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TREATMENT VALIDITY OF THE DEXAMETHASONE SUPPRESSION TEST

The University of North Carolina at Greensboro PH.D. 1986

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TREATMENT VALIDITY OF THE DEXAMETHASONE SUPPRESSION TEST

by

Dennis L. McKnight

A Dissertation submitted to
the Faculty of the Graduate School at
The University of North Carolina at Greensboro
in Partial Fulfillment
of the Requirements for the Degree
Doctor of Philosophy

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Approved by

Rosemary O. Nelson

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APPROVAL PAGE

This dissertation has been approved by the following committee of the Faculty of the Graduate School at the University of North Carolina at Greensboro.

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McKNIGHT, DENNIS L. Treatment Validity of the Dexamethasone Suppression Test. (1986)
Directed by: Dr. Rosemery O. Nelson. Pp. 228.

This dissertation examined the value of the dexamethasone suppression test in selecting subjects who are responsive to different types of treatment for depression, thereby testing the "treatment validity" of the dexamethasone suppression test. In addition, the effects of a biologically-oriented treatment and a psychologically-oriented treatment on the dexamethasone suppression test and on subjects' dysfuntional thoughts was examined.

Forty-three outpatient subjects diagnosed with Major Depression along with a high level of dysfunctional thoughts participated in this project. Subjects were identified as either abnormal or normal dexamethasone suppression test responders according to cortisol blood levels, and then randomly assigned to receive either a biologically-oriented treatment (tricyclic antidepressant medication) or a psychologically-oriented treatment (cognitive therapy). Self-report depression measures, diagnoses, the questionnaire measuring dysfunctional thoughts, and the dexamethasone suppression test were administered at pre-intervention and post-intervention and were subjected to statistical analyses.

In short, the results showed that for both treatments, subjects overall reported significantly less depressive symptoms according to global measures of depression from the Depression Scale of the Minnesota Multiphasic Personality Inventory, the Beck Depression Inventory, the Lubin Depression Adjective Checklist, and diagnoses based on the

Diagnostic and Statistical Manual of Mental Disorders, third edition. These measures also showed that normal dexamethasone suppression test responders reported a significantly greater change (improvement) in depressive symptoms at post-intervention than abnormal dexamethasone suppression test responders, regardless of type of treatment. Furthermore, while normal dexamethasone suppression test responders showed no significant change on the dexamethasone suppression test from pre-intervention to post-intervention, the abnormal dexamethasone suppression test responders did show significant reductions in the dexamethasone suppression test (indicating improvement) from pre-intervention to post-intervention after receiving either antidepressant medication or cognitive therapy, with no difference at post-intervention between the two types of treatment. In addition, there was a significant reduction overall in depressives' dysfunctional thoughts (according to the Personal Beliefs Inventory) after receiving either treatment, with subjects receiving cognitive therapy having significantly fewer dysfunctional thoughts than subjects receiving antidepressant medication.

These findings are examined in detail and interpretations discussed.

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The author recognizes with appreciation the anonymous subjects without whom this research would have been impossible. It is the author's hope that they somehow benefited from this project.

The author expresses sincere gratitude to his family, who provided endless support and caring throughout my undergraduate and graduate career.

Finally, the author dedicates this dissertation to his wife, Jill, for her never-ending emotional support, sacrifice, unlimited patience and understanding, and her many, many hours of hard work and flawless typing from the beginning of this project to its completion.

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CHAPTER I

INTRODUCTION

Depression, perhaps more than any other psychological disorder, has resulted in inordinate amounts of human suffering, loss of productivity, and even loss of life. The President's Commission on Mental Health (Task Panel reports submitted to the President's Commission on Mental Health, Volume IV, Appendix, 1978; cited by Craighead, 1979) estimates that approximately twenty percent (20%) of Americans will experience an affective disorder during their life, rivaling schizophrenia as the nation's number one mental health problem. On an international scale, Teuting, Kaslow, and Hirschfeld (1981) estimate that over 127 million of the world's people suffer from depression, while approximately 8 to 20 million Americans currently suffer from depression. In this same report, Teuting et al. (1981) estimate that treatment for depression costs over $10 billion each year, in addition to the "human cost" of depression in terms of discomfort, divorce, suicide, alcohol and drug abuse, dysfunctional family life, unemployment, and child abuse. Questions such as the following have occupied investigators' interest since this clinical syndrome was first identified by Hippocrates in the fourth century B.C. (Boyd & Levis, 1980). What is depression? What causes this disorder, and what factors are responsible for maintaining it over time?

Researchers have approached the above questions from different vantage points. These vantage points, broadly defined, involve the

biological, the psychodynamic, and the social approaches (behavioral and cognitive) to the etiology and treatment of affective disorders.

While it is recognized that affective disorders are a serious and complicated area of study that can be approached from a variety of theoretical viewpoints, this dissertation limited its focus to the biological and cognitive assessment and treatment of nonbipolar, nonpsychotic depression. Specifically, this study examined the "treatment validity" of the dexamethasone suppression test by testing whether the treatment of abnormal or normal dexamethasone suppression test responders was maximized by using either a biologically-oriented treatment (antidepressant medication) or a psychologically-oriented treatment (cognitive therapy), respectively. Questions addressed in this dissertation were as follows: Does an abnormal dexamethasone suppression test, which many believe represents a biologically-based depression, respond better to a biologically-oriented treatment (antidepressant medication) versus a psychologically-oriented treatment (cognitive therapy); and, conversely, does a normal dexamethasone suppression test, which many believe represents a psychologically-based depression, respond better to a psychologically-oriented treatment versus a biologically-oriented treatment? Related to this question, the theoretical distinction of endogenous versus exogenous depression was explored in terms of its function in predicting behaviors associated with the distinction (presence or absence of melancholia) and its function in predicting treatment response. Another question posed was if a conversion from an abnormal to normal dexamethasone suppression test after somatic treatment indicates clinical recovery, what effect

does a psychological treatment (cognitive therapy) have on the dexamethasone suppression test? Finally, what effect does a biologically-oriented treatment and a psychologically-oriented treatment have on a subject's dysfunctional thoughts?

The context in which these questions are posed requires review. First, the affective disorder of depression is defined, and theoretical distinctions are discussed. Next, a brief review of biological approaches and the cognitive approach to depression are examined, specifically focusing on effective treatments for depressives: somatic treatments (e.g., chemotherapy) and Beck's cognitive therapy. Next, the dexamethasone suppression test is discussed as it relates to the etiology and treatment of depressive disorders. After this, relevant philosophical/conceptual issues are briefly mentioned. Finally, the concept and methodology of "treatment validity" is described.

Depression: Definition and Theoretical Distinctions

According to the Diagnostic and Statistical Manual of Mental Disorders, third edition (American Psychiatric Association, 1980), the "affective disorders" are divided into two main groups, Major Affective Disorders and Other Specific Affective Disorders. This dissertation was concerned with the group Major Affective Disorders, which are subdivided into Bipolar Disorder and Major Depression. Theses two subcategories are distinguished on the basis of whether or not a manic episode has ever occurred in the patient. A client is diagnosed as having bipolar disorder if he/she is currently experiencing or has ever experienced a manic episode. A manic episode is defined by the Diagnostic and

Statistical Manual of Mental Disorders, third edition (American Psychiatric Association, 1980) as having a duration of at least one week and including at least three of the following symptoms: increased activity, pressured speech, flight of ideas, exaggerated self-esteem, decreased need for sleep, distractibility, and excessive involvement in activities which have the potential for negative consequences (e.g., buying sprees, reckless driving). Therefore, a person may be currently experiencing a depressive episode, but would be diagnosed as "bipolar disorder-depressed" if the person had a history of manic symptoms. This dissertation excluded subjects that had a diagnosis of bipolar disorder since presently there are no data to suggest that clients with bipolar disorder respond to psychotherapy equally well or better than they respond to lithium or antidepressant medication (Rush, 1982). Therefore, subjects included in this dissertation fit the diagnosis of major depression, and included "individuals who have never experienced, or are unlikely to experience a manic episode" (Hollon, 1981).

According to the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1980), a major depressive episode is characterized by "a dysphoric mood or loss of interest or pleasure in all or almost all usual activities and pastimes." The dysphoric mood is characterized by symptoms such as the following: "sad, depressed, blue, hopeless, low, down in the dumps, irritable." In addition, at least four of the following symptoms have to be present nearly every day for a period of at least two weeks: poor appetite, significant weight loss, insomnia or hypersomnia, psychomotor retardation or agitation, loss of interest or pleasure in usual

activities, decrease in sexual drive, loss of energy, feelings of worthlessness or excessive guilt, inability to concentrate, or thoughts of death, suicidal ideation, or suicide attempt. A current episode of major depression may also be subclassified as with or without melancholia. With melancholia would include the following symptom cluster: loss of pleasure in all or almost all activities, lack of reactivity to usually pleasurable stimuli, and at least three of the following: distinct quality of depressed mood that is different from the feeling associated with the loss of a loved one, the depression is worst in the morning, early morning awakening, marked psychomotor retardation or agitation, significant anorexia or weight loss, or excessive or inappropriate guilt. Subjects in this research project were subclassified as with or without melancholia.

In summary, subjects included in this study were diagnosed as major depression, with or without melancholia. Subjects who exhibited any other psychiatric disorder, including bipolar disorder (manic or depressed) or any type of psychosis (impairment in reality testing) were excluded from this research project.

Additional Theoretical Distinctions

Attempts have been made to classify a major depressive episode in addition to the Diagnostic and Statistical Manual (American Psychiatric Association, 1980). These attempts vary extensively depending on how one chooses to view depression. Currently used categories have classified this syndrome according to whether the cause is internal or external (endogenous versus exogenous), the nature of the response

pattern (autonomous versus reactive), the level of anxiety (agitated versus retarded), the occurrence of manic episodes (unipolar versus bipolar), the level of reality testing (psychotic versus neurotic), and whether the depression is the main difficulty or if it is resulting from another disorder (primary versus secondary) (Boyd & Levis, 1980). Subtypes of depression have also been classified along personality dimensions such as passive-dependent, obsessive, hysteriod, paranoid, schizoid, bipolar-manic, and schizo-affective (Becker, 1977).

This dissertation was concerned with the theoretical distinction of endogenous versus exogenous depression because of its close association with the dexamethasone suppression test. Therefore, a review of this distinction is warranted.

The Endogenous-Exogenous Distinction

The endogenous-exogenous distinction is one of the most widely used but highly controversial distinctions in depression. This distinction has been conceptualized in terms of the behaviors associated with each category, the etiology that is implied by each category, and the response to treatment based on each category.

Endogenous depressions, based on factor analytic studies, have consistently been found to have a set of characteristic behaviors or symptoms including terminal sleep disturbance, weight loss, difficulty concentrating, psychomotor retardation, severely depressed mood, and an inability to respond to pleasant changes in the situation or environment (Rosenthal & Klerman, 1966). Exogenous depression is often characterized by behaviors or symptoms including low self-esteem,

feelings of helplessness, irritability, anger, unhappiness, a histronic attitude, self-pity, responsivity of mood, sudden onset, initial insomnia, and precipitating factors (Klein, 1974). In other words, endogenous depressions involve depressions with primarily negatative disturbances, while exogenous depressions involve more of an emphasis on subjective feelings of depression.

Based on the above symptom clusters, different etiologies for endogenous versus exogenous depression have often been inferred. As Sachar (1982) points out, the distinction was based on the following observation: the clinical syndrome of endogenous depression was associated with several symptoms suggesting hypothalamic dysfunction, such as disturbances in mood, sex drive, sleep, appetite, and autonomic activity. He goes on to state that if endogenous depression is associated with hypothalamic dysfunction, then it is possible that neuroendocrine function would also be disturbed. In addition, the same neurotransmitters implicated in the chemical pathology of depressive illness (e.g., noradrenaline, serotenin, and acetylcholine) also regulate the secretion of the hypothalamic neuroendocrine cells which control pituitary function. Therefore, deficiencies in the functional activity of these neurotransmitters would be expected to be reflected in the hormonal responses they regulate. This has been supported by studies showing significant abnormalities in the secretion of cortisol, growth hormone (HGH), and thyroid stimulating hormone (TSH) in endogenous depressions (Brown, Johnston & Mayfield, 1979; Checkley, 1979; Gold, Goodwin, & Wehr, 1976; Maeda, Kato, & Ohgo, 1975; Prange, Wilson, & Lara, 1972; Sachar, 1975; Van Praag & Korf, 1979; Whybrow &

Hurwitz, 1976). In addition, studies examining amine metabolite abnormalities (e.g., MHPG, HVA, and 5HIAA) have also supported this distinction (Asberg, Thoren, & Traskman, 1976; Goodwin & Potter, 1979; McLeod & McLeod, 1972; Post & Goodwin, 1974; Subrahmanyan, 1975). These data led many to view endogenous depression as a depression that was caused from "within," while exogenous depression was believed to be caused from "without." Since it is so difficult to determine whether a depression is caused by some precipitating event, many researchers simply define endogenous depression phenomenologically, that is, on the basis of symptomatology. In fact, Klein (1974) has proposed that the term "endogenomorphic" depression be used instead, while the Diagnostic and Statistical Manual (American Psychiatric Association, 1980) uses the term "with melancholia" to refer to endogenous depressions. Nevertheless, these types of depressions are often viewed as having some type of faulty biological system (e.g., pleasure mechanism) whether a precipitating event is evident or not (Klein, 1974).

Finally, the response to treatment has also been examined based on this endogenous-exogenous distinction. As Andreasen (1982) points out: "the clinical and neurochemical or neuroendocrine findings are fairly strong in the support of such a distinction, and the endogenous syndrome appears to be a healthy predictor of response to treatments such as tricyclics or ECT." Several studies have examined the effectiveness of a chemotheraputic approach (e.g., tricyclic antidepressants) using this distinction, finding that the endogenous depressions usually respond well to antidepressants (Bielski & Friedel, 1976;, Kiloh, Ball, & Garside, 1962; Paykel, 1972; Rao & Coppen, 1979; Raskin & Crook, 1976),

as compared to the exogenous depressions. These treatment studies have also been used to support the biochemical abnormalities in the etiology of endogenous depressions. The relation of this distinction to the dexamethasone suppression test, in addition to relevant philosophical/ conceptual issues, are discussed later in this introduction. The cognitive model of depression along with the biological-disease model are briefly reviewed next, in addition to the relevant treatment associated with each model.

Cognitive Model of Depression: Beck

The theory relating depression to cognitive variables was proposed by Beck and his associates (1967, 1972, 1979). Beck maintains that negative thought patterns developed during childhood and adolescence can be later activated by environmental stresses, which makes the individual susceptible to depression. Beck conceptualizes a "primary triad" which consists of the distorted perceptions that the depressed person holds about himself/herself, the present, and the future. These distortions occur when the person commits the following logical errors despite disconfirming evidence: (a) arbitrary inference - the person draws conclusions which cannot be supported by environmental data; (b) selective abstraction - the person emphasizes some details and not others; (c) overgeneralization - the person draws conclusions about his or her ability, performance, or worth on the basis of a single incident; (d) magnification/minimization - the individual exaggerates or slights the importance of events; (e) all-or-none thinking - the person thinks in absolute terms. Beck goes on to say that the classic emotional,

motivational, behavioral, and vegetative signs of depressions follow these distorted, negative, logical errors in thought. Beck states that the relationship between depressive symptoms and negative thoughts results in a "vicious circle causing the downward spiral of depression."

Over the years, there has been considerable accumulation of empirical data that supports the cognitive theory of depression. Several review articles have provided summaries of the data available (Hollon & Beck, 1979; Lewinsohn & Hoberman, 1981; Wright & Beck, 1983). Lewinsohn and Hoberman (1981) organized the data in terms of looking at the effects of success and failure, perceptual distortion, and negative experiences in depressed individuals.

Lewinsohn and Hoberman (1981) report on a number of studies that focused on the differential effects of success and failure experiences on self-esteem, mood, and expectation of future success on task-and-performance evaluation in depressed and nondepressed groups. For example, Filippo and Lewinsohn (1971) found that when "success" and "failure" were experimentally produced by either manipulating the time allowed for completing the task or by manipulating the length of the task, that systematic change in the depressed individual's self-esteem occurred. In addition, other relevant studies have also suggested that increases in depressive's expectations after successful performance were related to subsequent improved performance (Klein & Seligman, 1976; Loeb, Beck, & Diggory, 1971).

