor weekly changes in symptom severity. Participants in the treatment and waitlist conditions were also given the BDI-II at post-test and at the 3-month follow-up. In order to assess for the presence of Major Depressive Disorder, the SCID-NP was administered at pretest, post-test, and at the 3-month follow-up for both the treatment and waitlist conditions, while the RHRSD was completed by the therapist at those same points for both groups.

Known as one of the most widely used instruments to assess for depression, the Beck Depression Inventory (BDI; Beck et al., 1961) focuses on evaluation in both psychiatric and nonpsychiatric populations (Beck, Steer, & Garbin, 1988). According to Beck, et al. (1961), the BDI was derived from clinical observations about attitudes and symptoms displayed frequently by depressed psychiatric patients and
infrequently by nondepressed psychiatric patients. In order to correspond more closely with the diagnostic criteria for major depression in the DSM-IV, the BDI has been upgraded to the BDI-II (Steer, Ball, Ranieri, & Beck, 1997). Not unlike the original BDI, the BDI-II contains 21 items that measure 21 symptoms of depression. A four-point Likert-type scale, ranging from 0 to 3 is utilized for each item. The BDI-II measures depressive symptomatology during the two preceding weeks, unlike the original BDI that measured symptoms for the previous week only. Summing the ratings for the 21 items scores the BDI-II. The final score can then be converted to a depression rating and the guidelines for such ratings are as follows: 0–13, minimal depression; 14–19, mild depression; 20–28, moderate depression; 29–63, severe depression.

The BDI-II has demonstrated clinical utility and reliable psychometric characteristics across a broad spectrum of both clinical and nonclinical populations (Beck et al., 1996). According to Beck et al. (1996), the BDI-II had an alpha reliability coefficient of 0.92 when administered to a sample of 500 outpatients from four different psychiatric clinics. Beck et al. (1996) also confirmed the test-retest reliability by administering the BDI-II to 26 outpatients. Participants completed the BDI-II before their first therapy session and a week later, before their second therapy session. Similar results were reported for both administrations ($r = 0.93, p < 0.001$).

To provide construct validity for the BDI-II, Steer et al. (1997) administered both the BDI-II and the Symptom Checklist-90-R (SCL-90-R), an inventory often employed for assessing self-reported depression and anxiety (Derogotis, 1983) to 210
adult outpatients being evaluated for psychiatric problems. The results confirmed the construct validity of the BDI-II as the self-ratings on the BDI-II were more positively correlated with the scores on the Depression subscale than they were with the scores on the Anxiety subscale of the SCL-90-R (Hotelling $T^2 = 8.40$, $p < 0.001$). In the same study, the internal consistency for the BDI-II was also established as the mean total score on the BDI-II was 24.4 (SD = 13.3) and the coefficient alpha was 0.92. According to Steer et al. (1997), this represents high internal consistency, as the sample was moderately depressed according to the diagnostic ranges presented by Beck et al. (1996). Finally, in order to attest to the convergent validity of the BDI-II, Beck et al. (1996) compared the BDI-II to the HRSD (Hamilton, 1960) and also to the Hamilton Rating Scale for Anxiety (HARS; Hamilton, 1959). Findings presented the BDI-II as more positively correlated with the HRSD ($r = 0.71$) than with the HARS ($r = 0.47$).

Developed in the late 1950's, the HDRS is a widely used standardized interview to measure the index of severity of depressive symptoms. Because the HDRS is not intended to be a diagnostic measure, it is best completed following a clinical interview. There have been two modifications to the HSRD since the original version and it is now referred to as the Revised Hamilton Rating Scale for Depression (RHRSD; Katz, Shaw, Vallis, and Kaiser, 1995). The revised version of the HDRS now consists of 22 items and contains descriptive anchor points for each of the values for each item. Furthermore, "cognitive" items assessing hopelessness, helplessness, and worthlessness have been added. These items serve only as descriptors and are
not included in the total score for severity. Of the 17 scorable items, nine are rated on 5-point scales (0–4), and 8 on 3-point scales (0–2). Total scores range from 0 to 52. Scores on the RHRSD of 6 or below are considered to reflect normal, nondepressed functioning levels; scores of 7–17 are considered to reflect mild depression; scores of 18–24 reflect moderate depression; and scores of 25 or more are considered to reflect severe depression (Katz et al., 1995).

As slight modifications were made to the HRSD to develop the RHRSD, the strong psychometric properties of the former were not compromised (Katz et al., 1995). Thus the reliability and validity coefficients for the HRSD will be reported. In a study conducted to test the reliability and validity of the HRSD (Riskind, Beck, Brown, & Steer, 1987), 120 psychiatric outpatients were administered both the HRSD and the HARS. Results indicated the Chronbach alpha coefficient for the HRSD to be 0.73, while the average item-total correlation was 0.47, indicating satisfactory internal consistency (Riskind et al., 1987). As noted by Katz et al. (1995), data on the interrater reliability of the HRSD are impressive. In a study conducted by Hedlung and Vieweg (1979), a systematic search was executed to locate all available research reported on the HRSD from 1967 to 1979. Of the nine studies reviewed, the interrater reliability coefficient was 0.84 or above. In addition, Ziegler, Meyer, Rosen, & Biggs (1978) examined the interrater reliability of the HRSD. Ratings were made from a videotape of an interview conducted by psychiatric residents and compared to the actual interview ratings made by experienced psychologists. Videotaped ratings correlated 0.97 with the actual interview ratings.
The final inventory utilized in this study was the SCID-NP (First et al., 1997). This is a semi-structured interview administered by a trained clinician that assesses 33 of the more frequently diagnosed DSM-IV disorders in adults. The SCID-NP is designed for use in studies in which participants are not identified as psychiatric patients, such as family studies and research conducted in medical settings (Spitzer et al., 1992).

The basic structure of the SCID-NP interview is as follows. First, basic demographic information is obtained. This is followed by questions that elicit the primary complaint, history of present and past periods of psychiatric disturbance, and treatment history. To end the interview, the clinician asks general questions about current functioning, including mood, physical health, use of medications, and social functioning. A decision tree approach is utilized to test the clinician’s diagnostic hypotheses (Spitzer et al., 1992).

The SCID-NP was initially used in this investigation at intake to select the study population of interest and ensure that all participants met the conditions that were necessary for a DSM-IV diagnosis of major depression. Furthermore, it ensured that participants did not meet certain DSM-IV diagnoses that would deem them ineligible for the study. The SCID-NP was then employed at post-test and at the 3-month follow-up for both conditions to assess for major DSM-IV diagnoses and documentation of those criteria that were met.

The psychometric data on the SCID confirms it to be a reliable structured interview. In a large-scale study using 592 participants (Williams et al., 1992), the
test-retest reliability of the SCID was examined. This study included randomly matched pairs of two professionals who independently evaluated and rated the same subject within a 2-week period. Combining all disorders on the SCID yielded a weighted Kappa of 0.61 for current disorders and 0.68 for lifetime disorders. According to Williams et al. (1992), these values are comparable to those obtained with other structured diagnostic instruments.

While the reliability of the SCID has been empirically established in numerous studies (Segal, Hersen, & Van Hasselt, 1994), there has been little effort to evaluate its validity (Malgady, Rogler, & Tyron, 1992). In fact, as noted by Kranzler et al. (1996), prior to the execution of their validity study on the SCID, they found no published reports assessing the validity of the SCID. To validate SCID diagnoses, Kranzler et al. (1996) divided a sample of 100 substance abuse patients into subgroups based on the presence or absence of SCID diagnoses of different substance use disorders and comorbid conditions. These subgroups were compared on scores from interviews and questionnaires administered at the time of treatment and six months after discharge from treatment.

The authors first found support for the concurrent validity of the alcohol abuse/dependence diagnosis when, as expected, those patients diagnosed with the disorder had a more extensive family history of alcoholism compared with patients who had never met criteria for alcoholism ($F = 10.7, p < 0.001$). Excellent discriminant validity for both current ($F = 29.14, p < 0.001$) and lifetime ($F = 22.43, p < 0.001$) alcohol and drug abuse/dependence was also established for this sample.
As predicted, alcohol-related problems were less prevalent in drug-dependent patients who were not also alcoholic. Additionally, fewer drug-related problems were exuded in alcoholic patients who were also not drug dependent (Kranzler et al., 1996).

Procedure

Adult participants seeking mental health services for major depression were recruited for this study. Recruitment materials included the use of newspaper advertisements, public service announcements, public postings, and solicitation from community professionals and other healthcare agencies.

Interested individuals contacted the Anxiety Disorders research group’s telephone line and left a message requesting to participate in the study. All those interested were called back and an initial telephone screening was conducted (Appendix B). During this screening, the nature of the study was explained and individuals were assessed for depression symptoms. In addition, individuals were asked if they were currently using any psychotropic medication or currently enrolled and participating in psychotherapy of any sort. People who were not experiencing symptoms of depression, who had been taking prescription medication for their depression for less than six weeks, and/or who were currently in other psychological treatments were deemed ineligible for participation. Appropriate referrals for mental health services were offered to all ineligible callers.

Eligible individuals were then invited to participate in a second intake/assessment session held at the Psychology Clinic. Prior to the completion of
any outcome measures, individuals were presented with two consent forms (Appendix C) explaining the nature of the study and guaranteeing their confidentiality. Answers to any questions regarding the study were then provided. Individuals were then asked to sign the consent forms indicating their approval for participation. One consent form was returned to the assessor while the individual kept the second for their records.

The next step of this intake involved individuals' completing a brief demographic questionnaire (Appendix D). The BDI-II (Appendix E) was then completed and if a score of at least 20 was obtained, individuals were subsequently interviewed with the SCID-NP (available upon request). If a diagnosis of major depression was obtained and other disorders that would deem a person ineligible were ruled out, individuals were thanked for their time and told they would be notified within one week regarding their eligibility in the study. Finally, assessors completed the RHRSD (Appendix F) and if a score of at least 14 was obtained, this person was deemed eligible and allowed to progress to the next step of the study.

Eligible individuals were then telephoned within one week and invited to participate in the study. If they chose to participate, each person was assigned a research code number to be used on all subsequent forms and randomly assigned into an immediate treatment or waitlist control condition. Participants in the immediate condition were scheduled for their first appointment to begin treatment. Alternatively, those in the waitlist condition were told they would first participate in an “assessment phase” for which information about their depression would be
gathered in advance to starting therapy. These participants then came into the clinic every other week, for a total of three visits, while on the 6-week waitlist. During this visit, participants simply filled out the BDI-II and a subsequent appointment was scheduled. If the participant scored at least a 20 on the BDI-II at the end of six weeks, they were scheduled for their first appointment to begin treatment.

Participants in both the immediate and delayed treatment conditions underwent 10 one-hour sessions of BA on a weekly basis. The treatment of BA was delivered in a standardized manner by a therapist at the Psychology Clinic, whose role was, as mentioned previously, very similar to that of a “personal trainer.” At the very beginning of treatment, therapists clarified that their job was to help clients identify what was wrong in their lives and guide them in finding activities and behaviors that provide them with the pleasure and interest that was absent from their lives. This rationale was delivered with optimism, as clients were encouraged to consider changes in their behavior that would in turn lessen their depression (Jacobson et al., 1997).

Each BA treatment session involved a distinctive beginning, middle, and end. The beginning of each session included greeting the client and asking them to complete the BDI-II. Issues that would be covered throughout the rest of the session were then placed on an agenda as the therapist and client worked collaboratively to determine the most important topics for that week. Next, the BDI-II was reviewed, paying close attention to the specific questions that target suicidal behavior and
weekly activity levels. Any homework assignments the client completed between sessions were also discussed.

About 10 minutes into the session, the client and therapist progressed to the middle of the session where they worked jointly on the previously set agenda items. The therapist typically did not stray from the prescribed agenda unless an extraordinary issue arose (e.g., suicidality). Essentially, the middle part of the session was used to work on the issues of importance.

As the session came to a close, the therapist briefly reviewed the topics that were covered and assigned a between-session assignment in relation to what was discussed that day. It was the therapist’s responsibility to make sure the homework was well understood by the client and that it would be completed. As treatment progressed, the client began to assume responsibility for reviewing the session and assigning homework to him or herself. Finally, the client was given the opportunity to comment on that days’ session and a date was set for the next session.

After 10 weeks of treatment, each participant returned to the clinic the following week for a post-test session. Each participant met with a clinical assessor who administered the BDI-II and the SCID-NP. Finally, the assessor independently completed the RHRSD.

Next, telephone calls were made to schedule a three-month follow-up appointment. During this visit, participants completed the BDI-II and were interviewed with the SCID-NP. Again, the assessor independently completed the RHRSD. At this time, assessment was complete and participants’ involvement in the
study was concluded. Research folders were then closed and transferred to the
Department of Psychology at Western Michigan University where they will remain in
a locked cabinet for at least three years. They will then be destroyed.

Finally, the research data for each participant was kept in personal folders in a
locked filing system established at the Psychology Clinic. Maintaining these filing
systems and participant folders was the responsibility of the researcher. A master list
(Appendix G), used to ensure the confidentiality of the research data, was the only
link between participants and their research code numbers. Additionally, a universal
data collection form (Appendix H) was used to record all assessment information for
each participant. Progress notes and other clinic related treatment documentation for
each participant were kept separate from the research data and was managed by the
Psychology Clinic.
CHAPTER IV

RESULTS

Analysis Plan

For this investigation, it was hypothesized that depressive symptoms would decrease for individuals in the immediate and waitlist comparison groups only after treatment had been administered to each group (see Figure 1). Principal measures included outcome ratings on the Beck Depression Inventory-II and Revised Hamilton Rating Scale for Depression.

Figure 1. Projected Therapeutic Gains on the BDI-II for the Total Sample, by Condition.
In order to test this hypothesis, this study incorporated a pretest/post-test waitlist comparison group and included a 2 X 3 repeated-measures design. Two levels of the between-group factor included the waitlist and immediate treatment conditions. Three levels of the within-group factor consisted of assessment time (pretest, post-test, and follow-up). All participants were randomly assigned into an immediate treatment or waitlist control condition. A planned comparison between individuals receiving immediate treatment versus those assigned to a 6-week waitlist was expected to demonstrate whether the treatment was effective. Likewise, it was expected that waitlist-assigned individuals would demonstrate symptom reduction only after they had received the BA intervention. The within-group comparisons were expected to reveal change for subjects from pretest to post-test for both conditions, with a stable symptom pattern for the waitlist-assigned individuals during their pretreatment phase.

Analyses were computed for all participants who fulfilled minimum requirements of their assigned condition. For both groups, data were included in the analysis if participants completed a minimum of six sessions of BA. The first primary analysis of the study consisted of a repeated-measures analyses of variance (ANOVA) that examined outcome measures across time for participants in both groups who completed BA. Given the significant omnibus F-tests, follow-up paired t-tests were computed separately for each dependent variable and time course. More specifically, for the immediate treatment condition, differences on each dependent measure were examined between (a) pretest and post-test, (b) pretest and 3-month
follow-up, and (c) post-test and 3-month follow-up. For the waitlist condition, differences on dependent measures were examined at those same assessment points, but also between (d) the beginning and end of the waitlist assessment.

In order to detect differences in depression levels between the immediate treatment and waitlist condition, further comparisons were made at (a) pretest, (b) post-test, and (c) 3-month follow-up. Independent samples t-tests were performed at those time periods with BDI-II and RHRSD scores as dependent variables.

So as to maximize the number of total participants entered into the analyses, and therefore increase the power of the study, the immediate and waitlist group data were thereupon combined and assessed for change over time. Repeated-measures ANOVA's were performed on the collapsed group. Planned within-group comparisons in mean BDI-II and RHRSD scores for this total sample were then examined between (a) pretest and post-test, (b) pretest and 3-month follow-up, and (c) post-test and 3-month follow-up. In a subsequent 'missing data' analysis, post-test scores for each participant were carried forward to substitute for the missing follow-up data where necessary. These analyses were also conducted using multiple repeated-measures ANOVAS, along with within-subject contrasts to detect exactly where the differences between times existed.

Because this computation added only two more participants into the analysis, a subsequent ANOVA was employed. Instead of post-test BDI-II scores, week-10 BDI-II scores were carried forward and placed into missing post-test and follow-up cells respectively. This makes intuitive sense given only one week passed between
session 10 and the post-test assessment time. This procedure added five more participants into the analysis. Because therapists did not assess participants with the RHRSD at session 10, this analysis was only completed using BDI-II scores.

In a final attempt to include all 17 participants in the collapsed group analysis, a less conservative ANOVA was subsequently conducted. It was possible to include all 17 participants by taking the mean BDI-II and RHRSD scores for the whole distribution at a point in time (pre-, post-, and follow-up) and substituting that number for the missing data for each variable accordingly. Finally, an intent to treat analysis was performed on the entire sample to control for attrition.

Preliminary Analyses

Demographic information for the entire sample (i.e., completers and dropouts) is displayed in Table 1. Of the 17 participants who completed the study, 65% were male (n = 11) and 35% were female (n = 6). The mean age of participants was 37.8 years old with a range from 20 to 61 years of age. At the time of evaluation, the racial composition of the group was 94% Caucasian and 6% Alaskan-American. Nearly one-half of the participants were divorced (47%); the remainder was single/never married (41%), or married (12%). All participants in the study reported receiving their high school diploma. A total of 12% of the group completed high school or received their GED, 47% reported their years of education as more than 12 years, but less than 16, while 18% received at least 16 years of schooling, and 23% reported receiving 16-plus years of education. The household income of the group
## Table 1

Demographic Variables for Completers, Dropouts and the Total Sample

<table>
<thead>
<tr>
<th>Variable</th>
<th>Completers (n = 14)</th>
<th>Dropouts (n = 14)</th>
<th>Total Sample (n = 13)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>11 (64.7%)</td>
<td>8 (57.1%)</td>
<td>19 (61.3%)</td>
</tr>
<tr>
<td>Female</td>
<td>6 (35.3%)</td>
<td>6 (42.9%)</td>
<td>12 (38.7%)</td>
</tr>
<tr>
<td><strong>Mean Age</strong></td>
<td>37.8</td>
<td>38</td>
<td>37.9</td>
</tr>
<tr>
<td><strong>Ethnic Group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>0 (0.0%)</td>
<td>1 (7.1%)</td>
<td>1 (3.2%)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>0 (0.0%)</td>
<td>1 (7.1%)</td>
<td>1 (3.2%)</td>
</tr>
<tr>
<td>International</td>
<td>0 (0.0%)</td>
<td>1 (7.1%)</td>
<td>1 (3.2%)</td>
</tr>
<tr>
<td>Alaskan American</td>
<td>1 (5.9%)</td>
<td>0 (0.0%)</td>
<td>1 (3.2%)</td>
</tr>
<tr>
<td>Caucasian</td>
<td>16 (94.1%)</td>
<td>10 (71.4%)</td>
<td>26 (83.9%)</td>
</tr>
<tr>
<td>Did Not Report</td>
<td>0 (0.0%)</td>
<td>1 (7.1%)</td>
<td>1 (3.2%)</td>
</tr>
<tr>
<td><strong>Relationship Status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single, Never Married</td>
<td>7 (41.2%)</td>
<td>5 (35.7%)</td>
<td>12 (38.7%)</td>
</tr>
<tr>
<td>Divorced</td>
<td>8 (47.1%)</td>
<td>3 (21.4%)</td>
<td>11 (35.5%)</td>
</tr>
<tr>
<td>Married</td>
<td>2 (11.8%)</td>
<td>6 (42.9%)</td>
<td>8 (25.8%)</td>
</tr>
<tr>
<td><strong>Years of Education</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than 12 Years</td>
<td>0 (0.0%)</td>
<td>2 (14.3%)</td>
<td>2 (6.5%)</td>
</tr>
<tr>
<td>12 Years or GED</td>
<td>2 (11.8%)</td>
<td>3 (21.4%)</td>
<td>5 (16.1%)</td>
</tr>
<tr>
<td>More than 12, Less than 16</td>
<td>8 (47.1%)</td>
<td>5 (35.7%)</td>
<td>13 (41.9%)</td>
</tr>
<tr>
<td>16 Years</td>
<td>3 (17.6%)</td>
<td>0 (0.00%)</td>
<td>3 (9.7%)</td>
</tr>
<tr>
<td>16+ Years</td>
<td>4 (23.5%)</td>
<td>4 (28.6%)</td>
<td>8 (25.8%)</td>
</tr>
<tr>
<td><strong>Household Income</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Under $10,000 Per Year</td>
<td>4 (23.5%)</td>
<td>4 (28.6%)</td>
<td>8 (25.8%)</td>
</tr>
<tr>
<td>$10,000 to $20,000 Per Year</td>
<td>5 (29.4%)</td>
<td>2 (14.3%)</td>
<td>7 (22.6%)</td>
</tr>
<tr>
<td>$20,000 to $30,000 Per Year</td>
<td>4 (23.5%)</td>
<td>3 (21.4%)</td>
<td>7 (22.6%)</td>
</tr>
<tr>
<td>Over $30,000 Per Year</td>
<td>4 (23.5%)</td>
<td>3 (21.4%)</td>
<td>7 (22.6%)</td>
</tr>
<tr>
<td>Did Not Report</td>
<td>0 (0.00%)</td>
<td>2 (14.3%)</td>
<td>2 (6.5%)</td>
</tr>
</tbody>
</table>
was 24% earning under $10,000/year, 29% between $10–20,000/year, 24% between
$20–30,000/year, and 23% earned over $30,000/year.