With regard to perceptual distortion, depressed subjects have been found to overestimate the amount of negative feedback and underestimate the amount of positive feedback they receive after performing

experimental tasks (DeMonbreun & Craighead, 1977; Nelson & Craighead, 1977). In addition, depressed subjects have difficulties in assessing the accuracy of feedback. For example, Hammen and Krantz (1976) and Rizley (1978) found that in experiments in which feedback was not related to actual performance, depressed subjects readily accepted the validity of false negative feedback and attributed their "failure" to personal inadequacies such as lack of effort or poor ability. Subjects who were not depressed attributed successful performance to their own personal abilities, even though this success was due to luck. These studies have been used in support of a cognitive theory of depression.

Studies examining memory distortion have also shown findings relevant to the cognitive theory of depression. While studies have shown that depressed subjects have recall deficits on most memory tasks, the differences between depressed subjects and control subjects increases on tasks involving higher degrees of complexity, abstraction, or effort (Braff & Beck, 1974; Cohen, Weingartner, & Smallberg, 1982; Miller, 1975). Also, deficits in memory can be amelioriated by improvements in mood by either antidepressants or by success experiences (Glass, Uhlenhuth, Weinrub, Fischman, & Teuch, 1978).

Finally, expected relationships between negative expectancies and mood have also been found (Beck, 1972). In addition, high levels of hopelessness have been found consistently among depressed patients (Beck, Kovacs, & Weissman, 1975; Minkoff, Bergman, & Beck, 1973; Rush, Beck, & Kovacs, 1982).

In summary, the above studies have been used to support Beck's view of the relationship between dysfunctional cognitions and depression. In

spite of these studies, a number of studies have also questioned this relationship. For example, Rosenzweig (1960) did not find depressed subjects to be more negative than nondepressed subjects following a success or failure experience. Also, Alloy and Abramson (1979) studied the ability of depressed and nondepressed subjects to detect the degree of contingency between responding and outcome in a series of problems that varied in the actual degree of objective contingency between the performance responses and outcomes obtained. No differences were found between depressed and nondepressed subjects. Furthermore, nondepressed subjects judged that they had more control (erroneously) in a "win" situation than a "lose" situation, while depressed subjects accurately reported the amount of control in both situations (Lewinsohn & Hoberman, 1981). Lewinsohn, Mischel, Chaplin, and Barton (1980) found that depressives' ratings of their social competence were likely to match objective observers' ratings of the depressives' competence, while psychiatric and normal controls were likely to rate themselves more positively than the observers did. In another study by Lewinsohn, Steinmetz, Larson, and Franklin (1981), a longitudinal study was conducted to assess whether cognitions known to be correlated with depression precede, accompany, or follow an episode of depression. While the results were generally consistent with the hypothesis that depression-related cognitions arise concomittantly with an episode of depression, subjects who were to become depressed later during the course of the study did not differ from controls on the cognitive measures, and depressive cognitions did not seem to be permanent residuals of a depressive episode. The authors concluded that this

study supported the notion that cognitive dysfunction was a correlate of and not an antecedent of a depressive episode. In a similar vein, Silverman, Silverman, and Eardley (1984) administered the Dysfunctional Attitude Scale (DAS) and found that dysfunctional thinking was more prominent while the subject was depressed versus when the subject was asymptomatic as the result of antidepressant treatment. Similarly, Simons, Garfield, and Murphy (1984) treated depressed patients with medication or cognitive therapy and found identical changes on cognitive measures for both groups. In all, these studies argue that dysfunctional thinking may be a "result" or "correlate" of depression rather than the "cause."

In conclusion, while there are a number of studies supporting the relationship between dysfunctional thinking and depression, a number of studies question the causal nature of this relationship. Therefore, at this time, Beck's theory of depression cannot be accepted as a comprehensive, all encompassing explanation of depression. Nevertheless, Beck's model has resulted in the development of a comprehensive and powerful therapy for depression, which Beck describes in detail in his book Cognitive Therapy of Depression (Beck, Rush, Shaw, & Emery, 1979).

Beck's time-limited therapy package focuses not only on modifying dysfunctional thoughts, but also focuses on modification of overt behavior (e.g., graded task assignments, activity schedules, assertion training) as a way to generate data contradictory to the client's negative self-perception. Cognitive modification procedures teach the client that his/her depression is related to maladaptive thought

patterns and trains the client to identify and logically challenge the erroneous assumptions underlying these cognitions. These basic assumptions are conceptualized as hypotheses to be tested. The client learns to challenge the validity of his/her negative attitudes by examining the disconfirmatory data. In summary, Beck's strategy is as follows: (a) teach the client that a relationship exists between thoughts, feelings, and behavior; (b) teach the client to monitor his/her automatic thoughts and to determine the underlying depressogenic assumptions; (c) teach the client to identify logical errors and depressive assumptions made; (d) help the client evaluate and look for alternative explanations for negative thoughts and logical errors; (e) teach the subject to evaluate and correct dysfunctional thoughts and assumptions by learning to cope with logical errors and designing experiments; and (f) help the client test out his/her hypotheses and adjust dysfunctional thoughts and faulty assumptions accordingly.

Outcome data on Beck's cognitive therapy has been quite impressive, particularly when the subject population has been nonpsychotic, nonbipolar depressed outpatients. Several reviews summarize the outcome data (Kendall, 1984; Lewinsohn & Hoberman, 1981; Wright & Beck, 1983). To illustrate, Kovacs, Rush, and Beck (1981) randomly assigned unipolar, depressed outpatients to either cognitive therapy or imipramine therapy with medicine checks. The results showed that patients receiving cognitive therapy showed greater improvement in depressive symptoms than patients receiving imipramine. Although both groups were significantly improved after therapy, the cognitive therapy group was more likely to maintain its gains at a three-month follow-up when compared to the

imipramine group. While not statistically significant, this trend continued at a six-month follow-up and at a one-year follow-up. This study was essentially a replication in methodology and results of a study done by Rush, Beck, Kovacs, and Hollon in 1977. While these studies had similar methodological difficulties (e.g., mainly the adequacy of drug therapy was questioned in terms of blood levels), they conclusively showed the effectiveness of cognitive therapy. Other studies have also shown this effectiveness of cognitive therapy. For example, Shaw (1977) found that depressed outpatients treated individually with cognitive therapy improved to a significant degree when compared to a behavioral group therapy, a nondirective group therapy, or no therapy. McLean and Hakstain (1979) studied 170 outpatients who were significantly depressed according to the Research Diagnostic Criteria (RDC) and the Beck Depression Inventory (BDI). The patients were randomly assigned to treatment with cognitive therapy, psychodynamically oriented therapy, relaxation training, or amitriptyline therapy (adequacy of this therapy was measured by blood levels). Cognitive therapy was shown to be superior to the other three groups. Finally, Blackburn, Bishop, and Glen (1981) compared cognitive therapy with pharmacotherapy and combined treatment. Overall, the combined treatment gave the best results, with cognitive therapy alone and pharmacotherapy alone also producing significant improvements.

In summary, Wright and Beck (1983) conclude that "to date, all outcome studies on outpatients who met the Research Diagnostic Criteria for depression have found cognitive therapy to be an effective

treatment. Cognitive therapy has been at least equal to pharmacotherapy in all studies comparing the two treatments."

Biological-Disease Model of Depression

The biological-disease model of depression assumes that factors such as genetics or a malfunction of some biochemical system is the cause of the disorder. Therefore, the "signs" and "symptoms" of depression serve to reflect the underlying organic disorder. Since the cause is seen as an organic factor, medicines are often used to treat the underlying malfunction. Data used to support this approach to depressive disorders are: (1) genetic studies; (2) biogenic amine metabolism studies; and (3) studies of endocrine abnormalities. This section briefly reviews these studies, with emphasis on endocrine studies due to the close relation with the dexamethasone suppression test. Effective chemotheraputic approaches to the treatment of depression are also examined.

The role of heredity in psychopathology, and particularly depressive disorders, is inferred from evidence on correlations of depression among members of the same family. Methodologies commonly used to examine the genetic influence on psychopathology are the pedigree analysis; the family method; twin studies; and adoption studies. A number of reviews of the above methodologies as they relate to depression are available (Dunner, Gershon, & Goodwin, 1976; Gershon, Baron, & Leckman, 1975; Gershon, Bunney, & Leckman, 1976; Hutchings & Mednick, 1975; Mendlewicz, Fleiss, & Frene, 1975; Rieder & Gershon, 1978; Suslak, Shopsin, Silbey, Mendlewicz, & Gershon, 1976; Winokur &

Cadoret, 1977). As Winokur and Cadoret (1977) conclude, the research involving genetic studies in depressive disorders shows that: (a) family members of depressive patients are more likely themselves to be ill with an affective disorder than are members of the general population; and (b) in twins where one member has a depression, concordance is greater in monozygotic pairs than dizygotic pairs. In spite of this conclusion, it cannot be concluded that depression is an inherited disorder. The fact that correlations or concordance rates are not 100% argues for environmental influences. In addition, there are several methodological difficulties present in many studies that examine genetic influences, such as inaccurate diagnoses, the use of different diagnostic systems, an inability to locate family members, and small sample sizes.

Biogenic amine metabolism studies have been examined in relation to depressive disorders. As Zis and Goodwin (1982) point out, the biogenic amine hypothesis of affective disorders grew out of associations between observations of the clinical effects of certain drugs and the neurochemical effects of these drugs in animal brain. In its most basic form, this hypothesis points that depression results from an absolute or relative deficiency of the catecholamines norepinephrine and dopamine, and of the indoleamine serotonin at functionally important adrenergic receptors in the brain. All of the above compounds are known as monoamines collectively. The physiological activity of monoamines can be altered by interfering with any of the processes involved in synthesis, in storage, in release, in metabolism, or in controlling the sensitivity of receptors to one or another monoamine (Schildkraut,

1977a). Evidence for the biogenic amine hypothesis has been accumulated, based on (a) the physiological effects of substances that appear to induce depression; (b) the physiological effects of drugs that alleviate it; and (c) the levels of metabolic by-products of the activity of these brain chemicals which show up in body fluids. These data are briefly reviewed.

Reserpine, a drug used as an anti-hypertensive medication, was found to induce depressive symptoms in some individuals. Animal studies revealed that the drug's primary mode of action was to impair the capacity of neurons to retain monoamines in storage granules, and that the brain levels of the biogenic amines, norepinephrine, dopamine, and serotonin, were depleted after administration of reserpine (Schildkraut, 1977a). Zis and Goodwin (1982) conclude that "overall, drugs that increase the functional output of catecholamine systems act either as stimulants and precipitants of mania or as antidepressants. Conversely, drugs that decrease the functional output of these systems act as sedatives or antimanics." They are also quick to point out that not all of the drug findings are consistent in connecting the biogenic amines to affective disorders. Zis and Goodwin (1982) cite several studies showing contradictory results (Coppen, Gupta, & Montgomery, 1976; Davis, Colburn, Murphy, & Robinson, 1979; Fann, Davis, & Janowsky, 1973; Murphy, Campbell, & Costa, 1978; Shopsin, Gershon, Goldstein, & Friedman, 1975; Van Praag, 1978).

The effectiveness of antidepressant drugs has been used as support for the biogenic amine hypothesis. The two main classes of drugs used to treat depression are: (a) tricyclics, and (b) monoamine oxidase

inhibitors. It has been found that monamine oxidase inhibitors, which were initially used in the treatment of tuberculosis, also have antidepressant effects in some patients. These drugs, as the name implies, inhibit the enzyme monoamine oxidase, resulting in increases of norepinephrine, dopamine, and serotonin in the brain (Becker, 1977; Schildkraut, 1977b). In addition, tricyclic antidepressants, which operate differently than monoamine oxidase inhibitors, interfere with the re-uptake of norepinephrine and serotonin once they are released at receptor sites. Even though this information supports the biogenic amine hypothesis, problems in accounting for certain phenomena exist. For example, the immediate effect of administering either an monoamine oxidase inhibitor or a tricyclic antidepressant is the increase in available norepinephrine or serotonin, but clinical improvement requires two or three weeks (Fraser, 1975). In addition, chronic administration of tricyclic antidepressants results in a decrease in sensitivity of various receptor sites, without the concomitant behavioral changes (Mendels & Fraser, 1974).

Finally, examination of amine metabolites in the cerebrospinal fluid (CSF) and urine in depressed versus normal controls has been used to support the biogenic amine hypothesis. These metabolites consist of 3-methoxy-4-hydroxyphenylglycol (MHPG), which is considered the major product of norepinephrine, and 5-hydroxyindol-eacetic acid (5HIAA), which is considered the major by-product of serotonin (Zis & Goodwin, 1982). In their review of the literature, Zis and Goodwin report several studies which report decreased baseline levels in depressives of 5HIAA as compared to "control" groups (Asberg, Thoren, & Traskinan,

1976; Ashecroft, Crawford, & Eccleston, 1966; Coppen, Prange, & Whybrow, 1972; Denker, Malm, Roos, & Werdinius, 1966; McLeod & McLeod, 1972; Subrahmanyan, 1975; Van Praag & Korf, 1971), while others do not find differences (Berger, Faull, & Kilkowski, 1980; Bowers, Heninger, & Gerbode, 1969; Fotherby, Ashcroft, & Affleck, 1963; Goodwin, Post, & Dunner, 1973; Papeschi & McClure, 1971). Likewise, Schildkraut (1977a) reports several studies showing lower MHPG levels in depressives versus "control" subjects (Bond, Jenner, & Sampson, 1972; Greenspan, Schildkraut, Gordon, Baer, Aranoff, & Durell, 1970; Jones, Maas, Dekirmenyian, & Fawcett, 1973; Schildkraut, Keeler, Rogers, & Draskoczy, 1973), while other studies do not find this relationship (Bunney, Goodwin, Murphy, House, & Gordon, 1972; Shopsin, Wilk, Gershon, Roffman, & Goldstein, 1973).

In summary, a number of different types of data have been used to support the biogenic amine hypothesis of depression. As Zis and Goodwin point out, these various studies have not provided clear, unequivocal support for the "too little-too much" amine hypotheses. They note that many confounding variables, such as non-standardized diagnostic systems, inconsistent demographic variables across studies, a person's amount of activity, and phase differences in terms of what phase of the illness the data are collected may account for many of the discrepancies found in the data. Prange (1974) has revised the biogenic amine hypothesis (calling it the "permissive biogenic amine hypothesis") in order to try and account for some of the data. He espouses the belief that a deficit in a central indoleaminergic transmission (e.g., serotonin) is a necessary, but not sufficient cause. If a deficit is accompanied by

lower catecholaminergic transmission (e.g., norepinephrine), then depression occurs. While Prange (1974) does not speculate on the cause of the changes in the levels of these transmitters, the person's genetics are seen as playing the major role.

As was mentioned earlier, studies have examined the role of the endocrine system in depressive disorders. As Sachar (1982) points out, the rationale in examining the endocrine system was based on the observation that "endogenous" or "melancholic" depressions have symptoms suggestive of hypothalamic dysfunction (e.g., disturbances in sleep, sex drive, appetite, mood), which would indicate a possible dysfunction in the neuroendocrine system. Furthermore, the same neurotransmitters previously mentioned (e.g., norepinephrine, serotonin) are involved in the functioning of the hypothalamic neuroendocrine cells. Three types of data have been used to examine the effects of neuroendocrine function in depressives: (a) cortisol; (b) growth hormone (HGH); and (c) thyroid stimulating hormone (TSH).

Various techniques have been used to examine cortisol secretion as it relates to depressive disorders (e.g., isotope dilution methods). Sachar (1975, 1982) has found that half of the patients with major depression have substantially increased cortisol levels, which appear to remit with improvement in the client's condition. They also report that in examining the 24 hour plasma cortisol pattern that depressives disproportionately secrete excess cortisol in the afternoon, evening, and early morning hours. During these times, cortisol secretion is usually minimal. Another index of cortisol secretion is the dexamethasone suppression test. Carroll (1978, 1980) has reported that

a significant subgroup of depressives (approximately 40%) exhibit abnormal secretion of cortisol 17 to 24 hours after ingesting 1 mg. of dexamethasone, which would normally inhibit cortisol secretion. One hypothesis concerning this finding suggests that the inability of depressives to respond to the administration of dexamethasone is due to a deficiency of norepinephrine in the hypothalamic pathways (Carroll, 1978; Sachar, Asnis, & Nathan, 1980). Another theory asserts that the primary abnormality in this subtype of depression is an excess of plasma cortisol, which results in a deficiency of brain serotonin (Hatotani, 1979; Nomura & Inoue, 1979). These theories are consistent with the "permissive biogenic amine hypotheses" by Prange (1974) presented earlier. Since the nature of this dissertation involves the dexamethasone suppression test, it is examined in detail later.