Background data related to mental health history was also collected for each
person who completed and dropped out of the investigation (see Table 2). Of the 17
completers, 13 participants entered the study with an Axis I diagnosis of Major
Depressive Disorder, Recurrent/Moderate, while two participants were diagnosed
with Major Depressive Disorder, Recurrent/Severe without psychotic features. One
participant suffered from Major Depressive Disorder, Recurrent/Mild and one from
Major Depressive Disorder, Single Episode/Mild. It should also be noted that the
SCID-NP also identified three participants with Dysthymia, rendering them a formal
diagnosis of “double depression.” No participants obtained a formal Axis II
diagnosis. On Axis III, one participant reported high blood pressure, one suffered
from high cholesterol, one reported carrying a diagnosis of multiple sclerosis, and one
reported having diabetes. Twelve participants did not qualify for an Axis III
diagnosis. For Axis IV, one participant reported parent/child relational problems, one
reported economic problems, two participants reported relational problems-NOS, and
six participants reported occupational problems. Furthermore, seven participants
reported a secondary Axis IV diagnosis of problems with primary support group (n = 3),
relational problems-NOS (n = 1), parent/child relational problems (n = 1),
academic problems (n = 1), and economic problems (n = 1). One participant was
given a third Axis IV diagnosis of occupational problems. Five participants were not

53
Table 2

Clinical Characteristics of Completers and Dropouts

<table>
<thead>
<tr>
<th>Variable</th>
<th>Completers (n = 17)</th>
<th>Dropouts (n = 14)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medication Status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>5 (29.4%)</td>
<td>6 (42.9%)</td>
</tr>
<tr>
<td>No</td>
<td>12 (70.6%)</td>
<td>8 (57.1%)</td>
</tr>
<tr>
<td>Current RX</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prozac</td>
<td>2 (11.8%)</td>
<td>2 (14.3%)</td>
</tr>
<tr>
<td>Celexa</td>
<td>2 (11.8%)</td>
<td>0 (0.00%)</td>
</tr>
<tr>
<td>Paxil</td>
<td>1 (5.9%)</td>
<td>1 (7.1%)</td>
</tr>
<tr>
<td>Remeron</td>
<td>0 (0.00%)</td>
<td>1 (7.1%)</td>
</tr>
<tr>
<td>Did Not Report</td>
<td>0 (0.00%)</td>
<td>2 (14.3%)</td>
</tr>
<tr>
<td>Previous TX for MDD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>11 (64.7%)</td>
<td>13 (92.9%)</td>
</tr>
<tr>
<td>No</td>
<td>6 (35.3%)</td>
<td>0 (0.00%)</td>
</tr>
<tr>
<td>Did Not Report</td>
<td>0 (0.00%)</td>
<td>1 (7.1%)</td>
</tr>
<tr>
<td>Number of TX Episodes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>6 (35.3%)</td>
<td>0 (0.00%)</td>
</tr>
<tr>
<td>1</td>
<td>2 (11.8%)</td>
<td>3 (21.4%)</td>
</tr>
<tr>
<td>2</td>
<td>0 (0.00%)</td>
<td>2 (14.3%)</td>
</tr>
<tr>
<td>3</td>
<td>1 (5.9%)</td>
<td>0 (0.00%)</td>
</tr>
<tr>
<td>4 or more</td>
<td>2 (11.8%)</td>
<td>4 (28.4%)</td>
</tr>
<tr>
<td>Did Not Report</td>
<td>0 (0.00%)</td>
<td>5 (35.7%)</td>
</tr>
</tbody>
</table>

Note. RX = Prescription; TX = Treatment; MDD = Major Depressive Disorder.
given an Axis IV diagnosis. Finally, the Axis V Global Assessment of Functioning (GAF) scores ranged from 51-68, with a mean GAF of 60.

When asked to describe any existing treatment for depression at the time of the initial screening, five participants (29.4%) reported taking one psychotropic medication to treat their depression. Two of those participants reported taking Prozac, two were being treated with Celexa, and one reported taking Paxil. A majority of participants (70.6%; n = 12) reportedly were not using medication for their depression. All 17 participants reported they were not concurrently receiving another type of psychotherapy. In regards to previous episodes of treatment for depression, two participants reported they had received one previous episode of treatment, one had received three previous episodes of treatment, and two participants reported receiving four or more episodes of treatment for their depression in the past. Twelve participants reported this episode of treatment as their first.

Demographic information for dropouts (i.e., those who terminated treatment before completing at least six sessions) is also displayed in Table I. Of the 14 dropouts, 8 (57.1%) were male and 6 (42.9%) were female. The mean age for this sample was 38 years old, with a range from 19 to 66 years of age. The racial composition of this group was 71.4% Caucasian, while African-American, Hispanic, and international/non-US citizen each made up 7.1%. One person did not report their ethnicity. Forty-three percent of dropouts were married; the remainder was single/never married (35.7%) or divorced (21.5%). Two individuals (14.3%) who dropped out of the study reported not completing high school, 21.4% reported
completing high school or GED, while the remaining 64.3% reported their years of education as more than 12 years of education. The household income of this group was 28.6% earning under $10,000/year, 14.3% between $10–20,000/year, 21.4% between $20–30,000/year, and 21.4% over $30,000/year.

The clinical characteristics of those who dropped out are also shown in Table 2. All 14 dropouts entered the study with an Axis I diagnosis of Major Depressive Disorder. Over half (57.1%) of the dropouts reported their depression as recurrent/moderate. Moreover, single and recurrent episode/moderate and recurrent episode/severe each made up 14.3%. On the remaining axes, those who terminated treatment prematurely did not report clinical information that made them significantly different from those who completed treatment.

When asked to describe any existing treatment for depression at the time of the initial screening, slightly one-half of dropouts (42.9%; n = 6) reported taking one psychotropic medication. The remaining 57.1% (n = 8) reportedly were not taking medication for their depression. All 14 dropouts reported they were not concurrently receiving another type of psychotherapy. In regards to previous episodes of treatment for depression, three participants reported they had received one previous episode of treatment, two had received two episodes of treatment, and three reported receiving four or more episodes of treatment for their depression in the past. Five dropouts reported this episode of treatment as their first.

In regards to the point at which these individuals dropped out of the investigation, over one-half (64.3%; n = 9) of the 14 dropouts terminated participation.
after intake but before they were randomized into the immediate treatment or waitlist condition. Of the five people who actually began treatment, three were randomized into the immediate treatment group and two into the waitlist group. Moreover, a single person dropped out after session one, after session three and after session four, while two people terminated after session two. Finally, it may be important to note that those who dropped out were not significantly more or less depressed at the initial screening than those who completed the study, according to the BDI-II, \( t(29) = 0.74, \ p = \text{ns} \), and the RHRSD, \( t(29) = 0.61, \ p = \text{ns} \).

Primary Analyses

The means, standard deviations, and results of the between group analyses are presented in Table 3 and Figure 2. The first point of analysis consisted of an

Table 3

<table>
<thead>
<tr>
<th></th>
<th>TX Group</th>
<th>WL Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Pre-</td>
<td>32.78</td>
<td>(6.3)</td>
</tr>
<tr>
<td>Post-</td>
<td>3.83</td>
<td>(3.3)</td>
</tr>
<tr>
<td>Follow-up</td>
<td>7.17</td>
<td>(8.2)</td>
</tr>
</tbody>
</table>

Note. BDI-II = Beck Depression Inventory-Second Edition; TX = Treatment; WL = Waitlist.
independent-samples $t$-test to assess group differences at intake. There were no significant differences at time of initial intake on both the BDI-II, $t(15) = 1.04, p = ns$, or the RHRSD, $t(15) = 0.94, p = ns$. This suggests that both immediate treatment and waitlist conditions started treatment at comparable levels of symptom severity (see Table 4 and Figure 3).

The next point of analysis examined group BDI-II means immediately following treatment for those in the immediate treatment group and immediately following the waiting period for those in the waitlist group. The results of this independent samples $t$-test was significant, $t(12) = 3.58, p < 0.004, r = 0.72$. In a similar analysis, an independent samples $t$-test compared group means at the same
Table 4
BDI-II and RHRSD Scores at Pretreatment

<table>
<thead>
<tr>
<th>Variable</th>
<th>TX Group (n = 9)</th>
<th>WL Group (n = 22)</th>
<th>t(df) and p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>BDI-II</td>
<td>32.8 (6.3)</td>
<td>29.8 (5.6)</td>
<td>t(15) = 1.04, p = 0.32</td>
</tr>
<tr>
<td>RHRSD</td>
<td>18.1 (4.0)</td>
<td>20.0 (4.2)</td>
<td>t(15) = 0.94, p = 0.36</td>
</tr>
</tbody>
</table>

Note. TX = Treatment; WL = Waitlist; BDI-II = Beck Depression Inventory- Second Edition; RHRSD = Revised Hamilton Rating Scale for Depression.

Figure 3. Mean BDI-II and RHRSD Scores at Pretreatment.

point for the waitlist condition but at 3-month follow-up for the immediate treatment condition. This analysis revealed statistically significant differences in mean BDI-II
scores with $t(12) = 2.88, p < 0.02, r = 0.64$. These results suggest that treatment with BA was superior to remaining on a waitlist and that it was simply not the passage of time that caused reductions in depression.

The next independent sample $t$-tests compared means at post-test on each dependent variable and significant differences were found between the groups, with $t(6) = 2.63, p < 0.04, r = 0.73$ on the BDI-II and $t(5) = 5.04, p < 0.004, r = 0.92$ on the RHRSD. This result suggests that on both dependent measures, those in the immediate treatment condition were significantly less depressed than those in the waitlist condition after receiving BA treatment, but at the 3-month follow up, significant differences were not found on the BDI-II [$t(7) = 0.21, p = \text{ns}$], but apparent on RHRSD scores, $t(7) = 2.55, p < 0.04, r = 0.69$. With respect to the RHRSD, the significant difference suggested that the immediate treatment group showed the greatest reduction in symptoms.

Although both immediate and waitlist groups started out with comparatively similar depression levels, after receiving treatment, one group appeared more depressed than the other. This result should be interpreted cautiously as only two waitlist condition participants entered into the analysis at post-test for each outcome measure, and three waitlist condition participants at follow-up on each outcome measure. Despite low participant numbers, effect sizes for each independent $t$-test appear large enough to conclude that BA treatment is more effective in decreasing depression when compared to a 6-week wait period and that participant’s depression levels did not decrease simply due to passage of time.
The next set of analyses was computed within-groups for the immediate and waitlist conditions separately over three assessment times (i.e., pretest, post-test, and follow-up) on both dependent measures (see Figures 4 and 5). For the immediate treatment group, there was a significant reduction in BDI-II scores from pretest through to follow-up, with \( F(2, 8) = 82.15, p < 0.001, \eta = 0.97 \). Results were similar for RHRSD scores, with \( F(2, 8) = 140.77, p < 0.001, \eta = 0.99 \). Given the significant omnibus \( F \)-tests, within-subjects contrasts were computed on both dependent measures. Significant reductions in scores were found between pretest and post-test on the BDI-II, \( F(1, 4) = 93.52, p < 0.001, \eta = 0.98 \), and the RHRSD, \( F(1, 4) = 196.00, p < 0.001, \eta = 0.99 \). Likewise, significant reductions in depression scores between pretest and follow-up were found on the BDI-II, \( F(1, 4) = 90.99, p < 0.001, \eta = 0.98 \), and the RHRSD, \( F(1, 4) = 130.0, p < 0.001, \eta = 0.98 \). A final within-subjects contrast examined means at post-test versus 3-month follow-up. There was no significant differences found at these points on both the BDI-II, \( F(1, 4) = 0.022, p = ns \), and the RHRSD, \( F(1, 4) = 3.33, p = ns \). These results suggest that for those in the immediate treatment group, depression levels decreased significantly over time. Furthermore, these individuals maintained treatment gains for at least three months after finishing BA treatment.

Because only three participants finished treatment through to the 3-month follow-up in the waitlist condition, it was not possible to conduct a repeated-measures ANOVA on this group separately. Alternatively, paired samples \( t \)-tests were performed on both dependent measures. The results of the pretest to post-test
Figure 4. Pretreatment, Post-treatment, and 3-Month Follow-up Means for BDI-II Scores, by Condition.

Figure 5. Pretreatment, Post-treatment, and 3-Month Follow-up Means for RHRSD Scores, by Condition.
comparison showed a significant reduction in BDI-II scores, with $t(1) = 31.0, p = 0.02$, but not RHRSD scores, with $t(1) = 3.33, p = ns$. The second comparison of means, between pretest and follow-up, showed no significant change in BDI-II scores, with $t(2) = 2.65, p = ns$. On the contrary, a significant difference was found at this same point in RHRSD scores, with $t(2) = 4.5, p < 0.05$. The comparison between post-test and follow-up means could not be calculated, as there were not enough participants to enter into the analysis.

In order to control for the relatively small sample size, the immediate and waitlist groups were then combined, allowing for a within-subjects comparison from intake to post-test to the 3-month follow-up. Prior to collapsing the two groups, it was necessary to show that participants in the waitlist condition did not change (i.e., become significantly more or less depressed) from the initial screening to the end of the 6-week wait period. A repeated-measures ANOVA, examining the means at pretest through to the end of the 6-week waitlist period showed that participants’ depression levels remained uniformly the same, $F(3, 21) = 0.101, p = ns$.

Given this non-significant finding, the immediate and waitlist conditions were then collapsed and assessed for change over time. This analysis was performed for all participants who completed at least six sessions of treatment, regardless of their treatment condition. Although only 6 of 17 participants completed all outcome measures at pretest, post-test and follow-up, results revealed a significant reduction in both BDI-II scores, $F(2, 10) = 47.34, p < 0.001$, eta = 0.95, and RHRSD scores, $F(2, 10) = 83.68, p < 0.001$, eta = 0.97. Furthermore, within-subject contrasts revealed a
significant reduction in BDI-II scores from pretest to post-test, $F(1, 5) = 70.68, p < 0.001$, eta = 0.97, and in RHRSD scores at that same assessment point, $F(1, 5) = 81.22, p < 0.001$, eta = 0.97. Similarly, within-subjects contrasts revealed a statistically significant reduction in BDI-II scores from pretest to follow-up, $F(1, 5) = 40.86, p = 0.001$, eta = 0.99, and in RHRSD scores at that same assessment point, $F(1, 5) = 116.02, p < 0.001$, eta = 0.98. Depression levels remained at a relative constant from post-test to follow-up as significant differences were not found on both the BDI-II, $F(1, 5) = 0.30, p = ns$, or the RHRSD, $F(1, 5) = 0.56, p = ns$.

In the next analysis, post-test scores for each participant were carried forward to complete the missing follow-up data where necessary. A repeated-measures ANOVA on the total sample revealed a significant reduction from pretest through to the 3-month follow-up on both the BDI-II, $F(2, 14) = 66.69, p < 0.001$, eta = 0.95, and the RHRSD, $F(2, 12) = 117.59, p < 0.001$, eta = 0.98. Further within-subject contrasts unveiled a significant reduction in BDI-II scores from pretest to post-test, $F(1, 7) = 89.33, p < 0.001$, eta = 0.96, and in RHRSD scores from pretest to post-test, $F(1, 6) = 114.09, p < 0.001$, eta = 0.97. Similarly, within-subjects contrasts revealed a statistically significant reduction in BDI-II scores from pretest to follow-up, $F(1, 7) = 59.7, p < 0.001$, eta = 0.95, and in RHRSD scores at the same assessment point, $F(1, 6) = 163.36, p < 0.001$, eta = 0.98. Depression levels remained at a relative constant from post-test to follow-up as significant differences were not found on both the BDI-II, $F(1, 7) = 0.31, p = ns$, or the RHRSD, $F(1, 6) = 0.56, p = ns$. 

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Unfortunately this computation added only two more participants (n = 8) into the analysis, so a similar ANOVA was employed increasing the sample size to 13. For those who finished ten weeks of BA, their 10-week BDI-II score was carried forward and placed into missing post-test and follow-up cells respectively. The results yielded a statistically significant difference over time from pretest through to follow-up, $F(2, 24) = 73.00, p < 0.001, \eta = 0.93$. Further within-subjects contrasts revealed exactly where those differences existed. As would be expected, there was significant differences between pretest and post-test, $F(1, 12) = 87.81, p < 0.001, \eta = 0.94$, and between pretest and follow up, $F(1, 12) = 83.64, p < 0.001, \eta = 0.94$. Participants BDI-II scores stayed relatively constant from post-test to follow-up, $F(1, 12) = 0.96, p = ns$.

So as to include all 17 participants and increase the power in the collapsed group analysis, a less conservative ANOVA was subsequently conducted (see Table 5 for means and standard deviations and Figure 6). It was possible to include all 17 participants by taking the mean BDI-II and RHRSD scores for the whole sample at a given point in time, and placing that mean into the missing data for each variable accordingly. Statistically significant differences were found from pretest through to follow-up on the BDI-II, $F(2, 32) = 180.69, p < 0.001, \eta = 0.96$, and on the RHRSD, $F(2, 32) = 223.19, p < 0.001, \eta = 0.97$. Given the significant results, further within-subject contrasts were computed. Significant differences were found between pretest and post-test on the BDI-II, $F(1, 16) = 266.89, p < 0.001, \eta = 0.97$. 

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Table 5

Pretreatment, Post-treatment, and 3-Month Follow-up Means for BDI-II and RHRSD Scores for the Total Sample

<table>
<thead>
<tr>
<th>Depression Measure</th>
<th>n</th>
<th>M</th>
<th>(SD)</th>
<th>F(dfs) and p</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI-II</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Across time</td>
<td>17</td>
<td>31.35</td>
<td>(6.0)</td>
<td>F (2, 32) = 180.69, p &lt; 0.001&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Pre-</td>
<td>17</td>
<td>5.94</td>
<td>(3.4)</td>
<td>F (1, 16) = 266.89, p &lt; 0.001&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Post-</td>
<td>17</td>
<td>7.76</td>
<td>(5.2)</td>
<td>F (1, 16) = 1.92, p &lt; ns&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Follow-up</td>
<td>17</td>
<td>19.0</td>
<td>(4.1)</td>
<td>F (1, 16) = 228.25, p &lt; 0.001&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>RHRSD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Across time</td>
<td>17</td>
<td>3.82</td>
<td>(1.8)</td>
<td>F (1, 16) = 0.21, p &lt; 0.001&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Pre-</td>
<td>17</td>
<td>4.0</td>
<td>(1.3)</td>
<td>F (1, 16) = 255.00, p &lt; 0.001&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>Post-</td>
<td>17</td>
<td>1.8</td>
<td></td>
<td>F (1, 16) = 0.96, p = ns,</td>
</tr>
<tr>
<td>Follow-up</td>
<td>17</td>
<td>4.0</td>
<td>(1.3)</td>
<td>F (1, 16) = 0.21, p = ns.</td>
</tr>
</tbody>
</table>

Note: BDI-II = Beck Depression Inventory-Second Edition; RHRSD = Revised Hamilton Rating Scale for Depression; TX = Treatment; WL = Waitlist.

and on the RHRSD, F (1, 16) = 228.25, p < 0.001, eta = 0.97. Similar differences were found between pretest and 3-month follow-up on both outcome measures, F (1, 16) = 220.58, p < 0.001, eta = 0.97, on the BDI-II, and F (1, 16) = 255.0, p < 0.001, eta = 0.97, on the RHRSD. Statistically significant differences were not found between post-test and 3-month follow-up on BDI-II scores, F (1, 16) = 0.96, p = ns, and RHRSD scores, F (1, 16) = 0.21, p = ns.

Although calculated in different manners, these various analyses for the total sample suggest that a significant reduction in depression scores existed from
pretreatment to post-treatment, while those gains were maintained respectively at the 3-month follow-up point. Overall, it implies that depression levels decreased significantly for those participants who received six or more sessions of BA.

Intent-to-Treat Analyses

In order to control for participants who dropped out of the study, an intent-to-treat analysis was performed by utilizing the dropouts’ pretest scores on both dependent measures as their post-treatment scores. The following analysis is particularly meaningful for this study due to the high attrition rate, established at approximately 45% (14 of 31 participants dropped out).
Because this analysis assumes that those who prematurely dropped out of the study achieved no gains from BA treatment, this is considered a conservative analysis. This analysis guides our understanding regarding whether BA was effective for the entire sample, as opposed to just those who completed the study. For the intent-to-treat analyses, changes in BDI-II scores, RHRSD scores, and changes in diagnosis from pretest to post-test were examined. Paired samples $t$-tests showed a significant reduction in depression symptomatology in both BDI-II scores, $t(31) = 6.24, p < 0.001, r = 0.75$, and RHRSD scores, $t(31) = 2.86, p < 0.01, r = 0.46$. These analyses suggest that even when controlling for dropouts, BA was associated with a significant reduction in depression levels. Furthermore, BA was associated with a significant reduction in the number of Major Depression diagnoses from pretest to post-test, even when dropouts were factored into the analysis, $t(30) = 3.23, p < 0.01, r = 0.51$.