As Becker (1977) asserts, evidence for possible neuroendocrine dysfunction associated with biogenic amines comes from growth hormone (GH) response to insulin-induced hypoglycemia, which appears to be involved with the monoamines. Carroll (1978) reports that growth hormone response is diminished in major "endogenous" depressions. Gruen, Sachar, and Altman (1975) matched females in terms of age and phase of menopause (pre-menopause is associated with lower growth hormone response, therefore all subjects were post-menopause) and found depressives to have a lower growth hormone response when compared to "normal" controls.

Finally, studies have examined thyroid function and thyroid stimulating hormone (TSH). Results have been conflicting, with some studies showing no abnormality in thyroid function in depressives

(Kirkegaard, Norlein, & Lauridsen, 1975) while other studies have shown depressives to have a deficient thyroid stimulating hormone response (Maeda, Kato, & Ohgo, 1975; Prange, Wilson, & Breeze, 1976).

In summary, there have been a number of conflicting findings in the examination of neuroendocrine systems as they relate to depressive disorders. As with studies in the genetic area and the biogenic amine research, the neuroendocrine studies have provided support for a biological-disease model for depression, but the heterogeniety in the findings has prevented a biological-disease model from being accepted as a comprehensive, all encompassing explanation of depression.

Even though much controversy surrounds the biological-disease model of depression, effective chemotherapeutic approaches have been established over the last twenty years. This dissertation was concerned with the antidepressants called tricyclic antidepressants (e.g., imipramine, nortriptyline, doxepin, desipramine). Much is known about their beneficial effects in treating depressive disorders and about their unwanted side effects. Several review articles are available concerning tricyclic medications (Bennett, 1966; Klein & Davis, 1969; Mandel & Klerman, 1979; Mindham, 1982; Morris & Beck, 1973; Wechsler, Grosser, & Greenblatt, 1965). Overall, these studies report that tricyclic antidepressants are the most effective when compared to other types of antidepressants; that tricyclic antidepressants are consistently superior to placebos; and that little difference in effectiveness has been found when comparing different tricyclics to each other. Therefore, as Mindham (1982) concludes, "the weight of evidence is certainly in favor of the tricyclic drugs being superior to placebo

in the treatment of depressive patients." Finally, several studies have shown both cognitive therapy and chemotherapy (using mainly tricyclic antidepressants) to be essentially equal in effectiveness for the treatment of depressive disorders (Blackburn, Bishop, & Glen, 1981; Kovacs, Rush, & Beck, 1981; Wright & Beck, 1983).

Dexamethasone Suppression Test

Since this dissertation dealt specifically with the dexamethasone suppression test, closer examination of this test is needed. It was previously mentioned that studies of neuroendocrine abnormalities have supported a biological-disease model of depression. It was also pointed out that examination of cortisol secretion is one method of assessing neuroendocrine function. One index of cortisol secretion that is frequently used in psychiatry is the dexamethasone suppression test. Clinical application of the dexamethasone suppression test consists of administration orally of 1 mg. of dexamethasone (a synthetic corticosteroid) which is ingested and affects the cortisol "feedback" receptors in the brain and normally turns off the endogenous secretion of cortisol (e.g., causes suppression of cortisol). Blood tests to assess plasma cortisol levels are then drawn 17 to 24 hours later. An abnormal dexamethasone suppression test (e.g., a positive dexamethasone suppression test) results in nonsuppression of cortisol 17 to 24 hours later. This procedure has been standardized (Carroll, 1980), with the accepted cortisol level for abnormality being greater than or equal to 5 ug/dl.

This test was introduced in 1960 to study Cushing's syndrome, with the results used to support the hypothesis of cortisol hypersecretion in patients with this syndrome (Flori & Davis, 1984). It also supported hypothalamus dysfunction since cortisol was not being suppressed. Since depression in patients with Cushing's disease was well documented, this led to the use of the dexamethasone suppression test to determine whether endocrine abnormalities (e.g., disturbance with the hypothalamic-pituitary-adrenal (HPA) axis) was occurring in depression.

Approximately ten years ago, Carroll (1976) reported that a significant subgroup of depressives (approximately 40%) exhibited abnormal results according to the dexamethasone suppression test. It was also shown by this author that these results were not due to stress, agitation, or the administration of psychotropic drugs. In addition, abnormal dexamethasone suppression test results were not found in populations of normals, schizophrenics, drug abusers, and character disorders. Subsequent studies confirmed these results (Brown & Shuey, 1979; Carroll, Feinberg, & Greden, 1981; Stokes, Pick, & Stoll, 1975) while other studies refuted these results (Holsboer, Bender, & Benkert, 1980; Sachar, Asnis, & Nathan, 1980; Shopsin & Gershon, 1971). As Brown and Shuey (1979) and Sachar (1982) point out, much of the inconsistency from study to study may have been a result of conducting and evaluating the dexamethasone suppression test differently. For example, some studies gave 1 mg. of dexamethasone, while others gave 2 mg. Some studies sampled plasma cortisol levels at 4, 8, 16, or 24 hours after ingestion of dexamethasone. Also, various studies used different cortisol levels to determine abnormality. Finally, diagnostic

classification was inconsistent across studies. As mentioned above, this led to research to standardize the procedure, which consists of 1 mg. of dexamethasone administered and cortisol measured 17 to 24 hours later, with the criteria for an abnormal dexamethasone suppression test set at greater than or equal to 5 mg/dl plasma cortisol secretion (Carroll, 1980).

Controversy currently exists as to the clinical definition of the subgroup of depressives showing abnormal results on the dexamethasone suppression test. Carroll, Feinberg, and Greden (1981) have presented strong data showing that the dexamethasone suppression test correctly identified 96% of inpatients who were depressed with a diagnosis of melancholia. They argue that this result represents the subgroup of depressives that are characterized as "endogenomorphic" depression as presented by Klein (1974), with the assumption that this class of depressives have a biological defect that would respond well to antidepressants. Other studies using similar criteria as Carroll et al. (1981) have found abnormal dexamethasone suppression test results in depressed patients not exhibiting melancholia (Coppen, Rao, & Ruthven, 1980; Stokes, Pick, & Stoll, 1975). It has also been argued that abnormal results on the dexamethasone suppression test distinguish between primary versus secondary depressions. Brown, Johnson, and Mayfield (1979) along with Schlesser, Winokur, and Sherman (1979) report results suggesting that an abnormal dexamethasone suppression tests indicates a primary depression while only 0% to 4% of schizophrenics, drug abuses, normals, or character disorders have an abnormal dexamethasone suppression test. In contrast, Insel and Goodwin (1983)

present results which assert that the dexamethasone suppression test does not distinguish between depressives and patients with obsessive-compulsive disorders, dementia, or eating disorders. These authors state that "to conclude from dexamethasone suppression data such disorders are actually variants of depression is, at this point, to beg the question of the diagnostic usefulness of the test." Finally, Brown et al. (1979) points out that another problem with the dexamethasone suppression test is that consistent clinical differences between depressed patients who have normal versus abnormal results on this test have not been found.

In summary, a great deal of controversy exists over what subgroup of depressives, if any, the dexamethasone suppression test defines. A comprehensive review by the National Institute of Mental Health (Hirschfeld, Kaslow, & Kupfer, 1983) on the clinical utility of the dexamethasone suppression test concludes that: (a) clinical differences in patients with affective disorders between dexamethasone suppression test suppressors and nonsuppressors should be investigated further; (b) while some diagnostic strategies have yielded promising results (e.g., primary versus secondary; endogenous versus exogenous), further investigation and replication is needed; and (c) prevalence rates of dexamethasone suppression test nonsuppressors must be accurately determined in large groups of patients whose conditions have been carefully described and diagnosed (e.g., depressive subtypes, schizophrenia, organic brain syndromes). In a separate review of the dexamethasone suppression test, Insel and Goodwin (1983) conclude that this test is most useful in the research arena since diagnostic groups

are often well diagnosed and delineated. They also assert that further research is needed before the clinician can confidently use the dexamethasone suppression test to differentiate between diagnostic groups.

In spite of the controversy over using the dexamethasone suppression test to differentiate between types of depressions and diagnostic groups, it is generally believed by most researchers and clinicians examining and using this neuroendocrine test that nonsuppression (an abnormal test) is a manifestation of some abnormal neuroendocrine state requiring some type of chemotheraputic approach for its treatment (Brown, Johnson, & Mayfield, 1979; Brown & Shuey, 1979; Carroll, 1980; Fraser, 1975; Greden, Albala, & Haskett, 1980; Gwirtsman, Gerner, & Sternbach, 1982; Insel & Goodwin, 1983; Nemeroff & Evans, 1984; Sachar, 1982; Schlesser, Winokur, & Sherman, 1979). This is exemplified by the quote by Akiskal and McKinney (1975):

> It would appear that no matter what interpersonal factors mobilize depressive behaviors, once the latter reach melancholic stage, they become biologically autonomous and refractory to psychotheraputic intervention.

Even though this is widely believed, the review of the dexamethasone suppression test by The National Institute of Mental Health (Hirschfeld et al., 1983) and by Nemeroff and Evans (1984) concludes that no studies have definitely answered the question of whether suppression or nonsuppression at the time of original examination predicts response to various types of treatment, and that "the search should continue for possible specificity of treatment modality on the basis of the dexamethasone suppression test" (National Institute of Mental Health, Hirschfeld et al., 1983). In other words, whether the dexamethasone

suppression test is a biological marker for a subtype of depression that responds well to biological treatments has not been demonstrated. There have been no well-controlled studies looking at this question, although a few preliminary reports show a relationship between an abnormal dexamethasone suppression test and a good response to tricyclic antidepressants (Brown, Johnson, & Mayfield, 1979; Brown & Shuey, 1979; Greden, Albala, & Haskett, 1980). Others have questioned whether cognitive therapy can be effective for abnormal dexamethasone suppression test responders (Carroll, Feinberg, & Greden, 1981; Rush, 1982; Williams, 1984). These studies have numerous methodological difficulties, such as small sample sizes, poor outcome measures, poor diagnostic procedures, and a post hoc examination of treatment response. Nemeroff and Evans (1984), along with other researchers (Bowie & Beaini, 1985; Greden et al., 1980; Herschfeld, Kaslow, & Kupfer, 1983; Spar & Rue, 1983) have found that the dexamethasone suppression test normalizes after successful treatment of the depression with medications, and that depressives whose test failed to normalize showed significantly less clinical improvement and a high risk of relapse. These studies also had various methodological difficulties, and the results are in need of replication. Finally, there have been no studies examining the relationship between the dexamethasone suppression test and non-chemotheraputic treatment approaches to depression (e.g., cognitive therapy) (Insel & Goodwin, 1983). This dissertation attempted to examine whether the possible specificity of treatment modalities used to treat depression can be made based on the dexamethasone suppression test in order to maximize treatment effectiveness. In other words, the

"treatment validity" of the dexamethasone suppression test was examined. The "treatment validity" approach is examined next.

Treatment Validity: Contribution of Assessment to Treatment Effectiveness and its Relation to Depression

One of the main questions examined in this dissertation is the contribution of the dexamethasone suppression test to treatment effectiveness. In the area of behavioral assessment, this has been termed "treatment validity" (Nelson & Hayes, 1979). Since the goals of behavioral assessment are to "identify meaningful response units and their controlling variables for the purpose of understanding and altering behavior" (Nelson & Hayes, 1979), the concept and study of treatment validity has allowed for the evaluation of the quality of the data generated by behavioral assessment. While the treatment validity approach has been mainly applied to the data generated by behavioral assessment, this dissertation utilized this concept and the associated methodology to examine the treatment validity of the dexamethasone suppression test. Therefore, a review of the treatment validity approach, along with its relevance to depression is warranted.

Review of Treatment Validity

During the last ten years, behavioral assessment has come to be viewed as an assessment approach with its own unique set of assumptions and goals. Prior to this time, behavioral clinical psychology was mainly concerned with treatment interventions and independent variables. The initial developments in behavioral assessment often mirrored those

in behavior therapy. As a result, behavioral assessment was not well defined. In its early emergence as a separate topic, behavioral assessment was thought to be merely a sub-area of traditional psychological assessment. Only recently has behavioral assessment become well-defined and concerned with its own research questions. This interest has been manifested by the advent of journals such as Behavioral Assessment (Pergamon and the Association for Advancement of Behavior Therapy) and Journal of Behavioral Assessment (Plenum), along with the emergence of several books by Hersen and Bellack (1976), Cone and Hawkins (1977), Ciminero, Calhoun, and Adams (1977), Haynes (1978), Haynes and Wilson (1979), Keefe, Kopel, and Gordon (1978), Barlow (1981), Mash and Terdal (1981), and Nelson and Hayes (1981).

One critical question that was facing behavioral assessors was: How do we evaluate the quality of behavioral assessment? As behavioral assessors seriously began to consider this question, it became evident that methods used to evaluate traditional assessment, such as psychometric theory or generalizability theory, were quite limited in their usefulness in evaluating behavioral assessment. For example, psychometric theory is mainly concerned with the reliability and validity of data. Reliability, on the one hand, involves the consistency of the measure being used and is expressed as a correlation. Consistency across time (test-retest reliability), across test items (split-half reliability), and across different forms of the test (parallel forms reliability) is seen as an indication of a good assessment device because more of the "true" score is being measured instead of measurement error (Cronbach, 1970). Validity, on the other

hand, examines "the extent to which a test measures what it purports to measure" (Cronbach, 1970). One type of validity is criterion-related validity, which is the extent to which the test (an assessment device) results correlate with some external variable which supposedly measures the same variable as the test does. The two types of criterion-related validity are: concurrent validity, which is the extent to which test results correlate with some other concurrent measure of the same variable, and predictive validity, which is the extent to which the test (any assessment device) results correlate with some future behavior. Other types of validity are: content validity, which involves the extent to which relevant samples of the criterion situation are represented in the test situation; convergent validity, which involves taking several assessment devices which are supposedly related and actually examine the degree of relationship; and finally, construct validity, which is the extent to which the test measures a theoretical construct or trait. These traditional psychometric standards have also been relabelled according to generalizability theory (Cronbach, Gleser, Nanda, & Rajaratnam, 1972). This was accomplished by relabelling the different types of reliability and validity as universes of generalization (conditions of the assessment situation) which could affect the generalizability of behavioral assessment techniques. These are: (a) scores (parallel forms reliability); (b) items (split-half reliability); (c) time (test-retest reliability); (d) settings (temporal consistency or external validity; (e) method (convergent validity); (f) dimension (construct validity, concurrent validity, and discriminant validity).

By using analyses of variance, the proportion of variance accounted for by each "universe of generalization" can be determined.

While Nelson and Hayes (1981) and Hayes, Nelson, and Jarrett (in press) acknowledge specific uses for psychometric and generalizability theory in the area of behavioral assessment, they conclude that these theories do not provide an adequate means of evaluating the quality of behavioral assessment. Hayes et al. (in press) assert that psychometric theory and generalizability theory are not adequate because (a) their assumptions about behavior; (b) level of analysis; and (c) model of causality differences from a behavioral perspective. For example, psychometric theory (reliability and validity) assume that behavior is enduring and stable, and consistency is seen as a hallmark of a good assessment device (Hayes et al., in press). Behavioral assessment, however, assumes that behavior is not necessarily enduring and consistent, and that inconsistency in measurement may be the result of actual changes in behavior rather than an imprecise behavior assessment technique. Psychometric theory is also based on the level of analysis involving group data, while behavioral assessment focuses on the analysis of the individual. As Hayes et al. (in press) note: "the issue is not one of number (few versus many) but of the level of analysis (individual versus group) upon which principles and findings are based." Finally, psychometric theory and behavioral assessment differ fundamentally in their view of causality. As Hayes et al. argue: "In psychometrics, events can be explained based on the structure of the organism (e.g., the structure of the mind). That is, structure can assume causal status. In modern behaviorism, the

structure of the organism is itself something to be explained by the functional interaction between the organism and the world over both short time frames (e.g., in the lifetime of the individual) and long time frames (e.g., in the lifetime of the species). Structure is not unimportant in this view, but it is not a cause. Instead it is a host for causal agents."