Post-hoc Analyses

In order to clarify the role of behavioral activation in the treatment of more "real world" people who suffer from Major Depressive Disorder, comparisons were made between persons who were and were not being treated with psychotropic drugs. These are the people who realistically present themselves in practice settings for treatment. Therefore, a set of post-hoc analyses were conducted by separating those participants who were receiving independently prescribed pharmacological treatment for depression while receiving BA, from those who were receiving only BA (i.e.,
medication participants versus no medication participants). Each sample was examined at three assessment points (i.e., pretest, post-test, and follow-up) and mean BDI-II and RHRSD scores served as dependent variables. For this sample of participants who completed the treatment, approximately one-third (29.4%) were taking medications for their depression (n = 5).

Due to similar attrition problems as seen in previous analyses, a more liberal analysis was computed to increase the number of participants entered into the analysis. For all between-group and within-group computations, the mean post-test and follow-up score for that sample was substituted for missing data at each respective assessment point. Independent samples t-tests with medication status as the independent variable and BDI-II and RHRSD scores as dependent variables revealed non-significant findings at each assessment point (see Table 6 and Figures 7 and 8 for details). More specifically, statistically significant differences were not found at pretest on both the BDI-II, $t(15) = 1.09, p = ns$, and RHRSD, $t(15) = 1.33, p = ns$. Also at post-test, results revealed no significant difference between the two groups on the BDI-II, $t(15) = 1.58, p = ns$, and RHRSD, $t(15) = 0.91, p = ns$. The same followed at the 3-month follow up, with no significant differences found in BDI-II scores, $t(15) = 0.62, p = ns$, and RHRSD scores, $t(15) = 1.28, p = ns$. This analysis suggests that those taking medication did not have higher or lower BDI-II or RHRSD scores ($M = 33.8$) prior to starting treatment than those who were not taking medication ($M = 30.3$). Furthermore, after receiving BA treatment, those that were taking medication did not achieve significantly lower or higher BDI-II or RHRSD.
Table 6

Pretreatment, Post-treatment, and 3-Month Follow-up Means for BDI-II and RHRSD Scores as a Function of Medication Status

<table>
<thead>
<tr>
<th></th>
<th>No Medication</th>
<th>Medication</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>M</td>
</tr>
<tr>
<td>BDI-II</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-</td>
<td>12</td>
<td>30.33</td>
</tr>
<tr>
<td>Post-</td>
<td>12</td>
<td>6.75</td>
</tr>
<tr>
<td>Follow-up</td>
<td>12</td>
<td>7.25</td>
</tr>
<tr>
<td>RHRSD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-</td>
<td>12</td>
<td>19.83</td>
</tr>
<tr>
<td>Post-</td>
<td>12</td>
<td>4.08</td>
</tr>
<tr>
<td>Follow-up</td>
<td>12</td>
<td>4.25</td>
</tr>
</tbody>
</table>

Note. BDI-II = Beck Depression Inventory-Second Edition; RHRSD = Revised Hamilton Rating Scale for Depression.

scores than those who received BA singly. Thus, this analysis does not argue for an additive effect of medication in this investigation.

A more conservative between-group comparison in which exact pretest, posttest and follow-up data were examined between the medication versus no medication participants showed very similar results as above, with no significant differences found at any assessment point on both outcome measures. Again at pretest, participants did not differ significantly on their BDI-II or RHRSD scores,
Figure 7. Mean BDI-II Scores as a Function of Medication Status.

Figure 8. Mean RHRSD Scores as a Function of Medication Status.
\( t(15) = 1.09, p = \text{ns}, \) and \( t(15) = 1.33, p = \text{ns}. \) Similar results followed at post-test on BDI-II and RHRSD scores, \( t(6) = 1.47, p = \text{ns}; t(5) = 0.88, p = \text{ns}, \) and at the 3-month follow-up on BDI-II and RHRSD scores, \( t(7) = 0.59, p = \text{ns}; t(7) = 1.21, p = \text{ns}. \)

In order to describe how each group of participants responded to BA separately, within-group computations were executed. For those being treated with both medication and BA, a repeated-measures ANOVA was executed, again implementing the more liberal procedure in which whole sample post-test and follow-up means are substituted for missing post-test and follow-up data respectively. This analysis was chosen over the more conservative procedure of using exact pre-, post- and follow-up means as only two participants entered into that particular analysis. Results displayed a significant decrease over time from pretest to post-test through to follow-up on both BDI-II scores, \( F(2, 8) = 54.03, p < 0.001, \text{eta} = 0.96, \) and RHRSD scores, \( F(2, 8) = 59.22, p < 0.001, \text{eta} = 0.97. \) Further within-subjects comparisons were made to detect where exact reductions in depression scores appeared. Statistically significant differences were found from pretest to post-test on the BDI-II and RHRSD, \( F(1, 4) = 102.78, p = 0.001, \text{eta} = 0.98; F(1, 4) = 67.06, p = 0.001, \text{eta} = 0.97, \) and from pretest to the 3-month follow-up on the BDI-II and RHRSD, \( F(1, 4) = 65.85, p = 0.001, \text{eta} = 0.97; F(1, 4) = 56.74, p = 0.002, \text{eta} = 0.97. \) Depression levels remained relatively constant for this group from post-test to follow-up as no significant differences were revealed between these assessment points on either the BDI-II, \( F(1, 4) = 2.43, p = \text{ns}, \) or the RHRSD, \( F(1, 4) = 0.17, p = \text{ns}. \)
Similar reductions in BDI-II and RHRSD scores over time were found for those participants receiving BA singly. As in the previous analysis, the more liberal ANOVA procedure was instituted, as opposed to comparing exact means due to low sample sizes included in this analysis. Results displayed a significant decrease over time from pretest to post-test through to follow-up on both BDI-II scores, $F(2, 22) = 139.48, p < 0.001, \eta = 0.96$, and RHRSD scores, $F(2, 22) = 161.67, p < 0.001, \eta = 0.97$. Given this finding, within-subjects comparisons were made. Statistically significant differences were found from pretest to post-test on the BDI-II and RHRSD, $F(1, 11) = 210.41, p < 0.001, \eta = 0.98; F(1, 11) = 160.32, p < 0.001, \eta = 0.97$, and from pretest to follow-up on the BDI-II and RHRSD, $F(1, 11) = 143.86, p < 0.001, \eta = 0.96; F(1, 11) = 199.20, p < 0.001, \eta = 0.97$. Depression levels remained relatively constant for this group from post-test to the 3-month follow-up as no significant differences were found between these assessment points on either the BDI-II, $F(1, 11) = 0.17, p = \text{ns}$, or RHRSD, $F(1, 11) = 0.10, p = \text{ns}$. Overall, the post-hoc analyses suggest that in this investigation the addition of medication to BA treatment does not significantly reduce depression levels any more than treatment with BA alone.

Diagnostic Outcome and Recovery

In order to assess for clinically significant decreases in depressive symptomatology, this investigation also compared the number of participants who met *DSM-IV* criteria at initial screening to those who met criteria after receiving
treatment with BA. The Structured Clinical Interview for DSM-IV determined diagnostic outcome.

At the initial screening, all 17 completers (100%) met DSM-IV criteria for Major Depressive Disorder. At the end of treatment, of the three that returned for assessment at post-test, no participants met DSM-IV criteria for Major Depressive Disorder. Similarly, at the 3-month follow-up point, no participants out of the four that returned for assessment met DSM-IV criteria for Major Depressive Disorder. This information obtained from the SCID suggests that all participants who returned for post-test and/or follow-up assessments showed reductions in depressive symptoms of a large enough magnitude such that they no longer rendered a formal DSM-IV diagnosis of Major Depressive Disorder.

In an attempt to further assess clinical significance, recovery rates for Major Depression were also investigated. Similar to cutoffs used in the Jacobson et al. (1996) study, those participants who can be considered “recovered” have scores of less than eight on the BDI-II at post-treatment. Because of the low sample size at post-test for this study, the last data point for each participant was investigated. For this sample, approximately one-third (35%) of completers can be classified by the BDI-II as “recovered” from Major Depressive Disorder attributable to BA, as compared to the 50–60% of participants considered “recovered” in the Jacobson et al. (1996) study. It may be important to note that only three of 17 participants (18%) could still be considered clinically depressed at post-treatment, according to a score of 20 or higher on the BDI-II.
Treatment Adherence

In order to be certain that the treatment delivered from the prescribed manual was in fact BA, treatment integrity data were collected on each participant who completed the study. Trained observers were provided with a modified version of the National Institute of Mental Health Collaborative Study Psychotherapy Rating Scale (CSPRS; Hollon et al., 1984) that included the steps in BA, along with a checklist of prescribed BA techniques and proscribed cognitive therapy techniques.

A random sample of the video taped treatment sessions (11%) was reviewed separately by two different observers (representing 53% of participants on whom treatment integrity was completed). Both observers checked off the presence of each step in the outline, along with the specific treatment interventions used that session. All therapists were included in the reviewed sample.

In order to determine where rater discrepancies appeared, checklist compliance percentages for the most relevant items was first computed. On 100% of the observed occasions, raters responded “yes” to the checklist item, “therapist implemented behavioral activation interventions.” Furthermore, on 100% of the observed occasions, raters responded “no” to the item, “therapist did use cognitive interventions,” indicating cognitive therapy was not implemented at any point throughout treatment in the observed sessions. This is key to treatment integrity as it strongly suggests that therapists in fact administered BA, and not cognitive therapy, to the participants.
Also important in the delivery of BA was the adherence to the prescribed treatment protocol in terms of completing all procedural components. On 94% of observed occasions, therapists asked participants to complete the BDI-II at the beginning of the session and then reviewed the questionnaire, examining overall scores and key items as that which endorsed suicidal ideation and participation in activity. Furthermore, therapists and participants reviewed the previous weeks’ homework on approximately 94% of the observed sessions and then presented homework to be completed over the upcoming week.