Therefore, in response to the above arguments against psychometric and generalizability theory in evaluating behavioral assessment, Nelson and Hayes (1979) put forth the idea that in order to evaluate the quality of behavioral assessment, one must consider the function served by behavioral assessment. Nelson and Hayes (1979) state that at least two functions seem critical: the function of behavioral assessment in increasing our understanding of behavior, which is termed "conceptual validity," and the function of behavioral assessment in contributing to treatment effectiveness, which is termed "treatment validity." Conceptual validity, according to Nelson and Hayes (1979), asks the question, "Does the design of this procedure or experiment and its results enable us to support, extend, modify, or elaborate behavioral principles and assumptions?" Conceptual validity cannot be measured quantitatively, and becomes apparent only after the passage of time as more general and conceptually consistent principles of behavior are evolved (Nelson & Hayes, 1979). Nelson and Hayes (1979) also assert that "treatment validity" can be shown by examining the extent to which behavioral assessment contributes to treatment effectiveness. The treatment validity approach was be examined in this research project and therefore is considered in greater detail.

Hayes et al. (in press) assert that treatment validity can be demonstrated empirically by showing that a particular behavioral assessment led to a better treatment than would have occurred without the treatment. Hayes et al. reviewed the multiple methodologies available to examine treatment validity. These methodologies are reviewed in the context of their relevance to depression. Treatment validity in the area of depression is examined when one attempts to match the depressive's "characteristics" or problematic target behaviors to specific types of treatment for depression. Special emphasis will be placed on the treatment validity methodology being utilized in this dissertation.

Treatment Validity Methodologies and Their Relation to Depression

According to Hayes et al. (in press), the treatment validity approach can be used to examine questions such as: Does the quality of assessment devices or strategies, or the nature of theoretical distinctions, or the quantity of assessment contribute to the effectiveness of treatment? Hayes et al. (in press) divide the methodologies used to examine these questions into three main categories: (a) post hoc identification of dimensions; (b) a priori-single dimension studies which are sub-divided into manipulated assessment, manipulated match, and observed differences studies; and (c) a priori-multiple dimension studies. These type of methodologies can be done in a group or single- subject design format.

Post hoc identification of dimensions involves administering treatments to many persons and identifying post hoc aspects of the

assessment devices which help predict therapy responders. For example, Hayes et al. (in press) report a study by Bielski and Friedel (1976) which showed that higher social class, insidious onset, anorexia, weight loss, middle and late insomnia, and psychomotor disturbance were all positively related to a favorable response to tricyclic medication. In contrast, neurotic, hypochondriacal, and hysterical traits, multiple prior episodes, and delusions predicted a poor response to imipramine and amityptyline.

A priori-single dimension studies, as mentioned before, are subdivided into (a) manipulated assessment, (b) manipulated match, and (c) observed differences studies. The manipulated assessment treatment validity studies focus on the utilization of assessment data. A single group of subjects is divided into two groups, and a single aspect of assessment is varied systematically. Hayes et al. (in press) gives the example of information collected on all subjects but made available to therapist in one group and not the other. The therapist in the former group would design and implement the treatment according to the assessment data available. Differential outcomes between groups would confirm the treatment validity of the assessment characteristics manipulated. Manipulated match treatment validity studies manipulate the correspondence between assessment information and treatment. A study by McKnight, Nelson, Hayes & Jarrett (1984) manipulated the correspondence between three target behaviors (high frequency of irrational thoughts, social skill deficits, or both irrational thoughts and social skill deficits) and treatment (social skills training versus cognitive therapy) in depressives. Results showed that not only the

global measures of depression, but also the specific measures of each related target behavior (e.g., irrational beliefs or social skill deficits) improved more when treatment was matched to the target behavior than when it was not. Finally, observed differences treatment validity studies divide subjects into groups non-randomly based on assessment differences. Subjects then receive one type of treatment. Differences between these groups then shows the treatment validity of these differences. For example, depressed subjects with a high frequency of irrational beliefs with and without anxiety might all be given cognitive therapy. If only those without anxiety showed changes in their depression and irrational beliefs, then the treatment validity of the distinction between these two groups would be established.

A priori-multiple dimension studies involve various combinations of the subgroups described above, such as: manipulated assessment-manipulated match studies; manipulated assessment-observed differences studies; manipulated match-observed differences studies; manipulated assessment-manipulated match-observed differences studies; and observed differences with two or more treatments studies (Hayes et al., in press). Since this dissertation employed the observed differences with two or more treatments methodology, it is examined in detail.

Hayes et al. (in press) assert that one way "observed differences with two or more treatments studies" are conducted is to cross distinct patient groups with two or more distinct treatment approaches. Hayes et al. feel that this type of research is "elegant when the nature of each patient group seems to imply a distinct treatment approach." They go on to say the factorial design that results tests not only the treatment

validity of the patient group distinctions but also the conceptual and theoretical distinctions which gave use to them and their implied therapies. This dissertation employed this particular strategy, with the two distinct patient groups being depressives with normal versus abnormal dexamethasone suppression test responders, and the two distinct treatment approaches being a chemotheraputic approach (tricyclic anti-depressants) and a psychological approach (cognitive therapy). Therefore this methodology examined the treatment validity of the patient group distinctions and their contribution to treatment effectiveness. More specifically, does abnormal dexamethasone suppression test findings result in a chemotheraputic approach being more effective than a psychological approach? There are also implications for the theoretical distinctions (e.g., in this case, the endogenous versus exogenous distinction as related to the dexamethasone suppression test) and the related therapies (chemotherapy versus cognitive therapy) previously discussed. In order to effectively address these issues, it was necessary to select a psychological treatment as effective as medication. Therefore, all subjects were chosen with a high frequency of dysfunctional in order to give cognitive therapy a chance to be effective. As Hayes et al. conclude, this particular treatment validity design can improve our understanding of behavior and contribute to an inductive clinical science. When a procedure or experiment contributes to the establishment of general and conceptually consistent principles, it is said to have "conceptual validity" or "theoretical validity" (Hayes et al., in press). In dealing with treatment validity studies that also have "theoretical

validity," certain philosophical/conceptual issues, such as the relationship between etiology and treatment, along with the mind-body problem, need to be addressed.

Acknowledgment of Important Philosophical/Conceptual Issues

Relationship Between Etiology and Treatment

In the study of depressive disorders, there is an interactive role between etiology and treatment outcome. Many times etiological research leads to treatment suggestions, or successful treatment leads to suggestions concerning etiological research. This is quite evident in the review of the biological and cognitive etiological models of depression and their associated treatments. A problem arises, however, when a researcher tries to validate a particular theory by utilizing the effectiveness of a particular treatment. Examples of logical errors involved in the relationship between etiology and treatment will clarify this problem. For example, Rimland (1964) points out the error of inferring etiology from treatment by using the "aspirin analogy." He coined this term after a review of the literature on autism in which an etiological hypothesis was made on the basis of treatment effectiveness. He points out the logical error of assuming that because contingency management can improve some response classes of autistic children, then autism must be "caused" by poor parental contingency management. This is logically the same as saying that if aspirin can help a headache, it must be the lack of aspirin that caused the headaches. Therefore, the success of cognitive therapy in treating depressives does not necessarily mean that irrational beliefs actually "caused" the depression.

This dissertation avoided the above-mentioned logical error by not asserting that the treatment outcome of this study proves the related theory associated with the theoretical distinctions examined. While this study may support the theoretical distinction and related theories, it can only provide speculation for future etiological research.

Mind-Body Issue

The relationship between etiology and treatment overlaps with the mind-body problem in this discussion. The mind-body problem dates back to the days of De Cartes and is primarily a philosophical religious issue. The issue deals with whether the mind and the body are separate systems (dualism), or are both composed of the same physical matter and are part of the same physical system (physical monism). In reference to the above discussion, the question becomes: With regard to one's view on etiology, how can nonphysical treatment (e.g., cognitive therapy) alter a disorder that has physical cause or how can a physical treatment (e.g., chemotherapy) have an effect on an affective disorder caused by irrational beliefs? A dualist might propose an interactionist position and say that the mind and body interact. Psychological treatments affect the "mind" which in turn affects (causes changes) in the physical body. Also, physical treatments affect the body resulting in corresponding changes in the "mind."

The view adopted for this dissertation is the physical monist view, which says both types of treatments are effective because they are both dealing with the same thing, physical matter. The only difference between the two treatments is the level of intervention. Therefore, the

physical system can be affected at a biochemical level of intervention, or an environmental level of intervention resulting in corresponding changes physiologically. Changes in cognitions, feelings, etc., are changes in physiology, or physical matter (it is realized that this is merely speculation). This study primatively addressed this mind-body position by examining the effects of medication on dysfunctional beliefs, and the effects of cognitive therapy on the dexamethasone suppression test.

Statement of Purpose

The research presented here examined the value of the dexamethasone suppression test in selecting subjects who are responsive to different types of treatment for depression. In other words, the "treatment validity" of the dexamethasone suppression test was evaluated by testing whether the treatment of abnormal or normal dexamethasone suppression test responders was maximized by using either a biologically-oriented treatment (antidepressant medication) or a psychologically-oriented treatment (cognitive therapy), respectively. Specifically, the question examined was: Does an abnormal dexamethasone suppression test, which many believe represents a biologically-based depression, respond better to a biologically-oriented treatment (antidepressant therapy) versus a psychologically-oriented treatment (cognitive therapy), and conversely, does a normal dexamethasone suppression test, which many believe represents a psychologically-based depression, respond better to a psychologically-oriented treatment versus a biologically-oriented treatment? Related to this question, the theoretical distinction of

endogenous versus exogenous depression was explored in terms of its function in predicting behaviors associated with the distinction (presence or absence of melancholic symptoms) and its function in predicting treatment response. This project also examined a second question: If a conversion from an abnormal to normal dexamethasone suppression test after somatic treatment indicates clinical recovery, what effect does a psychological treatment (cognitive therapy) have on the dexamethasone suppression test? Finally, this study examined the question: What effect does a biologically-oriented treatment and a psychologically-oriented treatment have on a subject's dysfunctional thoughts?

The treatment validity design employed was observed differences with two or more treatments design. This design allowed the crossing of two distinct patient groups (normal versus abnormal dexamethasone suppression test results) with two distinct, effective treatment approaches (chemotherapy versus cognitive therapy). Therefore, not only was the treatment validity of the patient group distinctions tested, but also the theoretical distinctions associated with each patient group was examined (endogenous versus exogenous depressions).

Prediction of Outcome

The first set of predictions involved the effectiveness of the two treatments along with the differential effectiveness of the treatments based on whether the subjects were classified as normal or abnormal dexamethasone suppression test responders. First, it was predicted that overall, subjects in this study would report significantly less

depressive symptoms at post-intervention. This prediction was based on the body of research supporting the effectiveness of both cognitive therapy and tricyclic antidepressant medication as treatments for depression. Second, it was predicted that abnormal dexamethasone suppression test responders would report a significantly greater change in depressive symptoms after receiving antidepressant medication versus cognitive therapy, while normal dexamethasone suppression test responders would show significantly greater change in depressive symptoms after receiving cognitive therapy versus antidepressant medication. In other words, it was predicted that the "treatment validity" of the dexamethasone suppression test would be supported. This prediction was based on studies suggesting that abnormal results on the dexamethasone suppression test may represent a biological marker for depression which may respond well to biologically-oriented treatments (Brown et al., 1979; Brown & Shuey, 1979; Greden et al., 1980). Second, it was predicted that cognitive therapy would not impact on abnormal dexamethasone suppression test results, whereas antidepressant treatment would result in a significant reduction in dexamethasone suppression test scores for abnormal responders. This prediction was based on studies suggesting that an abnormal dexamethasone suppression test result represents an "endogenous" depression which is suggestive of a biological abnormality and treatable only by a biologically-oriented treatment (antidepressant medication), along with studies showing conversion from abnormal to normal results on the dexamethasone suppression test after administration of antidepressant medication (Bowie & Beaini, 1985; Brown et al., 1979; Brown & Shuey, 1979; Greden

et al., 1980; Fraser, 1975; Spar & Rue, 1983). Therefore, it was predicted that cognitive therapy would have no effect on the dexamethasone suppression test for abnormal responders. The final prediction was based on Beck's (1967, 1972, 1979) assertion that negative thought patterns and distorted perceptions that the depressed person holds about himself/herself, the present, and the future result in depression. Therefore, Beck's cognitive therapy of depression focuses directly on modifying dysfunctional thoughts and overt behavior in order to change the patient's negative self-perception and by doing so alleviating the patient's depression. Based on this rationale and the effectiveness of Beck's treatment for depression, it was predicted that cognitive therapy would have a significant impact on the subject's dysfunctional thoughts (i.e., significantly reduce the number of dysfunctional thoughts) while antidepressant medication would not.

CHAPTER II

METHOD

Subject Description and Selection Procedures

Subjects for this research-treatment project consisted of 43 depressed females who met certain criteria in a two-stage screening process. Subjects for this study were obtained by several means. First, area physicians were informed of this project so that they could give their patients the opportunity to participate in the study (see Appendix A). In addition, an article about the study was placed in a local university newspaper and in the local community newspaper (see Appendix B). Finally, various community organizations (e.g., churches, women's organizations) were notified of this project and sent a descriptive flyer (see Appendix C).

Women who were interested in outpatient treatment for depression were requested to telephone Charter Mandala Psychiatric Center to schedule a screening interview. Only subjects who displayed the following characteristics were eligible for the first screening stage. First, subjects were female, 18 years or older. Second, subjects signed a statement affirming that they were free of any tranquilizing drugs or antidepressant medication for a minimum of two weeks, and were not presently under a physician's, psychiatrist's, or psychologist's care for the treatment of depression (see Appendix D). However, if the subject were under care elsewhere for depression, she submitted a "physician's statement" (see Appendix E) stating that she had been

allowed to discontinue drug use and was appropriate for psychological-psychiatric treatment. Third, subjects were required to sign a consent form which described the initial screening interview and procedures (see Appendix F).

Screening stage one consisted of the following battery: (a) the Minnesota Multiphasic Personality Inventory (MMPI), Depression Scale (Hathaway & McKinley, 1942) (Note: The MMPI is not included in the Appendix due to its familiarity); (b) the Diagnostic and Statistical Manual of Mental Disorders, third edition (DSM-III, American Psychiatric Association, 1980) used in conjunction with The Schedule for Affective Disorders and Schizophrenia interview outline (SADS) (Endicott & Spitzer, 1978) (see Appendix G-1 and G-2); (c) the Beck Depression Inventory (Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961) (see Appendix H); and (d) the Lubin Depression Adjective Checklist (DACL, Lubin & Levitt, 1979) (see Appendix I). These measures are described in detail later in this section. Subjects met the criteria specified below for inclusion in this study: (1) MMPI (Minnesota Multiphasic Personality Inventory). T-score $\geq$ 70 on depression scale (equivalent to a raw score of 29). (2) DSM-III (Diagnostic and Statistical Manual of Mental Disorders, third edition). Depression categorized as Major Depressive Episode and was major presenting psychopathology. (3) Beck Depression Inventory. Score $\geq$ 20. (4) Lubin Depression Adjective Checklist. Score $\geq$ 18.

Finally, during this stage, subjects were questioned about any suicidal thoughts they may have had. Such questions were included in The Schedule for Affective Disorders and Schizophrenia interview outline

used with the Diagnostic and Statistical Manual of Mental Disorders (see Appendix G-1). In addition, each subject completed the Scale for Suicide Ideation (Appendix J) (Beck & Rush, 1978). If the subject scored eight or above on this scale, or stated that she was seriously considering suicide, she was not considered suitable for the study. If a subject did not meet the criteria outlined in this screening stage, the subject was given a list of appropriate referrals (see Appendix K) along with it being the staff psychiatrist's and principal investigator's full responsibility to assure that adequate care was received.

In total, 212 people inquired about participating in the investigation. Of these 212 inquiries, 115 participated in screening stage one. The remaining inquiries were disqualified over the telephone because of either violating the criteria (e.g., were on medicine, were seeing a psychiatrist/psychologist for care and were not willing to discontinue their treatment and/or medicine), were not interested, or were not willing to pay for the laboratory fees. In any case, all of these people were mailed a list of referrals, along with referrals made over the telephone. Of the 115 people who participated in screening stage one, 75 met the criteria for screening stage one. Of the 40 subjects not accepted at screening stage one, 21 did not obtain a raw score of 29 or greater (or a T score of 70 or greater) on the Depression Scale of the Minnesota Multiphasic Personality Inventory, 9 did not obtain a raw score of 20 or greater on the Beck Depression Inventory, 7 did not qualify based on the Diagnostic and Statistical Manual of Mental Disorders, third edition, either because they did not receive a

diagnosis of Major Depressive Disorder, or depression was not their major presenting pathology (e.g., one subject's major problem area was alcoholism with secondary depression), two did not qualify because of strong suicidal ideation, and one did not qualify due to not having a score of 18 or greater on the Lubin Depression Adjective Checklist. The remaining 75 subjects were scheduled for screening stage two.

If a subject met the criteria of screening stage one, she was asked to return for screening stage two. This stage included identifying subjects with the response class of irrational cognitions, which is assumed to be problematic for many individuals labelled "depressed." To accomplish this, the assessor administered the Personal Beliefs Inventory (Munoz & Lewinsohn, 1976) (see Appendix L). In addition, subjects were administered the Dexamethasone Suppression Test (Carroll, Martin, & Davies, 1968) in order to identify abnormal and normal responders. It should also be noted that the Beck Depression Inventory and the Lubin Depression Adjective Checklist were re-administered during screening stage two. These devices are described in greater detail in this section.

Criteria used in screening stage two for inclusion in this study were as follows: (1) Personal Beliefs Inventory. Score of three or greater. (2) Dexamethasone Suppression Test. Identification of subjects as <u>either</u> abnormal or normal responders. Prior to the treatment phase of the study, 21 subjects were chosen as abnormal responders, 25 as normal responders. Abnormal responders were defined as having a serum cortisol level greater than or equal to 5 ug/dl, while normal responders were defined as having a serum cortisol level less

than 5 ug/dl according to blood samples. There was a clear dichotomous split between abnormal and normal responders, with normal responders overall having an average dexamethasone suppression test score of .84 ug/dl (with a range of 0.0 to 3.5 ug/dl), while abnormal responders overall having an average dexamethasone suppression test score of 5.7 ug/dl (with a range of 5.1 to 6.1 ug/dl). (3) Beck Depression Inventory. Score $\geq$ 20. (4) Lubin Depression Adjective Checklist. Score $\geq$ 18.

Finally, subjects were interviewed individually by a staff psychiatrist at Charter Mandala Center (Dr. Jarrett Barnhill) in order to eliminate subjects showing any contradiction to the use of antidepressant medication. Only patients who were in sound physical health, lacked cardiovascular disease, and lacked any other metabolic and/or physiological disorders were included in the study. Pregnancy was an absolute contraindication both for the antidepressant medication and the dexamethasone suppression test. In addition, both the staff psychiatrist and principal investigator met with each subject individually to assess for suicidal ideation. This assessment was accomplished by interviewing the subject along with administering the Suicide Ideation Questionnaire (see Appendix J). The interview consisted mainly of questioning the subjects about any suicidal thoughts or intentions she may have had, and were approached by asking the subject if she had reached the point where life just did not seem worth living anymore, or if the subject ever thought she might be better off dead. Subjects indicating suicidal intent or ideation, along with subjects scoring eight or greater on the Suicide Ideation Questionnaire

(according to Beck & Rush, 1978, who developed the questionnaire) were dropped from the study and referred elsewhere. It was the staff psychiatrist's and principal investigator's full responsibility to assure that appropriate treatment was arranged, including hospitalization at Charter Mandala Center, if necessary.

Seventy-five subjects were scheduled for screening session two, with 71 subjects actually completing the screening. Four subjects cancelled because of personal reasons or they decided not to continue with the study. Nine subjects did not obtain scores of three or greater on the Personal Beliefs Inventory, two did not maintain their scores on the Beck Depression Inventory (20 or greater), one did not maintain her score on the Lubin Depression Adjective Checklist (18 or greater), two developed strong suicidal ideation, one was determined by the staff psychiatrist to be unable to take antidepressant medication because of medical reasons, and 10 were not accepted into the study because of the inability to accept additional normal dexamethasone suppression test responders into the study. All subjects who were excluded from the study for any reason were given appropriate explanations and referrals.

In summary, a total of 46 subjects were selected prior to treatment. All of the subjects were diagnosed as depressed and were exhibiting a high degree of irrational cognitions. Forty-three subjects completed the study, with 20 of the subjects exhibiting abnormal results on the Dexamethasone Suppression test and 23 subjects exhibiting normal results on the Dexamethasone Suppression test. Three subjects (two with normal dexamethasone suppression test results, both receiving medicine; one with abnormal dexamethasone suppression test results receiving

cognitive therapy) dropped out after treatment began. Two of the subjects developed complications with the medicine, while the third subject developed strong suicidal ideation which warranted treatment elsewhere. No subjects were dropped during treatment because of increases in their Lubin Depression Adjective Checklist scores. Subjects not meeting the criteria in screening stages one and/or two or who dropped out after treatment began, were given a list of appropriate referrals, including Charter Mandala Psychiatric Center, community mental health centers, and psychologists/ psychiatrists in private practice (see Appendix K). Again, it was the staff psychiatrist's and principal investigator's full responsibility to assure that appropriate treatment was arranged, and adequate precautions were taken.

Forty-three subjects completed the project without complication. These 43 female subjects had an average age of 37.5 years and ranged in age from 24 years to 63 years of age (see Table 1, Appendix M). Subjects' occupations included housewives, salespeople, students, nurses, secretaries, reservationists, managers, computer operators, and retired (see Table 1, Appendix M). Every marital status was represented, with many of the subjects having children. There seemed to be no differences between subjects in the different groups in terms of previous treatment or family history of affective illness (see Table 1, Appendix M).

Experimental Design

The design employed for this investigation was a two (abnormal or normal dexamethasone suppression test results) by two (cognitive therapy or antidepressant medication) by two (pre-post) factorial design (see

Table 2, Appendix M). In actuality, difference scores were used in the statistical analysis. This design allowed an examination of whether it was theoretically worthwhile to classify depressed clients using the dexamethasone suppression test, by manipulating the correspondence between the assessment results (abnormal or normal responders on the dexamethasone suppression test) and treatment (antidepressant medication or cognitive therapy). In other words, the "treatment validity" of the dexamethasone suppression test was examined.

Once subjects passed screening stages one and two and were clearly experiencing a Major Depressive episode along with exhibiting a great deal of cognitive distortion, they were divided as to whether they were abnormal or normal responders according to the dexamethasone suppression test. Once identified as normal or abnormal responders, subjects were randomly assigned to one of the two treatment conditions. Half of the abnormal responders and half of the normal responders received eight weeks of an antidepressant medication, while the other abnormal and normal responders received eight sessions of cognitive therapy. This resulted in half of the abnormal responders receiving a supposedly related treatment of medication, while the other half received a supposedly unrelated treatment of cognitive therapy. Likewise, half of the normal responders received a related treatment of cognitive therapy, while the other half received a supposedly unrelated treatment of medication (see Tables 2, 3, and 4, Appendix M).

The primary investigator conducted the cognitive therapy (and was not informed as to who were abnormal or normal responders) while medication was administered and monitored by the staff psychiatrist (who

was also blind to who were abnormal or normal responders). Further information concerning the treatments can be found later in this section.

Measurements taken during this study consisted of (a) pre-post global measures of depression, (b) pre-post measures of cognitions and the dexamethasone suppression test, (c) a mid-study and post measure of the amount of antidepressant medication, and (d) within-treatment session assessments. These are described below.

Pre-Post Global Assessment of Depression

The pre-post global assessment of depression consisted of the three measures taken during screening stage one. These three measures were repeated after treatment was completed. Both times, they were administered by the principal investigator. These measures were: (a) the Minnesota Multiphasic Personality Inventory (MMPI), (b) the Beck Depression Inventory, and (c) the Lubin Depression Adjective Checklist. In addition, subjects were diagnosed pre- and post-treatment using the Diagnostic and Statistical Manual of Mental Disorders, third edition.

The MMPI (Hathaway & McKinley, 1942) is one of the best known self-report measures for depression (Lewinsohn & Lee, 1981). The depression scale of the MMPI consists of 60 true-false MMPI items, with no specific time limits for completion. Lewinsohn and Lee (1981) report that the MMPI has both high reliability and validity. For inclusion in this study, the criteria for the depression scale required subjects to have a T-score of 70 or above (which is a raw score of 29, and two

standard deviations above the mean). According to the norms, the average score on the MMPI-D scale is 50 (or a raw score of 19).

The Beck Depression Inventory (see Appendix H) was also used as a pre-post global measure of depression. The Beck Depression Inventory consists of 21 items which assess numerous characteristics of depression. Each item is scored on a range of 0 to 3, with the total possible score ranging from 0 to 63. The larger the score, the more severe the depression. Subjects must have had a score equal to or greater than 20 at pre-treatment. Again, the Beck Depression Inventory has been shown to have high reliability and validity (Lewinsohn & Lee, 1981).

The Lubin Depression Adjective Checklist (see Appendix I) was also used as a pre-post global assessment of depression, and as a weekly session measure of depression (see section on within-treatment session assessments). The Lubin Depression Adjective Checklist (DACL) consists of seven parallel lists of adjectives designed to measure an individual's "mood" at a particular moment in time. The subject is asked to check the words which describe "How You Feel Now--Today." The number of positive adjectives not checked plus the number of negative adjectives checked constitutes the mood score. The higher the score, the more depressed the person is judged to be. Normative data available (Lubin & Levitt, 1979) indicate that for females the average score on the DACL is 8, with a standard deviation of 5. Of all the self-report depression scales, the Lubin Depression Adjective Checklist has been given the most extensive psychometric development and has the highest reliability and validity (Lewinsohn & Lee, 1981). Subjects that were

included in this study had DACL scores ranging from 18 upward (two standard deviations above the mean). The same scores are applicable to all seven lists of the DACL. Versions of the DACL used with each subject were selected randomly for both the pre-post measures and the session-by-session measures. However, the version used for a particular subject at pre-intervention (screening stage one) was also used at post-intervention assessment. It should be noted that the Lubin Depression Adjective Checklist and the Beck Depression Inventory were also given during screening stage two.

The Diagnostic and Statistical Manual for Mental Disorders, third edition (DSM-III) (American Psychiatric Association, 1980), was used with the interview outline provided by the Schedule of Affective Disorders and Schizophrenia (Endicott & Spitzer, 1978) (see Appendix G-1 and G-2). The DSM-III attempts to provide clear criteria (number and kinds of symptoms required, severity level, duration, differential diagnosis) and is very similar to the Research Diagnostic Criteria (RDC, Spitzer, Endicott, & Robins, 1978) which is known to be highly reliable (Lewinsohn & Lee, 1981). Field trials examining the reliability of the DSM-III for diagnosis of Major Affective Disorder revealed high reliability (Kappa coefficient = .80). The field trials did not differentiate specifically for the diagnosis of melancholia. Subjects were interviewed by the principal investigator using the Schedule of Affective Disorders and Schizophrenia (see Appendix G) and fell in the category of Major Depressive Episode according to the DSM-III (see Appendix G-2). Reliability in using the interview outline to complete the DSM-III diagnosis was taken by a licensed master's level

psychological associate at the end of the study (this was done by audio-taping (see Appendix N) the interview by the principal investigator). The reliability checker was blind to (a) which tapes were pre-post, (b) which treatment the subjects received, and (c) the subject's classification on the dexamethasone suppression test. Reliability was calculated by taking the number of diagnostic agreements divided by the number of diagnostic agreements and disagreements. The total number of tapes listened to by the agreement judge was 62 (41 pre-intervention tapes and 21 post-intervention tapes). This number included both subjects who were included and not included in this study. This high percentage was chosen because the principal investigator conducted both the interview phases and the cognitive therapy. The Diagnostic and Statistical Manual of Mental Disorders, third edition, was quantified by recording the frequency of subjects who were classified as depressed or not depressed. Inter-judge agreement was calculated to be .887.

It should also be mentioned that not only were subjects diagnosed as Major Depression, but also the principal investigator diagnosed subjects as with or without melancholia, based on the specific criteria presented in the DSM-III (see Appendix G-2 for criteria). Diagnoses are presented in Table 4, Appendix M. Reliability was again taken by the licensed master's level psychological associate under the same conditions as previously mentioned, and was recalculated by taking the number of diagnostic agreements divided by the number of diagnostic agreements and disagreements. Inter-judge agreement was calculated to be .806.

Pre-Post Assessment of Irrational Cognitions and the Dexamethasone Suppression Test

During screening stage two and again after the treatment ended, the response class of irrational cognitions was sampled, and the dexamethasone suppression test was administered.

Irrational Cognitions

The response class of irrational cognitions was assessed by the Personal Beliefs Inventory (Munoz & Lewinsohn, 1976) (see Appendix L). The Personal Beliefs Inventory consists of 30 items involving various irrational thoughts (similar to Albert Ellis' eleven irrational beliefs, Ellis & Grieger, 1977) that an individual may believe. Subjects rate their extent of agreement or disagreement with each item on a five-point scale, with a one indicating total disagreement, and a five indicating complete agreement. Subjects included in this study had an average score of three or greater.

Dexamethasone Suppression Test

All subjects included in this study were administered the Dexamethasone Suppression Test (Carroll et al., 1968). The dexamethasone suppression test measures cortisol levels in the blood stream by the use of a blood test. Abnormal results are produced by the non-suppression of cortisol in the blood stream, which is presumably indicative of endogenous depression. Normal results are produced by the suppression of cortisol in the blood stream, which is supposedly related to exogenous depression. Half of the subjects in this study were

selected for abnormal results; the other half were selected for normal results. Administration of this test consisted of the staff psychiatrist providing the subject with 1 mg. of dexamethasone that was ingested at 11:00 p.m., then having blood drawn at 4:00 p.m. the next afternoon at a local laboratory. Through the blood test, cortisol levels in the blood stream were determined. The results of the blood tests were sent to Charter Mandala Center, where they were interpreted by a staff psychologist who assigned subjects to groups without the knowledge of the experimenters.

Within-Treatment Session Assessments

At the beginning of each treatment session, all subjects were administered the Lubin Depression Adjective Checklist, which has already been described in detail. The purpose of administering this questionnaire during each treatment session was to monitor the subject's mood, particularly if it worsened. If a subject's score on this questionnaire worsened, that is, her score on this checklist increased (worsened) by five points relative to her original score (one standard deviation according to normative data), she was taken out of the study. This did not occur during the course of the study. Also, prior to each treatment session, all subjects met with both the principal investigator and the staff psychiatrist to assess the patient's condition and suicide risk. Suicide risk was assessed at the beginning of each session by (a) asking each subject if she were seriously entertaining the notion of suicide, and (b) having each subject complete the Scale for Suicide Ideation (Beck & Rush, 1978) (see Appendix J). If a subject scored

eight or above on this scale, or verbalized suicide intent, she was taken out of the study. One subject was excluded for this reason, after treatment was initiated. If a subject became suicidal, or her condition worsened, it was the principal investigator's and the staff psychiatrist's full responsibility to assure the subject received adequate care, including hospitalization at Charter Mandala Center, if necessary (see Appendix K).

Finally, subjects were required at treatment session four and at post-treatment to have a blood test taken in order to assure that the subjects receiving medication were receiving a therapeutic level of the medication, and that no subjects in the study were taking mood altering drugs. This was done at a local laboratory. The lab results are described later.

Treatments

Twenty-two of the subjects in this study received a cognitive therapy approach consisting of a strategy first employed by Beck (1967, 1972; Beck, Rush, Shaw, & Emery, 1979) which is similar to Ellis' rational-emotive therapy (RET) (Ellis & Greiger, 1977) through which negative thought patterns are restructured. Therapy consisted of (a) giving the subject the rationale for the therapy, (b) teaching the subject to self-monitor her own thought patterns, (c) teaching the subject to identify logical errors and depressive assumptions made, (d) helping the client evaluate and look for alternative explanations for negative thoughts, (e) teaching the subject to evaluate and correct dysfunctional thoughts and assumptions by coping with logical errors,

and (f) helping the subject to design "experiments" to test out her hypotheses and adjust dysfunctional thoughts and faulty assumptions accordingly. This treatment was modeled after the cognitive-behavioral treatment detailed in Cognitive Therapy of Depression by Beck and his associates (1979). Details of this treatment package can be found in Appendix O. Therapy was administered to subjects individually by the principal investigator for eight one-hour sessions. The cognitive therapy was individualized in terms of each subject's particular cognitive distortions and her related problems. The purpose of the treatment package was to provide a general framework to be used by the principal investigator in order to apply the cognitive therapy approach.