Finally, inter-rater reliability was performed on the final judgment, which examined the number of agreements divided by the number of agreements plus disagreements. The inter-rater reliability correlation was .88, showing that the raters agreed 88% of the time that therapists were delivering BA according to the protocol described in the treatment manual.
The purpose of the present study was to test the effectiveness of behavioral activation with medicated and unmedicated clients who realistically presented for treatment in a more "real-world" setting. The aim of this study was to conduct a quasi-replication of the basic findings noted in the Jacobson et al. (1996) study, which concluded that the BA component of CBT was as successful as the full treatment package in reducing depression and altering negative thinking and attributional styles. It was hypothesized that at the completion of therapy, participants in both the treatment and waitlist conditions would be significantly less depressed on both a self-report measure and a clinician rating of symptom severity.

**Main Outcomes**

The results of this study support BA as an efficacious therapeutic tool to treat major depression and suggest that activation strategies alone may be sufficient to produce behavioral and cognitive changes that significantly reduce depressive symptoms. Conclusions are based on between-group differences, which showed that though both treatment and waitlist conditions started out at comparable levels of symptom severity, the treatment group had a better response to BA than those in the waitlist group at post-treatment. These differences were maintained at the 3-month
follow-up according to the RHRSD. Essentially, both treatment and waitlist condition participants experienced a significant decrease in depressive symptoms, but with respect to a clinician-rated assessment, the treatment group showed a greater reduction in symptoms. These results should be interpreted with caution due to the low sample size in the waitlist group.

Further statistical comparisons between groups support the efficacy of BA. For instance, the comparison made between the end of the wait period for the waitlist group and post-treatment for the treatment group showed that end of treatment means were significantly lower than end of the wait period means. These results are especially meaningful in that they suggest that treatment with BA is superior to remaining on a waitlist and that it was not simply the passage of time or spontaneous remission that accounted for apparent decreased symptomatology in this sample.

The inclusion of the waitlist control group and their absence of change over a 6-week wait period in depression symptoms bolster this conclusion. Essentially, participants in the waitlist group showed no significant reduction in BDI-II scores over the course of the wait period, again suggesting that the passage of time was not sufficient to decrease depressive symptomatology. Moreover, as waitlist participants began treatment with BA, they subsequently experienced a reduction in symptomatology.

In sum, those in the treatment group showed a significant decrease in depression, as they experienced a shift from moderate/severe to mild depression (as measured by the BDI-II). Those who were in the waitlist control group, waiting for
treatment to begin, did not show improvement over the 6-week wait period. Put simply, for this sample, when treated with BA, people experienced a decline in depression symptoms. Without treatment, depression levels falling in the moderate/severe range remained relatively the same.

Given that the treatment and waitlist conditions experienced a significant reduction in depression when assessed separately, it makes intuitive sense that after combining the groups, BA still fared well. Overall, depression levels significantly decreased for those who received six or more sessions of BA. Furthermore, there were no significant differences between the end of treatment and 3-month follow-up scores on both outcome measures. More specifically, for this sample, even three months after treatment ended, significant reductions in depression were still evident. As noted by Gortner et al. (1998), this comparison is important as it allows for the determination of the “ultimate impact of therapy,” showing both acute and longer-term responses to treatment (p. 379). This is an important point to highlight given the greatest risk for relapse is during the immediate post-recovery period, typically around the first few months after symptoms remit (Keller, Lavori, Lewis, & Klerman, 1983; Maj, Veltro, Pirozzi, Lobrace, & Magliano, 1992).

Moreover, this sample as a whole scored in the moderate/severe range of depression ($M = 30.6$) at the initial screening, according to the BDI-II. After receiving at least six weeks of treatment, the post-treatment mean ($M = 8$) placed this group in the minimal clinical depression category. This finding is similar to the BA group in the Jacobson et al. (1996) study in which participants also moved from a
moderate/severe level of depression to mild clinical depression, as measured by the BDI-II. Furthermore, 15 out of 17 participants (88.3%) who entered this study initially described themselves as suffering from chronic, recurrent depression. This is not surprising given the risk for repeated episodes of depression is approximately 80% and isolated depressive episodes are rare (14%; Judd, 1997). This closer examination of symptom severity and chronicity is meaningful in showing that BA was not simply sustaining a group of people suffering from a mild or subclinical level depression, or primarily those experiencing their first single episode of depression. It had a considerable effect on a difficult to treat population, those primarily suffering from moderate to severe cyclical depression, quite possibly for a number of years.

Given these findings, one cannot dispute the clinical effectiveness of BA for the treatment of major depression. Questions as to its theory of change and the decision to use BA singly or in combination with CT, therefore need to be addressed. Dating back to 1984, treatment outcome researchers began to question the cognitive component of CBT for depression, pondering whether “changing beliefs necessarily solves the clinical problem” (Latimer and Sweet, 1984, p. 21). As Latimer and Sweet (1984) critically reviewed the evidence for the efficacy of procedures specific to behavioral and cognitive therapy, they inquired whether the cognitive piece of CBT was a direct mechanism of change, or if the clinical shift was due to the use of behavioral procedures of established efficacy. Based on their review of 11 studies, it was concluded that because CT usually involves a variety of methods including
behavioral procedures, it’s questionable whether the cognitive piece alone makes a significant contribution to therapeutic outcomes.

Over the next decade, this line of inquisition spawned much theoretical debate, along with a line of solid clinical research comparing combined cognitive-behavioral treatment packages. A further review of treatment outcome research, published from 1982 through to 1989 (Sweet & Loizeaux, 1991), examined 70 articles that addressed the essential question: “Does the specific addition of a purely cognitive therapeutic procedure enhance the outcome of behavioral treatment methods for actual patient populations?” In this comparative review, most studies reflected an equivalence in outcome between CBT procedures and behavioral procedures alone, therefore allowing the authors to conclude that the behavioral aspect of CBT was central to its effectiveness, while the same could not be said for the cognitive component. Given this conclusion, the authors ingeniously proposed the need for clinical researchers to dismantle CBT.

As noted by Beidel and Turner (1986), the relationship between behavior and cognitive change is “complex and interactive, with change in one domain promoting change in the other” (p. 188). These authors have suggested that we can get positive outcome from behavior therapy alone, without the cognitive component, because behavioral activities provide a mechanism through which distorted cognitions can be evaluated, thus rendering a subsequent cognitive shift. Furthermore, as patients are encouraged in CBT to evaluate their thoughts in a more critical light, it has been argued that what they are actually attending to is a more careful self-monitoring of
environmental contingencies, which fits nicely into a stimulus discrimination paradigm. As patients are encouraged to try new behaviors and monitor the consequences, cognitive change occurs as a result. This then describes CBT as not very different from the more traditionally oriented behavior therapy, as both look to change overt and covert maladaptive depressive behaviors (Biedel & Turner, 1986).

This investigation's ability to show that in and of itself, BA is an effective treatment for depression, supports these theoretical underpinnings and refutes what cognitive therapists have long said about the mechanisms of action in CBT for depression. First described in the Jacobson et al. (1996) study, it was suggested that BA can be just as effective as the full CBT treatment package because as reinforcers are returned to depressives' lives, BA functions to change the way people think more effectively than explicit cognitive interventions. This line of reasoning supports traditional behavioral theory, which stresses the importance of learning histories for subsequent variation in cognitive content, and helps explain why BA may stand alone as an efficacious treatment intervention for major depression.

Secondary Outcomes

In this investigation, comparisons were made post-hoc between persons who were and were not being treated with psychotropic drugs. This was done so as to clarify the role of BA in the treatment of more "real world" people who suffer from major depression. As noted by Burrows (1992), when patients present for therapeutic treatment for depression in a clinic setting, many are already taking psychotropic
medication, leaving psychotherapists to contend with existing medication regimes. Therefore, this study set out to simulate a typical clinical setting as much as possible, thus making this important post-hoc comparison.

The medication and no medication groups were statistically compared at pretreatment, post-treatment, and the 3-month follow-up. While within-group comparisons revealed both groups experienced significant reductions in depressive symptoms, neither group proved superior to the other with regard to both outcome measures. Essentially, the combination of preexisting medication and BA did not provide a clinically meaningful advantage over BA alone.

While preexisting medication did not seem to hinder the recovery of those in the medication and BA treatment group, it appears that medication also did not enhance positive treatment outcome. One might argue that because combining the two interventions did not exceed that of BA alone, it would be more cost-effective to use BA singly. As suggested by Antonuccio, Thomas, and Danton (1997), in an era of managed care, treatment must not only be effective, but also cost-effective. In a study conducted by these same authors in which acute and long-term outcomes and dropout/relapse rates were considered over a 2-year period, medication alone resulted in 33% higher expected costs than individual CBT treatment, while the combination of treatments resulted in 23% higher costs than CBT alone. Although only one study, it makes a strong argument that getting treated with therapy alone can be greatly cost-effective in comparison to treatment with medication alone or some combination of therapies. This cost-effective model bodes well for BA as a single therapeutic tool,
which proved for this sample to be efficacious for treating depression whether patients were taking antidepressant medication or not.

One might ask why two therapeutic interventions were not more effective in this investigation for reducing depression symptoms than a single approach. Not only has some preliminary evidence supported the fact that patients who improve with cognitive-behavioral therapy show similar biological changes than those who improve with psychotropic medication (Antonuccio, Danton, DeNelsky, Greenberg, & Gordon, 1999), but it is also possible that those who were prescribed a medication regime were not completely compliant. Non-adherence to antidepressant medication regimes is not uncommon, as the research suggests that 40–50% of patients prescribed an antidepressant will not take it for the maximum period needed to achieve therapeutic gains. Discontinuing the medication because of undesirable side effects is also common (Schulberg, Katon, Simon, & Rush, 1999). With this sample, it is possible that the medication participants who were reportedly taking their medication as prescribed had in fact discontinued it over the 10-week treatment period or were not taking it consistently. Similarly, it is possible that participants were not taking the therapeutic dosage (i.e., dosages were suboptimal) necessary to clinically reduce depression. Because participants’ medication compliance and dosage levels were not monitored by present investigators at any point during their involvement in the study, it is impossible to know exactly how compliant they were with their regimes.
Treatment outcome literature in depression, investigating whether medication has an additive effect to therapeutic treatment, has shown mixed results. While some studies suggest the combination of psychotropic medication and CBT are more efficacious than either alone (Hollon et al., 1991; Hollon et al., 1992), a number of other studies have found similar results to that of this investigation, arguing that medication does not add anything to the existing therapeutic tool. For instance, in a quasi-experimental study conducted by Oei and Yeoh (1999), it was hypothesized that pre-existing medication would enhance treatment outcome for those receiving group CBT for depression, as compared to those coming into the study not taking medication. After 12 sessions of group CBT over a 3-month period, the authors actually found the opposite of their proposed hypothesis. Results indicated that pre-existing medication did not enhance or detract from the positive treatment outcome that participants experienced. Although a group format was investigated here, results are in line with those of this investigation, which also concluded that concurrent drug intake did not produce an additive effect to the psychotherapy portion of treatment.