Twenty-one subjects received a chemotherapeutic approach consisting of the class of tricyclic antidepressants. The class of tricyclic medications is relatively safe as compared to other classes of antidepressant medications, such as the MAO inhibitors, which require strict diets (Blackwell, Marley, Price, & Taylor, 1967). Typical side-effects of this class of antidepressants consist mainly of dryness of mouth or slight dizziness if one stands up too quickly. The most serious but rare side-effects are constipation, palpitations, or postural hypotension, which necessitates stopping the medication (Asberg, Cronholm, Sjogvist, & Tuck, 1970). Medication in this study was administered and monitored by a qualified staff psychiatrist at Charter Mandala Center (Dr. Jarrett Barnhill). The pharmacotherapy involved eight weekly individual sessions in which the focus was medication and the biological approach to treating depression. These sessions varied in length (not to exceed one hour) depending on the

individual, and were conducted by the staff psychiatrist. These individual sessions were variable in length in order to more closely approximate clinical reality. The psychiatrist followed a general outline during the sessions that emphasized the medicine and the biological approach to depression. Patients receiving medication typically see a psychiatrist for medication checks and to ask questions about the medication, rather than receiving active psychotherapy. The staff psychiatrist also determined which tricyclic antidepressant to use based on the subject's symptoms. For example, if a depressed subject was experiencing anxiety, then she may have received Elavil (amitriptyline) or nortriptyline which have sedative effects. If a subject was experiencing a retarded-type depression, then imipramine or desipramine (norpramine) may have been used for their stimulant properties. The staff psychiatrist wrote prescriptions weekly to guard against overdoes. Thirteen subjects received Elavil (amitriptyline) while eight subjects received desipramine (norpramine). These medicines were evenly distributed across the two medication groups (Table 4, Appendix M). The starting dosages at treatment session one for the tricyclic antidepressants was 75 mg. at bedtime. For session two and three, the dosage was raised to 150 mg. daily. At session four, subjects had a blood test done at the local lab to assess whether the level of antidepressant medication in the blood stream was at the therapeutic plasma level of 125 to 250 ng/ml. At this stage, fifteen subjects had therapeutic plasma levels, while six did not (two in the normal dexamethasone suppression test-antidepressant group, four in the abnormal dexamethasone suppression test-antidepressant group). If it

was not, the dosage at session four was raised anywhere from 200 mg. daily to 250 mg. daily, depending on plasma levels. This dosage was maintained until session eight, wherein another blood sample was drawn to assure therapeutic levels of the medication. All subjects receiving either antidepressant medication at post-intervention had therapeutic plasma levels of 125 to 250 ng/ml according to the blood tests. The two subjects experiencing serious side-effects were dropped from the study and given appropriate referrals. (See section on Subject Withdrawal and Referral.)

Experimenters

The experimenters in this study consisted of a principal investigator, a Charter Mandala Center staff psychiatrist, and a Charter Mandala staff psychologist who was a reliability checker for the Diagnostic and Statistical Manual for Mental Disorders, third edition, along with interpreting the results from the dexamethasone suppression test.

The principal investigator, a sixth-year male graduate student in clinical psychology and a full-time therapist at Charter Mandala Center, was responsible for conducting screening stage one, conducting screening stage two in conjunction with the staff psychiatrist, administering the cognitive therapy treatment approach, administering post-treatment assessment, and debriefing subjects at the end of the study. The principal investigator was qualified in carrying out these responsibilities; he has experience in assessing and treating depression, is licensed as a Psychological Associate by the North

Carolina State Board of Examiners, has been trained in the treatment packages being utilized (research projects conducted at UNC-G, Charter Mandala Center), and has had experience in writing treatment packages. In addition, these activities occurred under the supervision of a faculty member in clinical psychology (Dr. Rosemery Nelson) and the staff psychiatrist (Dr. Jarrett Barnhill).

The Charter Mandala staff psychiatrist was responsible for evaluating subjects in screening stage two for possible medication use, administered and monitored all medication usage, and prescribed all laboratory tests conducted (i.e., administration of the dexamethasone suppression test, administration of blood tests to assess therapeutic levels of antidepressant medication prescribed). This person has been in psychiatry for seven years, is a full-time psychiatrist at Charter Mandala Center, and service chief of the 400 unit with twenty-one beds, and routinely conducts and administers the medications and laboratory work utilized in this project.

Reliability in using the Diagnostic and Statistical Manual of Mental Disorders, third edition, in conjunction with the interview outline provided by the Schedule of Affective Disorders and Schizophrenia was taken by a full-time master's level psychological associate at Charter Mandala Center at the end of this study. In addition, this person interpreted the results of the dexamethasone suppression test (which merely requires looking at the laboratory work with the cortisol levels on it) and assigned subjects to groups. This person has worked at Charter Mandala Center for six years and is licensed as a Psychological Associate by the North Carolina State Board

of Examiners, in addition to routinely utilizing the Diagnostic and Statistical Manual of Mental Disorders in her work along with interpreting the dexamethasone suppression test.

Procedure

Subjects were recruited by the various methods mentioned on the first page of this section. During screening stage one, the principal investigator met with potential subjects to explain the subject selection procedures and obtained subject's informed consent for participation (see Appendix F). Emphasis was placed on the fact that this was a screening process only, with the intention of correcting any misconceptions which could have occurred due to non-acceptance in the project.

Clients were also required to sign a statement affirming that they were free of any tranquilizing drugs or antidepressant medication for a minimum of two weeks and would remain free of any drugs for the duration of this project unless the medication was part of this project and were not under a physicians' or psychologists' care for the treatment of depression (see Appendix D). However, if the subjects were under the care of a physician for depression, she was given a "physician's statement" stating that she was allowed to discontinue drug use and was appropriate for psychological/psychiatric treatment (see Appendix E). One subject was included in this category, in which she was placed on an antidepressant (was only on it for three day) and obtained permission to discontinue it.

The principal investigator administered the Minnesota Multiphasic Personality Inventory, the Beck Depression Inventory, the Lubin Depression Adjective Checklist, and interviewed subjects and made a diagnosis based on the Diagnostic and Statistical Manual for Mental Disorders (third edition). Subjects were also questioned about suicide and administered the Scale for Suicide Ideation. Subjects who were eligible based on previously cited criteria were telephoned and scheduled for screening stage two. All subjects not meeting the criteria were referred to Charter Mandala Center, to mental health centers, and to private psychologists/psychiatrists (see Subject Withdrawal and Referral).

At screening stage two, the principal investigator and the staff psychiatrist reviewed the subject selection procedure (including costs totaling $106.00 for laboratory work, plus costs for medicine, and the $50.00 refundable deposit) and obtained informed consent (see Appendix P). Each subject was then administered the Personal Beliefs Inventory, the Beck Depression Inventory, and the Lubin Depression Adjective checklist, in addition to questioning subjects about suicide and administering the Scale for Suicide Ideation. Subjects meeting the previously cited criteria were then evaluated by the staff psychiatrist for the possibility of being placed on an antidepressant medication (e.g., obtained a medical history, assured that the subject was not at high-risk for taking antidepressant medication). Finally, subjects were administered the Dexamethasone Suppression Test. This was accomplished by the staff psychiatrist providing 1 mg. of dexamethasone to each subject to be taken at 11:00 p.m. Each subject then had blood

drawn at 4:00 p.m. the next day at a local laboratory. The laboratory results were sent to Charter Mandala Center where each subject was classified as abnormal or normal responders based on the previously mentioned criteria for screening stage two. The lab technicians were also blind to which subjects were assigned to which treatment. These results were seen only by the staff psychologist at Charter Mandala Center in order for her to assign subjects to groups, without the principal investigator or staff psychiatrist knowing who were abnormal or normal responders. Subjects who did not meet the previously cited criteria, who showed a contradiction to taking medication, or who refused to consider the possibility of taking medication were appropriately referred. Forty-three subjects completed the study, twenty being abnormal responders and twenty-three subjects being normal responders according to the dexamethasone suppression test.

The treatment contract was thoroughly explained to each subject accepted into the study (see Appendix Q). It should be noted here that an element of the treatment contract was the requirement of each subject to make a fifty dollar ($50.00) deposit. The subject was told that this amount would be refunded if she attended all eight treatment sessions and participated in the post assessment. The contract stated that if she failed to do any of the requirements above, a certain portion of her deposit would have been forfeited. In actuality, at the end of the study, the subject's deposit was returned regardless of performance. Therefore, the subjects that could not continue after the treatment phase were refunded their $50.00 deposit. The purpose of the mild deception was to increase the client's motivation for participating in

the project once she had signed the contract. This deposit was separate from the costs of the laboratory tests (e.g., three blood tests) and the cost of the medication, which was explained to the subjects.

The principal investigator met with 22 of the subjects individually in order to apply the treatment strategy of cognitive therapy. These subjects were seen for eight one-hour sessions. The cognitive therapy treatment strategy was preplanned, and broken down into eight steps (see Appendix O). The staff psychiatrist met with 21 of the subjects individually to administer and monitor the tricyclic antidepressant medication on a weekly basis. Medication was prescribed on a weekly basis to minimize the chances of overdose. At the beginning of each treatment session, all subjects were administered the Lubin Depression Adjective Checklist, were interviewed by both the principal investigator and the staff psychiatrist individually, in order to assess suicidal ideation, and were administered the Scale for Suicide Ideation. The purpose of this was to drop subjects from the study (and utilize appropriate referrals) who developed suicidal ideation or whose mood significantly worsened after the study began. Finally, all subjects were required at session four to have blood drawn in order to check the blood level of antidepressant medication (for those in the medication group) and to assure that no mood altering drugs were being taken.

After each subject completed the eight week treatment program, she again met with the principal investigator and staff psychiatrist. This post-treatment phase consisted of each subject receiving the following: (a) the questionnaires, Minnesota Multiphasic Personality Inventory, Beck Depression Inventory, Lubin Depression Adjective Checklist, and

Personal Beliefs Inventory; (b) a diagnosis based on the Diagnostic and Statistical Manual of Mental Disorders, third edition used in conjunction with the Schedule of Affective Disorders and Schizophrenia interview outline; (c) a blood test in order to evaluate the dexamethasone suppression test and evaluate levels of antidepressant medication for those receiving medication. After this was completed, the principal investigator then gave each subject a written and verbal debriefing of the project (see Appendix R), and offered continued treatment and appropriate referrals. The subject's deposit was then returned in full.

CHAPTER III

RESULTS

Overview

The results section is divided into four major sections. The first section examines the pre-post dependent measures of depression and is organized according to the following questions: Was there the predicted interaction between the classification of depressives on the dexamethasone suppression test and the effectiveness of the two treatments? Were the treatments effective, and was one treatment more effective overall than the other? And, finally, did one type of subject (e.g., abnormal vs. normal dexamethasone test responder) change more than the other type?

The second section examines the analysis of the pre-post dexamethasone suppression test, and asks the question: Did the dexamethasone suppression test results change over the course of treatment?

The third section describes the analysis of the pre-post Personal Beliefs Inventory scores, and examines the question of whether irrational beliefs changed during the course of the treatment.

Finally, the fourth section presents a post hoc analysis examining the presence or absence of melancholic symptoms.

Analysis of Pre-Post Dependent Measures of Depression

The data analyzed in this first section were collected at two measurement occasions--pre-intervention and post-intervention. Difference scores were calculated, and subjected to 2 (dexamethasone suppression test result) x 2 (treatment) multivariate and univariate analyses of variance. There were three measures of depression: the Depression Scale of the Minnesota Multiphasic Personality Inventory, the Beck Depression Inventory, and the Lubin Depression Adjective Checklist. Least squared means were utilized as post hoc tests.

In addition, a binoimal test was performed on the post-treatment diagnoses, based on the Diagnostic and Statistical Manual of Mental Disorders, third edition (American Psychiatric Association, 1980). The probability of subjects being diagnosed major depressive versus non-depressed at post-intervention was examined.

The results in this first section are organized into three questions: Was the predicted interaction between the dexamethasone suppression test and the two treatments significant? Were the two treatments effective, and was one treatment more effective overall than the others? And, finally, did one type of subject (e.g., abnormal vs. normal dexamethasone suppression test responder) change more than the other type?

Was the Interaction Significant?

Based on the 2 (abnormal vs. normal dexamethasone suppression test) x 2 (cognitive therapy vs. antidepressant medication) multivariate and univariate analyses of variance on the three depression measures, it was

predicted that there would be a significant interaction between the dexamethasone suppression test (abnormal vs. normal results) and the two treatments (cognitive therapy vs. antidepressant medication). More specifically, it was believed that these results would support the prediction that subjects with abnormal dexamethasone suppression test results would improve significantly more when administered antidepressant medication versus cognitive therapy, while subjects with normal dexamethasone suppression test results would improve significantly more when treated with cognitive therapy versus antidepressant medication.

The predicted interaction effect was not supported by either the multivariate analysis of variance or the analysis of variance. The 2 (dexamethasone suppression test result) x 2 (treatment) multivariate analysis of variance on the difference scores from the three depression measures (Depression Scale of the Minnesota Multiphasic Personality Inventory; the Beck Depression Inventory; and the Lubin Depression Adjective Checklist) revealed a nonsignificant dexamethasone suppression test results x treatment interaction with a Wilk's lambda of .904, which is equivalent to F (3, 37) = 1.31, p = .29 (Table 5). The 2 (dexamethasone suppression test result) x 2 (treatment) analysis of variance on the difference scores from the Depression Scale of the Minnesota Multiphasic Personality Inventory also showed a nonsignificant dexamethasone suppression test x treatment interaction, F (1, 39) = 3.31, p = .08 (Table 6, Appendix M). This interaction, however, is marginally significant, so an examination of the means is worthwhile. This examination revealed that the mean difference scores did not

clearly support the predicted interaction. Rather, the least squared means post hoc comparisons showed that the normal dexamethasone suppression test responder--cognitive therapy group improved significantly more than the abnormal dexamethasone suppression test responders--cognitive therapy group (p = .002, mean difference scores of 14.9 vs. 9.5 respectively) and also more than the abnormal dexamethasone suppression test responders--antidepressant medication group (p = .05, mean difference scores of 14.9 vs. 11.6 respectively). The other mean difference scores did not differ significantly from each other (Table 6, Appendix M). Similarly, nonsignificant dexamethasone suppression test x treatment interactions were also found on the Beck Depression Inventory, F (1, 39) = .00, p = .97 (Table 8, Appendix M), and for the Lubin Depression Adjective Checklist, F (1, 39) = .17, p = .68 (Table 10, Appendix M). The means for the various groups on the various measures are located in Tables 7, 9, and 11, Appendix M. Therefore, the prediction of significant interactions on these three depression measures were not supported.

Were the Treatments Effective, and Was One Treatment More Effective Overall Than the Other?

It was predicted that, overall, subjects in this study would significantly improve on the three depression measures from pre-intervention to post-intervention, with no significant differences between the two treatments. This prediction was made because of the research that supports the effectiveness of the two treatments in treating depression.

The predictions were supported on the multivariate analysis of variance and the univariate analyses of variance performed on the difference scores from the Depression Scale of the Minnesota Multiphasic Personality Inventory, the Beck Depression Inventory, and the Lubin Depression Adjective Checklist. The main effect for treatment on these measures was not significant, showing that the two treatments were not differentially effective. On the least squared means analyses, difference scores from both treatments differed significantly from zero, but did not differ significantly from each other. These results show that both treatments were effective, but not differentially so. These results are now described in greater detail.

Least squared means post hoc comparisons on the Depression Scale of the Minnesota Multiphasic Personality Inventory revealed that subjects in both the cognitive therapy and antidepressant medication groups significantly improved with regard to their depression pre-intervention to post-intervention ($p \leq .0001$), with no difference in effectiveness between the two treatments ($p = .97$). Mean difference score for the cognitive treatment was 12.2, and for the antidepressant group it was 12.1. The lack of differential effectiveness was also supported by the 2 (dexamethasone suppression test results) x 2 (treatment) analysis of variance on this measure which showed a nonsignificant effect for treatments, F (1, 39) = .05, $p = .82$ (Table 6, Appendix M).

Furthermore, least squared means post hoc comparisons showed that subjects in both the cognitive therapy and antidepressant medication groups significantly improved according to their depression pre-intervention to post-intervention based on the Beck Depression

Inventory ($p \leq .0001$) and the Lubin Depression Adjective Checklist ($p \leq .0001$), with no difference in effectiveness between the two treatments according to the Beck Depression Inventory ($p = .6205$), and the Lubin Depression Adjective Checklist ($p = .4859$). Mean difference scores on the Beck Depression Inventory were 17.7 for the cognitive therapy group and 16.5 for the antidepressant group, while the mean difference scores on the Lubin Depression Adjective Checklist was 10.8 for the cognitive therapy group and 12.1 for the antidepressant group. Again, the lack of differential treatment effectiveness was supported by the 2 (dexamethasone suppression test result) x 2 (treatment) analysis of variance performed on the difference scores from these two measures, with nonsignificant treatment effects being found on the Beck Depression Inventory, F (1, 39) = .30, $p = .59$, and on the Lubin Depression Adjective Checklist, F (1, 39) = .38, $p = .54$ (Tables 8 and 10, Appendix M). In addition, the above results on the three depression measures of no differential treatment effectiveness were supported by the 2 (dexamethasone suppression test result) x 2 (treatment) multivariate analysis of variance performed on the difference scores from three measures which showed a nonsignificant treatment effect with a Wilk's lambda of .969, which is equivalent to F (3, 37) = .39, $p = .76$ (Table 5, Appendix M). Therefore, based on analyses of these three depression measures, the predictions that subjects overall would significantly improve with regard to their depression, with no differential treatment effectiveness, was supported. Figures 1, 2, and 3, Appendix S also show this result.

Finally, the above prediction was also supported by the binomial test performed on the subjects' diagnoses. Based on the Diagnostic and Statistical Manual of Mental Disorders, third edition, using the Schedule for Affective Disorders and Schizophrenia interview outline, at pre-intervention, all 43 subjects were given the diagnosis of Major Depression. After receiving one of the two treatments offered (cognitive therapy or antidepressant medication), only six subjects at post-intervention continued to meet the criteria for a major depressive disorder according to the principal investigator's diagnoses. One of these subjects fell in the normal dexamethasone suppression test responder--cognitive therapy group, while three of these subjects fell in the abnormal dexamethasone suppression test responder--cognitive therapy group and two subjects fell in the abnormal dexamethasone suppression test responder--antidepressant medication group. The probability of obtaining six depressive diagnoses at post-intervention was compared to the expected probability of .50. The binomial test demonstrated that the probability of obtaining six depressive diagnoses after treatment by chance was $\leq .05$, therefore supporting the prediction that subjects would not be depressed after treatment. Pre-intervention and post-intervention diagnoses can be found in Table 4, Appendix M.

Did One Type of Subject (Abnormal vs. Normal Dexamethasone Suppression Test Responders) Change More Than the Other Type?

It has already been shown that the interaction between the dexamethasone suppression test (normal vs. abnormal responders) and the

treatments (cognitive therapy vs. antidepressant medication) proved to be nonsignificant. A remaining question is whether normal or abnormal dexamethasone suppression test responders responded differently to treatment, regardless of whether the treatment was cognitive therapy or antidepressant medication.

The least squared means analyses, along with the multivariate analysis of variance and the analysis of variance performed on the difference scores from the Depression Scale of the Minnesota Multiphasic Personality Inventory, the Beck Depression Inventory, and the Lubin Depression Adjective Checklist revealed that both normal and abnormal dexamethasone suppression test responders improved with therapy; but the normal responders consistently improved more than the abnormal responders. These results are now presented in detail.

Least squared means post hoc analysis on the Depression Scale of the Minnesota Multiphasic Personality Inventory revealed that while both normal ($p \leq .0001$) and abnormal ($p \leq .0001$) dexamethasone suppression test responders significantly improved on this measure, subjects with normal dexamethasone suppression test results improved significantly more on this measure than abnormal dexamethasone test responders ($p = .0085$) <u>regardless</u> of the type of treatment. Mean difference scores for the normal responders was 13.8, while for the abnormal responders, it was 10.5. In other words, subjects with abnormal dexamethasone test results did not do as well according to this measure in treatment (whether that treatment was antidepressant medication or cognitive therapy) as subjects with normal dexamethasone suppression test results. The differential effectiveness based on the

dexamethasone suppression test was also supported by the 2 (dexamethasone suppression test result) x 2 (treatment) analysis of variance which revealed a significant main effect for the dexamethasone suppression test on this measure, F (1, 39) = 7.90, $p \leq .01$ (Table 6). These results can also be seen on the graphed data, Figure 1, Appendix S.

Similar results were also found when the Beck Depression Inventory was examined. Least squared means post hoc comparisons also showed that both normal ($p \leq .0001$) and abnormal ($p \leq .0001$) dexamethasone suppression test responders significantly improved on this measure, with subjects having normal dexamethasone suppression test results improving significantly more on this measure than abnormal dexamethasone test responders ($p = .0252$). Mean difference scores for the normal responders was 19.9, while for the abnormal responders, it was 14.2. Again, the differential effectiveness based on the dexamethasone suppression test was supported by the 2 (dexamethasone suppression test result) x 2 (treatment) analysis of variance which revealed a significant main effect for the dexamethasone suppression test on this measure, F (1, 39) = 5.42, $p \leq .025$ (Table 8). These results can be seen on the graphed data, Figure 2, Appendix S.

The same results were also found when examining the Lubin Depression Adjective Checklist. Least squared means comparisons revealed again that both normal ($p \leq .0001$) and abnormal ($p \leq .0001$) dexamethasone test responders significantly improved on this measure, while subjects with normal test results improved significantly more on this measure than abnormal dexamethasone test responders ($p = .0061$).

Mean difference scores for normal responders was 14.2, while for abnormal responders, it was 8.8. As with the Depression Scale on the Minnesota Multiphasic Personality Inventory and the Beck Depression Inventory, based on the Lubin Depression Adjective Checklist, subjects with abnormal dexamethasone suppression test results did not do as well in treatment (whether the treatment was antidepressant medication or cognitive therapy) as subjects with normal dexamethasone suppression test results did. The 2 (dexamethasone suppression test result) x 2 (treatment) analysis of variance showed a significant main effect for the dexamethasone suppression test on this measure, $\underline{F}$ (1, 39) = 8.46, $\underline{p}$ = .006 (Table 10). These results can be seen on the graphed data, Figure 3, Appendix S.

Finally, the above results according to the Depression Scale of the Minnesota Multiphasic Personality Inventory, the Beck Depression Inventory, and the Lubin Depression Adjective Checklist were supported by the 2 (dexamethasone suppression test result) x 2 (treatment) multivariate analysis of variance which showed a significant main effect for the dexamethasone suppression test results with a Wilk's lambda of .747, which is equivalent to $\underline{F}$ (3, 37) = 4.17, $\underline{p} \leq .01$ (Table 5, Appendix M).

In conclusion, the predictions based on the three depression measures utilized in this study that said subjects with abnormal dexamethasone suppression test results would improve significantly more when administered antidepressant medication versus cognitive therapy while normal responders would improve significantly more when treated with cognitive therapy rather than antidepressant medication were not

supported. Rather, calculated difference scores on the three depression measures subjected to 2 (dexamethasone suppression test results) x 2 (treatment) multivariate and univariate analysis of variance along with least squared means post hoc tests and the graphed data revealed that while both normal and abnormal dexamethasone suppression test responders significantly improved on these measures, subjects with normal dexamethasone suppression test results improved significantly more on these measures than abnormal dexamethasone test responders, regardless of treatment.

Analysis of Pre-Post Dexamethasone Suppression Test Scores--
Did the Dexamethasone Suppression Test Scores Change?

This question was important to examine due to the fact that if the "treatment validity" of the dexamethasone suppression test was to be supported, then one would expect that scores in the abnormal range on the dexamethasone suppression test would be reduced only after the administration of an antidepressant medication rather than cognitive therapy. As far as normal scores on the dexamethasone suppression test, no change would be expected. In spite of the fact that the interaction between the dexamethasone suppression test and the treatments was not significant, the question of whether a psychologically-oriented treatment (cognitive therapy) can effect improvement on an abnormal biological system as represented by abnormal dexamethasone suppression test responders was interesting to examine.

A one way analysis of variance was performed on the pre-post difference scores from the dexamethasone suppression test, with the

analysis of variance first performed on subjects who had normal dexamethasone suppression test results at pre-intervention, then again with subjects who had abnormal dexamethasone suppression test results at pre-intervention. The between-subject factor was the two treatments (antidepressant medication versus cognitive therapy). Least squared means were utilized as post hoc tests.

For the analysis of variance on the subjects who had normal dexamethasone suppression test results at pre-intervention, it was predicted that there would be no significant change from pre-intervention to post-intervention on the dexamethasone suppression test nor would the two treatments differentially affect the dexamethasone suppression test difference scores. In other words, it was predicted that subjects with normal dexamethasone suppression test results at pre-intervention would maintain the normal dexamethasone suppression test results at post-intervention, regardless if the subjects received cognitive therapy or antidepressant medication.

The results from the 2 (treatment) one way analysis of variance on the difference scores from the dexamethasone suppression test for subjects who had normal dexamethasone suppression test results at pre-intervention showed a nonsignificant effect for treatments, $F = (1, 21) = .22$, $p = .64$ (Table 14, Appendix M). Least squared means post hoc comparisons revealed that there were no significant changes from pre-intervention to post-intervention for subjects receiving cognitive therapy ($p = .93$) or subjects receiving antidepressant medication ($p \leq .47$), and there were no significant differences between subjects receiving cognitive therapy or antidepressant medication on the

dexamethasone suppression test at post intervention (p = .64). Mean difference scores for the cognitive therapy group was .02, while for the antidepressant group it was .24. Therefore, the predictions were supported in that for subjects with pre-intervention normal dexamethasone suppression test results there were no significant changes in the dexamethasone suppression test at post-intervention, and the difference scores were not significantly different between subjects who received cognitive therapy or antidepressant medication. Examination of Figure 5 also confirms these results. Inspection of the individual data (Table 4, Appendix M) shows that all subjects remained in the normal range ($\leq$ 5 ng/dl) according to the dexamethasone suppression test.

For the analysis of variance on the subjects who had abnormal dexamethasone suppression test results at pre-intervention, it was predicted that there would be a significant change from pre-intervention to post-intervention on the dexamethasone suppression test for those subjects who received antidepressant medication and not for those subjects who received cognitive therapy. Therefore, a treatment effect was predicted, with the belief that the "treatment validity" of the dexamethasone suppression test would be supported.

The results from the 2 (treatment) one way analysis of variance on the difference scores from the dexamethasone suppression test for subjects who had abnormal dexamethasone suppression test results at post-intervention revealed a nonsignificant effect for treatments, F (1, 18) = .58, p = .45 (Table 15, Appendix M). Least squared means post hoc comparisons showed that there were significant changes from pre-intervention to post-intervention for subjects receiving cognitive

therapy ($p \leq .02$) along with significant changes from pre-intervention to post-intervention for subjects receiving antidepressant medication ($p = .0024$). In addition, there were no significant differences between subjects receiving cognitive therapy or antidepressant medication on the dexamethasone suppression test at post-intervention ($p = .46$). Mean difference scores for the cognitive therapy group was 1.3, while for the antidepressant group it was 1.8 (Table 15, Appendix M). These results did not support the predictions that there would be a significant effect for treatments with a significant (reduction) from pre-intervention to post-intervention on the dexamethasone suppression test for only subjects receiving antidepressant medication and not for those receiving cognitive therapy. Rather, a significant reduction in the dexamethasone suppression test was found for subjects who received either cognitive therapy or antidepressant medication, with no significant differences between the two groups at post intervention. Figure 5 confirms this. Inspection of the individual data (Table 4, Appendix M) showed that for subjects receiving cognitive therapy with initial abnormal dexamethasone suppression test results (totalling 10 subjects), at post-intervention five subjects had dexamethasone suppression test results in the normal range. For subjects receiving antidepressant medication with initial abnormal test results (totalling 10 subjects), at post-intervention five subjects also had normal dexamethasone suppression test results. Therefore, the "treatment validity" of the dexamethasone suppression test was not supported.

Analysis of Pre-Post Cognitive Questionnaire-- Did the Irrational Beliefs Change?

A 2 (dexamethasone suppression test) x 2 (treatment) analysis of variance was performed on the pre-post difference scores obtained from the Personal Beliefs Inventory. The between-subject factors were the treatments (cognitive therapy versus antidepressant medication) and the pre-treatment results from the dexamethasone suppression test (normal versus abnormal results). Least squared means were utilized as post hoc tests.

It was predicted that there would be a significant main effect for treatment with subjects (regardless of the initial dexamethasone suppression test result) receiving cognitive therapy having significantly larger pre- to post-intervention difference scores on this measure than subjects receiving antidepressant medication. This is based on the rationale that since all subjects exhibited a high level of irrational beliefs at pre-intervention based on this high measure, only those receiving cognitive therapy would exhibit any change on this measure.

The results from the analysis of variance on the difference scores from the Personal Beliefs Inventory revealed a significant main effect for treatments, F (1, 39) = 6.37, p = .01 (Table 12, Appendix M). The difference scores was greater for subjects receiving cognitive therapy (x = .65) than drug therapy (x = .31). The main effect for the dexamethasone suppression test F (1, 39) = 2.11, p = .154, and the treatment x dexamethasone suppression test interaction F (1, 39) = .94, p = .339 were not significant (Table 12, Appendix M). Examination of

the least squared means showed that subjects (regardless of dexamethasone suppression test results) who received cognitive therapy (p = .0001) or antidepressant medication (p = .0024) significantly improved on this measure. In addition, subjects improved significantly more on this measure (p = .0158) after receiving cognitive therapy rather than antidepressant medication. While the cognitive therapy had a significantly greater effect on this measure as predicted, the significant improvement on this measure for subjects who received antidepressant medication was unexpected. Inspection of the individual data (Table 3, Appendix M) showed that for the 22 subjects receiving cognitive therapy, 17 had normal scores on the Personal Beliefs Inventory ($\leq$ 3.0) at post-intervention, while for the 21 subjects receiving antidepressant medication, 11 had normal results at post-intervention. These results can be examined on the graphed data found in Figure 4, Appendix S.

Post Hoc Analysis of Diagnoses Using With or Without Melancholia

When the principal investigator made the initial diagnoses utilizing the Diagnostic and Statistical Manual of Mental Disorders, third edition, it was recorded whether there was a presence or absence of melancholic symptoms (see Appendix G-2 and Table 4, Appendix M). While most studies do not record the presence or absence of melancholic symptoms, it was decided that it would be important to examine if melancholic symptoms covaried with results from the dexamethasone suppression test, and if dividing subjects based on the presence or

absence of melancholic symptoms would reveal the same results as subjects divided based on normal or abnormal dexamethasone suppression test results. Therefore, a point-biserial correlation was utilized post hoc to examine if melancholic symptoms correlated with dexamethasone suppression test results. In addition, 2 (presence or absence of melancholia) x 2 (treatment) multivariate and univariate analyses of variance utilizing difference scores were performed on the Depression Scale of the Minnesota Multiphasic Personality Inventory, the Beck Depression Inventory, and the Lubin Depression Adjective Checklist, along with a 2 (presence or absence of melancholia) x 2 (treatment) analysis of variance on the difference scores from the Personal Beliefs Inventory, and a one-way analysis of variance (treatments) using difference scores from the dexamethasone suppression test being performed first on subjects without melancholic symptoms, then again for subjects with melancholic symptoms. The number of subjects in each group was as follows: 9 in the with melancholia-cognitive therapy group; 13 in the without melancholia-cognitive therapy group; 7 in the with melancholia- antidepressant group; and 14 in the without melancholia-antidepressant group.

A point-biserial correlation was performed between abnormal and normal dexamethasone suppression test results and the presence or absence of pre-intervention melancholic symptoms (see Table 4, Appendix M). The point-biserial correlation allows one to correlate data where one variable is continuous and one variable is dichotomous. The correlation between abnormal responders and melancholic symptoms was

.74. Therefore, abnormal dexamethasone suppression test results correlated highly with the presence of melancholic symptoms.