In conclusion, the results regarding the additive effect of medication and BA should be interpreted cautiously as the possible lack of uniformity in medication regimes and dosages may have biased the results. On the contrary, one cannot dismiss these findings as the goal of this study was not that of rigorous control, as in “efficacy” research, but to simulate real-world clinical samples, while also adding to the smaller body of “effectiveness” research literature. Further research is needed in this area to uphold these findings (Oei & Yeoh, 1999).
Limitations of This Study

There are a number of limitations of the current study that merit discussion. The most notable is the small sample size, due to large attrition rates and difficulty with recruitment. As noted by Persons, Burns, and Perloff (1988), it is expected that in controlled research settings, participant dropout rates are lower than those seen in private clinic settings for several reasons: participants agree to a fixed number of sessions at the start of treatment, the cost of treatment is typically substantially reduced, research subjects are likely to be high in motivation given that they must apply and go through rigorous screening procedures, and great efforts are made by researchers to follow up missed sessions. Despite similar circumstances in this investigation, the dropout rate remained relatively high (45%), although not unlike dropout rates seen in psychiatric community outpatient clinics, which typically range from 20–60% (Simons, Levine, Lustman, & Murphy, 1984), and other effectiveness studies that describe a dropout rate between 40–60% (Chambliss & Ollendick, 2001).

Throughout the literature, many reasons for dropping out of treatment prematurely have been noted. For instance, if a patient shows resistance to change or to the treatment process itself attrition rates are likely to increase (Davis & Addis, 1999). In this study the therapy itself required patients to learn, utilize and practice active coping skills on a daily basis. Therefore, high attrition in this study may have occurred because these participants showed lower levels of self-efficacy, thus discontinuing treatment because they could not “buy into the idea” that they were
capable of actively learning and utilizing helpful coping strategies (Davis & Addis, 1999, p. 347).

The high attrition rate in this study possibly could have also been due to participants’ holding discrepant etiological and treatment beliefs. Furthermore, patients may have discontinued treatment if they felt a relief from depressive symptoms, thus assuming treatment was no longer warranted. Family and friends can also create an environment that fails to support a patient’s response to treatment, which increases the risk of patient dropout. Many other process variables, such as the patient’s perceptions of the therapeutic relationship and sociodemographic variables, have been shown to affect premature dropout. For instance, research shows that minorities, patients who are younger, and those who are single/living alone are more likely to drop out of treatment (Organista, Munoz, & Gonzalez, 1994). Davis and Addis (1999) report that attrition studies consistently underscore the importance of such process variables that illustrate the process by which attrition occurs. A better understanding of such factors may clarify why participants prematurely discontinue treatment.

Because a clear majority of dropouts in the current study did not give particular reasons for termination and were unable to be contacted, it is impossible to know exactly why each person prematurely ended treatment. It may be important to note that some studies show that increased depressive psychopathology is correlated with higher levels of attrition (Farmer, Locke, Liu, & Moscicki, 1994; Murphy, Carney, Knesevich, Wetzel, & Whitworth, 1995). But in this investigation, increased
psychopathology did not appear to be a causal factor in premature termination, as those who dropped out were not significantly more depressed than those who completed the study, according to initial pretreatment BDI-II and RHRSD scores.

A number of preventative interventions that target attrition have been cited in the literature as decreasing the risk of dropout. For instance, Brent, Holder, Kolko, Birmaher, Baugher, Roth, Iyengar, & Johnson (1997), report that dropouts have been shown to be more hopeless than those who continue in treatment. Therefore one’s hopelessness about the course of treatment could be targeted early on in order to enhance compliance. Also helpful may be to address attrition directly with the participant from the beginning of treatment, while making sure the therapist/participant agree on the conceptualization of his/her problems and the goals and tasks of therapy. Psychotherapy studies report a threefold increase in attrition rates when therapists ignore these issues (Epperson, Bushway, & Warman, 1983).

Because of high rates of withdrawal, in conjunction with the lack of further post-test and follow-up data in this study, the generalizability and external validity of the results were compromised. Even after collapsing the two treatment groups, the sample size remained low, which may have had an affect on the power of the study to detect real differences if they existed. Also limiting this investigations’ generalizability may have been the restrictive exclusion criteria employed during recruitment, which in turn led to studying a narrowly defined population. Although common practice in clinical trials, this limits our ability to generalize our findings to the full range of people with depression who seek treatment in a typical service.
setting. Though it would be impossible to establish the efficacy of depression treatment in every conceivable subgroup of depressed individuals, our focus on a small subset of people with "pure" depression accents how little practicing clinicians know about comprehensive groups of depressed people (Zimmerman, Mattia, & Posternak, 2002). Because the presence of simultaneous psychiatric disorders can greatly modify treatment outcome, it has been repeatedly suggested that researchers go beyond the study of "pure" depression and broaden their samples to include people with comorbid diagnoses, such as anxiety and personality disorders, and suicidal patients (Nezu, Nezu, Trunzo, & McClure, 1998).

Future Investigation and Conclusion

One direction for future BA research may include the obvious move to broaden the range of patients and settings in which BA appears to be effective. For instance, testing the effectiveness of BA in other "real-world" and naturalistic settings (e.g., private practice), with less stringent inclusion criteria (e.g., include those exhibiting suicidal behaviors and/or other comorbid Axis I and II disorders) could potentially increase the generalizability of the results.

Along similar lines, future research might test out BA with those suffering from a more severe and chronic depression. While all but two participants in this study reported their depression to be recurrent, depression levels fell on the cusp between moderate and severe, according to the BDI-II. Future studies might consider a sample in which BDI-II scores average 30 and higher, placing them in the severe
category of symptomatology. The severity and chronicity of this disorder has widespread community implications as both have been correlated with a higher financial burden to our national community. Research suggests that the more severe and chronic one’s depression, the more days he or she will lose at work, thus decreasing overall work productivity (Lecrubier 2001). Given this evidence, it seems imperative that future BA investigations are geared toward preventing and treating those suffering from a more severe and chronic depression.

Because of the aforementioned relapse rates amongst this group, future investigations might also employ a parametric research strategy, such as exploring a trial of BA that includes a specified number of intermittent booster sessions. For example, Nezu et al. (1998) suggests that for an individual with a history of symptom recurrence, semiannual “depression check-ups” that focus on relapse prevention techniques may be useful to maintain treatment gains (p. 509).

While it’s important to know whether a clinically significant reduction in symptoms has occurred, it is also of central clinical significance to determine whether the patient’s quality of life (e.g., health status, functional performance, life satisfaction, standard of living, etc.) has improved (Gladis, Gosch, Dishuk, & Crits-Christoph, 1999). Thus, in order to expand our view of clinical significance, future investigation may use other assessment tools that assess a combination of symptoms, level of functioning, but also quality of life. More recent instruments, developed specifically as quality of life measures for depressed patients, such as the Quality of Life in Depression Scale (Hunt & McKenna, 1992) and the SmithKline Beecham
Quality of Life Scale (Stoker, Dunbar, & Beaumont, 1992) may be employed along with more traditional measures as the BDI-II and RHRSD. As noted by Gladis et al. (1999), a therapeutic intervention is only fully evaluated when its assessment tools expand beyond symptom severity and the researcher documents its wide effects on all domains.

The inclusion of quality of life assessments also has implications in the area of managed care, as providers are developing their own parameters for mental health that include a wide array of criterion, many of which bear on the quality of life. Moreover, managed care providers frequently determine the length and type of treatment under the quality of life umbrella, which includes such indicators as life satisfaction, social relationships, and work performance. In sum, the use of quality of life measures can be viewed as a necessary adjunct to statistical approaches of defining clinical significance and meaningful change (Jacobson & Traux, 1991).

In order to determine which specific BA interventions are most “necessary, sufficient, and facilitative” of therapeutic change (Kazdin, 1992, p. 142), a final direction may be to conduct an investigation that dismantles BA. This could assist clinicians in the identification of BA techniques that are more “user-friendly” for both the patient and therapist. Moreover, such fine-tuning of BA may allow researchers and practitioners to understand the fundamental mechanisms of action in regard to decreasing overall depressive symptomatology.

Finally, it is important to acknowledge that BA may not be an effective and preferred therapeutic tool for every person suffering from depression. For instance, it
has been suggested by Addis & Jacobson (1996) that patients who show a strong tendency to search for the causes of their depression are less likely to experience a reduction in depressive symptoms with BA treatment. The examination of such causes may ultimately inhibit and prevent attempts at making meaningful behavior changes, thus maintaining one’s depression. Because each depressed person shows great variability in personal characteristics such as life circumstances, symptoms, developmental history and biological makeup, to suggest BA as the ultimate tool capable of solving society’s depression epidemic would be to ignore individual variability (Nezu, 1987).

In conclusion, the results of this investigation affirms that BA shows great promise as an effective treatment strategy for both medicated and unmedicated individuals suffering from Major Depressive Disorder. It has been proposed that one may not need to implement the full CBT treatment package in order to see significant reductions in symptoms, making BA a sufficient and cost-effective therapeutic tool.
Appendix A

HSIRB Approval Letter
Insert letter here
Appendix B

Treatment Integrity Checklist
### TREATMENT INTEGRITY CHECKLIST

<table>
<thead>
<tr>
<th>Subject Number</th>
<th>Session Number</th>
<th>Therapist</th>
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</table>

1. Client filled out the BDI-II—beginning of session

2. Items on BDI-II were reviewed

3. Therapist & Client set agenda

4. Therapist & Client discussed agenda items

5. Therapist & Client discussed homework (from last week)

6. Therapist implemented *Behavioral Activation* interventions
   - Functional Assessment
   - Mastery & Pleasure Ratings of Activities
   - Daily Activity Schedule Review
   - Assigning Activities to Increase Sense of Mastery or Pleasure
   - Explained ABC Model to Client
   - Encouraged an Active Rather than Passive Approach
   - Graded Task Assignments
   - Mental Rehearsal of Assigned Tasks/Activities
   - Examined Alternative Behaviors in Different Situations
   - Role-Playing Behavioral Assignments
   - Maximizing the Likelihood of Homework Success
   - Distraction from Problems or Unpleasant Events
   - Avoiding or Limiting Exposure to Unpleasant Situations/People
   - Direct Behavioral Instruction
   - Taught Client to Give Themselves Rewards
   - Dealt with Specific Behavior Problems (e.g., sleep)
   - Training-Social Skills Deficits

7. Therapist *did use* Cognitive Interventions
   - Attempted to Change Cognitive Distortions
   - Addressed Automatic/Dysfunctional Thoughts
   - Instructed Client to Record Automatic/Dysfunctional Thoughts
   - Reality Testing of Automatic/Dysfunctional Thoughts

8. Therapist *did not use* Cognitive Interventions

9. Therapist/Client discussed *following week* homework
Appendix C

Initial Recruitment Telephone Script
RECRUITMENT SCRIPT

“The Clinical Researchers at Western Michigan University are conducting a study to assess the effectiveness of a psychological treatment for depression. The treatment is designed to break the cycle of your depression and past research has shown this therapy to be very effective with adults. Treatment would take place once a week for 10 consecutive weeks and sessions will last approximately 60 minutes.

If you are experiencing problems with depression, you are encouraged to schedule an appointment for an intake interview to determine if you qualify for acceptance into the study. Participation in an interview in no way obligates you to participate in the study, but it serves to give both you and the researchers a better understanding of how appropriate the therapy would be for you. If you are accepted into the study, you would be expected to complete the 10-week course of treatment that would require approximately 60 minutes of your time each week. In addition, you would be expected to perform certain tasks between therapy sessions that are aimed at relieving your depression.

Would you like me to go ahead and proceed? I have a few questions I would now like to ask you?

1. "Are you currently taking any medication for psychological problems?"
   If yes, "what?"

2. If yes, explain: "In order to participate, we ask that you are stabilized on your medication, meaning you must have been taking it for at least 6 weeks. When did you initially start taking your medication?" DATE_________ Proceed to Question #3.

3. "Are you currently participating in any sort of therapy treatment for any psychological conditions?"
   If yes, explain: "In order to participate, we ask that you not be in another concurrent psychosocial treatment." Give referrals.

   If no, ask participants: “Would you like to come in for an intake?”

   • If yes, explain: “I will pass your name and phone number along to my assessor and he/she will call you within the next few days to schedule your intake appointment.”
Appendix D

Consent Form
Agreement to Participate in Research

Principal Investigator: C. Richard Spates, Ph.D.
Research Associate: Jenifer M. Cullen, M.A.

I, __________ have been invited to participate in a research project entitled "Testing the Effectiveness of Behavioral Activation Therapy in Acute Treatment of Unipolar Depression". I have been told that this research is intended to measure the efficacy of Behavioral Activation Treatment as a therapeutic tool to treat individuals suffering from Unipolar Depression. I have been told that this project is Jenifer M. Cullen's dissertation project.

My consent to participate in this project indicates that at the initial intake interview I will be asked to complete one 10-minute multiple choice inventory, along with a brief 5-minute personal information survey. In addition, I will be given two psychological interviews that should take no longer than one hour to administer. Only those persons who qualify for a diagnosis of Unipolar Depression and who are not enrolled in some other concurrent psychosocial therapy will be eligible to participate. Any other diagnoses will exclude me from the study.

If I am eligible for the study, I will be randomly assigned to one of two treatment conditions. In one condition, I will immediately begin Behavioral Activation treatment for depression. In the second condition, Behavioral Activation treatment will be delayed for approximately six weeks. At the end of the six-week period, I will again be tested with a 10-minute psychological questionnaire and given a 10-minute psychological interview. I will begin treatment immediately thereafter. Both groups will receive treatment for 10 weeks and will be tested weekly with the same 10-minute multiple choice survey. At the end of the 10-week Behavioral Activation treatment I will again be asked to complete the 10-minute multiple choice survey and then be interviewed with the two psychological interviews given previously. I will also be asked to complete a 5-minute Client Satisfaction Survey. I have also been told that three months after treatment has ended, I will be asked to return to the clinic to complete the same 10-minute multiple choice survey and be interviewed a last time with the two psychological interviews already given. It has been explained to me that there is a possibility that treatment sessions may be videotaped to assure therapy is assuredly executed. Videotapes will be stored in a locked file drawer and only project personnel will have access to those tapes.

As in all research, there may be unforeseen risks to the participant. If an accidental injury occurs, appropriate emergency measures will be taken; however, no compensation or treatment will be made available to me except as otherwise specified in this consent form. I realize that one potential risk of my participation in this project is that I may experience unpleasant emotions, including anger, frustration, depression, and disappointment, as I recall my problems and experiences and actively work to change certain behaviors in order to reduce my depression. I have been told

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that the Psychology Clinic is prepared to make a referral should emergency care become necessary. I will be responsible for the cost of emergency care should such care become necessary.

One way in which I may benefit from this study is that, as a result of receiving treatment, I may experience a reduction or elimination of my depressive symptoms. In addition, I may benefit from the knowledge that is gained by this research, as can others in the community who suffer from Unipolar Depression.

I have been told that all research information collected from me will be kept confidential. This means that my name will not appear on any research questionnaires I complete or on any other research forms that contain personal information I have provided. I further understand that these forms will be kept in a research folder in a locked file cabinet in this clinic during my participation in the study. I also have been told that the policy of this clinic requires that progress notes and further information about me be recorded and placed in a treatment folder. This is necessary because by participating in this study, I am also a client of this clinic. However, I realize that the information in my treatment folder belongs to the clinic and may not be used as data for this study. I have been told that forms used in this study may be duplicated and placed in my treatment folder where they will be retained until they are destroyed along with the rest of the papers in my treatment folder according to the policies of the clinic. At the end of my participation in the study my research folder will be moved to a locked cabinet in the Department of Psychology where it will be stored for at least three years after the completion of this study. It will then be destroyed. I have been told that Jenifer Cullen will keep a separate master list with the names and research code numbers of participants from this clinic. The master list will be the only link between the data on the recording forms and my identity. The master list will be destroyed once all data has been collected and analyzed.

I have been advised that I may refuse to participate or quit at any time during the study without prejudice or penalty or effect on my relationship with Western Michigan University. I am aware of alternative treatment services should this be the case. If I have any questions or concerns about this study, I may contact either Dr. Richard Spates at (616) 387-4329 or Jenifer Cullen at (616) 553-9836. If I have questions about my rights as a research participant or about any other aspects of my participation, I also may contact the Chair of the Human Subjects Institutional Review Board at (616) 387-8239 or the Vice President for Research at (616) 387-8293 with any concerns that I have. This consent document has been approved for use for one year by the Human Subjects Institutional Review Board (HSIRB) as indicated by the stamped date and signature of the board chair in the upper right corner of all pages. My signature below indicates that the purpose and requirements of the study have been made clear and that I agree to participate.

Signature _______________________________________________________

Date ____________________________________________________________
Appendix E

Demographics Questionnaire
DEMographics QUESTIONNAIRE

Research Code Number: _______  Age: _______  DOB: _______
Address: ________________________________
________________________________________

Emergency Contact Telephone # ________________

Gender: (Circle one)  Male  Female

Ethnicity: (Mark best choice)
  • African American ______
  • American Indian ______
  • Asian American ______
  • Alaskan American ______
  • Caucasian (white) ______
  • Hispanic ______
  • International/non US resident ______
  • Multiracial ______
  • Other (please specify): ________________________________

Relationship Status: (Mark best choice)
  • Single, never married ______
  • Living with significant other ______
  • Separated ______
  • Divorced ______
  • Married ______

Years of Education: (Mark best choice)
  • Less than 12 years ______
  • 12 years or GED ______
  • More than 12 and less than 16 years ______
  • 16 years ______
  • 16+ years ______

Household Income: (Mark best choice)
  • Under $10,000 per year ______
  • $10,000-$20,000 per year ______
  • $20,000-$30,000 per year ______
  • Over $30,000 per year ______
Are you currently receiving treatment for depression?
• Yes ______  
• No ______  
• If yes, what type of treatment? (Mark all that apply)
  • Medication Treatment ______  
  • Hospital (Inpatient or Partial Hospitalization) Care ______  
  • Pastoral Care ______  
  • Individual Therapy ______  
  • Group Therapy ______  
  • Support Group ______

Have you been in treatment for depression in the past?
• Yes ______  
• No ______  
• If yes, how many episodes of treatment have you been through? ______

Are you currently taking prescription medication(s) for depression?
• Yes ______  
• No ______  
• If yes, what medication(s) are you taking and what is the dosage?

Have you taken prescription medication(s) for depression in the past?
• Yes ______  
• No ______  
• If yes, what medication(s)? ________________________________

Are you currently in treatment for any psychological condition(s) other than depression?
• Yes (please specify) ________________________________  
• No ______

Have you been treated for any psychological conditions(s) other than depression in the past?
• Yes (please specify) ________________________________  
• No ______

Current Stressors:
_________________________________________________________  
_________________________________________________________  
_________________________________________________________

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Appendix F

Beck Depression Inventory—Second Edition
The Beck Depression Inventory-Second Edition is copyrighted by Aaron T. Beck, 1996. Persons interested in obtaining information regarding this instrument should contact The Psychological Corporation, 555 Academic Court, San Antonio, Texas 78204-2498.
Appendix G

Revised Hamilton Rating Scale for Depression
The Revised Hamilton Rating Scale for Depression is copyrighted by W. L. Warren, 1994. Persons interested in obtaining information regarding this instrument should contact Western Psychological Services, 12031 Wilshire, Boulevard, Los Angeles, California 90025-1251.
Appendix H

Master List
# Master List of Subjects

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Appendix I

Universal Data Collection Form
Universal Data Collection Form

Assessment Information for Participant # __________________

Intake Interview:
- BDI-II Score ________
- RHRSD Score ________
- SCID DX:
  - Axis I __________________________
  - Axis II __________________________
  - Axis III __________________________
  - Axis IV __________________________
  - GAF Score ________________________

BDI-II scores for WAIT LIST CONDITION ONLY during 6-week wait period
- BDI 1 ______
- BDI 2 ______
- BDI 3 ______

BDI-II scores during 10-week treatment:
- Week 1 ______
- Week 2 ______
- Week 3 ______
- Week 4 ______
- Week 5 ______
- Week 6 ______
- Week 7 ______
- Week 8 ______
- Week 9 ______
- Week 10 ______
Post-treatment:
- BDI-II ______
- RHRSD ______
- SCID DX:
  - Axis I ______________________________
  - Axis II ______________________________
  - Axis III ______________________________
  - Axis IV ______________________________
  - GAF Score ____________________________

3 Month Follow-Up:
- BDI-II ______
- RHRSD ______
- SCID DX:
  - Axis I ______________________________
  - Axis II ______________________________
  - Axis III ______________________________
  - Axis IV ______________________________
  - GAF Score ____________________________
  - All data collection Complete: Initials _____ Date _____
BIBLIOGRAPHY


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