The 2 (presence or absence of melancholic symptoms) x 2 (treatment) multivariate and univariate analyses of variance on the difference scores from the depression measures (Depression Scale of the Minnesota Multiphasic Personality Inventory, Beck Depression Inventory, and the Lubin Depression Adjective Checklist), using least squared means as post hoc tests, revealed a nonsignificant treatment effect and nonsignificant treatment x melancholia interaction according to the multivariate and univariate analyses on these depression measures, while the presence or absence of melancholia effect was significant (Tables 17, 18, 19, and 20, Appendix M). Least squared post hoc tests showed that overall, subjects with or without melancholic symptoms significantly improved with regard to their depressive symptoms, regardless of treatment. Furthermore, subjects without melancholia improved significantly more on these measures than subjects with melancholia (Tables 17, 18, 19, and 20, Appendix M). Therefore, the results obtained by dividing subjects based on the presence or absence of melancholic symptoms match the results and conclusions arrived at by dividing subjects on the basis of normal or abnormal dexamethasone suppression test results. In other words, the "treatment validity" of dividing subjects according to the presence or absence of melancholia was not supported.

The 2 (presence or absence of melancholia) x 2 (treatment) univariate analysis of variance on difference scores from the Personal Beliefs Inventory (Table 21, Appendix M) revealed a significant effect for treatments, along with a nonsignificant effect for melancholia

(presence or absence) and a nonsignificant effect for the treatment x melancholia (presence or absence) interaction. Examination of least squared means showed that subjects (regardless of the presence or absence of melancholia) who received cognitive therapy or antidepressant medication significantly improved on this measure, with subjects improving significantly more after receiving cognitive therapy rather than antidepressant medication (Table 21, Appendix M). Again, these results parallel the results obtained when subjects were divided based on the dexamethasone suppression test.

Finally, the one-way (treatment) analysis of variance using difference scores from the dexamethasone suppression test for subjects without melancholia showed a nonsignificant effect for treatments (Table 22, Appendix M). Least squared means showed no significant changes pre-intervention to post-intervention on this measure for subjects without melancholia (Table 22, Appendix M). Since there was a high correlation between normal dexamethasone suppression test results and the absence of melancholia, this finding is not surprising.

For the one-way (treatment) analysis of variance using difference scores from the dexamethasone suppression test for subjects with melancholia, this showed a nonsignificant effect for treatments (Table 23, Appendix M). Least squared means revealed that there were significant changes from pre-intervention to post-intervention on the dexamethasone suppression test for subjects receiving either cognitive therapy or antidepressant medication (Table 23, Appendix M). Again, this is not surprising in light of the high correlation between the presence of melancholic symptoms and abnormal dexamethasone suppression test results.

CHAPTER IV

DISCUSSION

The research presented here examined the value of the dexamethasone suppression test in selecting subjects who are responsive to different types of treatment for depression. In other words, the "treatment validity" of the dexamethasone suppression test was evaluated by testing whether the treatment of abnormal or normal dexamethasone suppression test responders was maximized by using either a biologically-oriented treatment (antidepressant medication) or a psychologically-oriented treatment (cognitive therapy), respectively. Specifically, the question examined was: Does an abnormal dexamethasone suppression test, which many believe represents a biologically-based depression, respond better to a biologically-oriented treatment (antidepressant therapy) versus a psychologically-oriented treatment (cognitive therapy), and conversely, does a normal dexamethasone suppression test, which many believe represents a psychologically-based depression, respond better to a psychologically-oriented treatment versus a biologically-oriented treatment? Related to this question, the theoretical distinction of endogenous versus exogenous depression was explored in terms of its function in predicting behaviors associated with the distinction (presence or absence of melancholic symptoms) and its function in predicting treatment response. This project also examined a second question: If a conversion from an abnormal to normal dexamethasone suppression test after somatic treatment indicates clinical recovery,

what effect does a psychological treatment (cognitive therapy) have on the dexamethasone suppression test? Finally, this study examined the question: What effect does a biologically-oriented treatment and a psychologically-oriented treatment have on a subject's dysfunctional thoughts?

In short, the results showed that for both treatments, subjects overall reported significantly less depressive symptoms according to global measures of depression from the Depression Scale of the Minnesota Multiphasic Personality Inventory, the Beck Depression Inventory, the Lubin Depression Adjective Checklist, and diagnoses based on the Diagnostic and Statistical Manual of Mental Disorders, third edition. These measures also showed that normal dexamethasone suppression test responders reported a significantly greater change in depressive symptoms at post-intervention than abnormal dexamethasone suppression test responders, regardless of type of treatment. Furthermore, while normal dexamethasone suppression test responders showed no significant change on the dexamethasone suppression test from pre-intervention to post-intervention, the abnormal dexamethasone suppression test responders did show significant reductions in the dexamethasone suppression test (indicating improvement) from pre-intervention to post-intervention after receiving either antidepressant medication or cognitive therapy, with no difference at post-intervention between the two types of treatment (50% were "normal" responders at post-intervention). Finally, there was a significant reduction overall in depressives' dysfunctional thoughts (according to the Personal Beliefs Inventory) after receiving either antidepressant medication or

cognitive therapy, with subjects receiving cognitive therapy having significantly fewer dysfunctional thoughts than subjects receiving antidepressant medication.

The preceding pattern of results raised the following questions: (a) What is the meaning of the fact that the "treatment validity" of the dexamethasone suppression test was not supported?; (b) Why did normal dexamethasone suppression test responders report a significantly greater change in depressive symptoms at post-intervention than abnormal dexamethasone suppression test responders, regardless of the treatment utilized?; (c) Why did a psychological treatment result in significant reductions (improvement) on dexamethasone suppression test scores?; (d) How did a biologically-oriented treatment result in a significant reduction in depressives' dysfunctional thoughts? As these questions are being discussed, the research findings are compared to initial predictions and past research.

Predictions, Results, and Discussion

Overall Treatment Effectiveness and Differential Effectiveness Based on Dexamethasone Suppression Test in Treating Depression

The first set of predictions involved the effectiveness of the two treatments along with the differential effectiveness of the treatments based on whether the subjects were classified as normal or abnormal dexamethasone suppression test responders. First, it was predicted that overall, subjects in this study would report significantly less

depressive symptoms at post-intervention. This prediction was based on the body of research supporting the effectiveness of both cognitive therapy and tricyclic antidepressant medication as treatments for depression. Second, it was predicted that abnormal dexamethasone suppression test responders would report a significantly greater change in depressive symptoms after receiving antidepressant medication versus cognitive therapy, while normal dexamethasone suppression test responders would show significantly greater change in depressive symptoms after receiving cognitive therapy versus antidepressant medication. In other words, it was predicted that the "treatment validity" of the dexamethasone suppression test would be supported. This prediction was based on studies suggesting that abnormal results on the dexamethasone suppression test may represent a biological marker for depression which may respond well to biologically-oriented treatments (Brown et al., 1979; Brown & Shuey, 1979; Greden et al., 1980).

Overall effectiveness of cognitive therapy and antidepressant medication. The present data are consistent with the demonstrated effectiveness of both cognitive therapy and tricyclic antidepressants in the treatment of depression. That is, based on the present investigation, subjects overall reported less depressive symptoms according to all global measures of depression, the Depression Scale of the Minnesota Multiphasic Personality Inventory, the Beck Depression Inventory, the Lubin Depression Adjective Checklist, and diagnoses based on the Diagnostic and Statistical Manual of Mental Disorders, third edition, after being exposed to either cognitive therapy or tricyclic

antidepressants. These data parallel past studies which support the effectiveness of cognitive therapy (Blackburn et al., 1981; Kendall, 1984; Kovacs et al., 1981; Lewinsohn & Hoberman, 1981; McLean & Hakstain, 1979; Rush et al., 1977; Shaw, 1977; Wright & Beck, 1983) and the effectiveness of tricyclic antidepressants (Bennett, 1966; Klein & Davis, 1969; Mandel & Klerman, 1979; Mindham, 1982; Morris & Beck, 1973; Wechsler et al., 1965). The fact that neither treatment was significantly more effective than the other is consistent with the conclusion from other studies (Blackburn et al., 1981; Kendall, 1984; Kovacs et al., 1981; Lewinsohn & Hoberman, 1981; McLean & Hakstain, 1979; Rush et al., 1977; Wright & Beck, 1983). Wright and Beck (1983) concluded that, "Cognitive therapy has been at least equal to pharmacotherapy in all studies comparing the two treatments."

It should be mentioned that while this study cannot assert that the two treatments utilized in this study produce results that are superior to control conditions, other studies strongly show that both tricyclic antidepressants and cognitive therapy produce superior results to control conditions (Bennett, 1966; Blackburn et al., 1981; Kendall, 1984; Klein & Davis, 1969; Kovacs et al., 1981; Lewinsohn & Hoberman, 1981; Mandel & Klerman, 1979; McLean & Hakstain, 1979; Mindham, 1982; Morris & Beck, 1973; Rush et al., 1977; Shaw, 1977; Wechsler et al., 1965; Wright & Beck, 1983). To illustrate, Figure 6 (Appendix S) contains data utilizing the Beck Depression Inventory and shows the relative effectiveness of cognitive therapy and antidepressant medication over control groups. The top graph in Figure 6 (Appendix S) contains data from Shaw (1977) and Rush et al. (1977) and shows, based

on the Beck Depression Inventory, that both cognitive therapy and antidepressant medication are significantly more effective in reducing self-reported depression over control conditions, and are comparable in effectiveness. The bottom graph in Figure 6 (Appendix S) is data from Simons et al. (1984) and shows virtually identical results in that based on the Beck Depression Inventory, both cognitive therapy and antidepressant medication are comparable in effectiveness, and are significantly more effective over control conditions. There are no studies available comparing antidepressant medication to a placebo medication that utilizes the Beck Depression Inventory. Because of these consistent findings, the inclusion of a waiting-list control group in this particular investigation was deemed both unnecessary and unethical.

Differential Effectiveness of Treatments Based on Dexamethasone Suppression Test Results

Treatment validity of the dexamethasone suppression test. The prediction was not supported that the "treatment validity" of the dexamethasone suppression test would be shown with abnormal responders reporting significantly less depressive symptoms after antidepressant medication rather than cognitive therapy, and normal responders reporting less depressive symptoms after cognitive therapy versus antidepressant medication. Rather, analysis of all three global measures of depression (Depression Scale of the Minnesota Multiphasic Personality Inventory; Beck Depression Inventory; and Lubin Depression Adjective Checklist) showed that regardless of type of treatment, normal

dexamethasone suppression test responders overall reported a significantly greater reduction in depressive symptoms than abnormal dexamethasone suppression test responders at post-intervention. Stated differently, while subjects overall significantly improved pre-intervention to post-intervention on the depression measures after being exposed to either antidepressant medication or cognitive therapy, normal dexamethasone suppression test responders responded significantly better to the treatment of their depression (whether the treatment was medication or cognitive therapy) than abnormal dexamethasone suppression test responders. Therefore, the use of the dexamethasone suppression test in making treatment decisions (in this case, between a biologically versus psychologically-oriented treatment) in order to maximize treatment outcome was not supported. The predicted treatment validity of the dexamethasone suppression test came from the fact that many researchers operating from the biological-disease model of depression have attempted to operationalize the endogenous-exogenous distinction, through the dexamethasone suppression test. Concomitantly, abnormal dexamethasone suppression test results were taken to represent an endogenous or biologically-based depression that responds well to somatic-oriented treatments, while normal dexamethasone suppression test results were taken to represent an exogenous-based depression that responds well to psychologically-oriented treatments. In fact, many of these researchers have presented data suggesting that abnormal dexamethasone suppression test responders show more improvement after receiving tricyclic antidepressants than normal responders (Brown et al., 1979; Brown & Shuey, 1979; Carroll, 1982; Fiori & Davis, 1984;

Fraser, 1975; Greden et al., 1980; Gwirtsman, Gerner, & Sternbach, 1982; Nemeroff & Evans, 1984) while others have questioned whether cognitive therapy can be effective for abnormal dexamethasone suppression test responders (Carroll, Feinberg, & Greden, 1981; Rush, 1982; Williams, 1984). These studies have been plagued by methodological difficulties such as small sample sizes, poor outcome measures, poor diagnostic procedures, and post hoc examination of treatment response. The present study did not support the views that abnormal responders on the dexamethasone suppression test should improve significantly more with antidepressant medication than normal responders, or that cognitive therapy should be ineffective for abnormal responders. The present study is the first to date that examines directly the effects of two different treatments (antidepressant medication versus cognitive therapy) based on the classification of subjects according to their responses to the dexamethasone suppression test. Other studies, however, have not supported the relationship between abnormal responders and significant improvement with antidepressant medication as compared to normal responders (Bowie & Beaini, 1985; Hirschfeld et al., 1983; Insel & Goodwin, 1983; Klein, Bender, & Mayr, 1984; Spar & Rue, 1983). This present study was different from other studies in that it showed that significant improvement in terms of depression for both normal and abnormal responders on the dexamethasone suppression test could result from receiving either antidepressant medication or cognitive therapy, with normal responders improving significantly more with regard to their depressive symptoms relative to abnormal responders.

There are several possible reasons why the prediction was not supported. First, the dexamethasone suppression test may not make the

endogenous-exogenous distinction. In other words, the dexamethasone suppression test is an indirect measure of hypothalamic-pituatary-adrenal functioning which is believed to be directly related to the neurotransmitters implicated in depression (e.g., serotonin, dopamine). It may be that the dexamethasone suppression test does not actually measure this system. Second, it may be that the dexamethasone suppression test is unreliable in and of itself. It may be, as Bowie & Beaini (1985) suggest, that there is a class of dexamethasone suppression test responders who naturally fluctuate over time, making it difficult to draw conclusions from only pre-post measures of cortisol. Third, it may be that the endogenous-exogenous distinction functionally does not distinguish between treatments. It may be that the distinction is useful in serving other functions (e.g., predicting behaviors associated with the depressive episode, predicting course of treatment), but is useless in treatment selection. Finally, it may be that treatment choice is not dependent on etiology. Even when the etiology of a disorder is known, different treatments unrelated to the etiology might none-the-less be effective. A simple example of this is known as "the aspirin analogy" (Rimland, 1964). Aspirin alleviates a headache, but the substances in aspirin may be totally unrelated to the etiology of the headache. This last position is viable in light of the theoretical argument involving physical monism, which is discussed in detail later.

Interpretation of differential effectiveness of treatments based on dexamethasone suppression test results. This investigation showed that

overall subjects significantly improved on the various depression measures, with normal responders improving significantly more than abnormal responders on the dexamethasone suppression test regardless of type of treatment. These results support the growing body of literature suggesting that the dexamethasone suppression test may have prognostic value, and that normalization of the test (abnormal to normal results) parallels clinical improvement (Bowie & Beaini, 1985; Greden et al., 1980; Hirschfeld et al., 1983; Insel & Goodwin, 1983; Nemeroff & Evans, 1984; Spar & Rue, 1983). In addition, one author found that failure to convert from an abnormal to normal responder after treatment resulted in significant chances for relapse (Nemeroff & Evans, 1984).

The prognostic value of the dexamethasone suppression test was supported in this study by the fact that a normal response predicted better outcome than an abnormal response. These results are consistent with a recent study by Spar and Rue (1983) which showed that for elderly patients (age $\geq$ 65), both normal and abnormal responders significantly improved on a number of measures (e.g., depression, cognitive impairment, agitation), with normal responders improving significantly more on these measures than abnormal responders. Therefore, abnormal responders may have a more intractable depression, requiring a longer period of treatment. Another explanation may be that there are a number of "systems" that can be affected during a depressive episode (e.g., biochemical, cognitive, behavioral systems). The abnormal responders in this study had at least two systems affected, the biochemical and the cognitive (all subjects had a high level of dysfunctional beliefs), according to the measures taken. It may require greater time to show

improvements in two systems. Relatedly, two types of treatment may be needed to affect two systems. Presently, only one system was treated (half the abnormal responders received cognitive therapy, the other half antidepressant medication).

This study also supports the research that suggests normalization of the dexamethasone suppression test parallels clinical improvement. Abnormal responders showed significant improvement in terms of their depression along with significant decreases with regard to their cortisol levels on the dexamethasone suppression test. This result can also be seen by examining the individual data (see Tables 3 and 4, Appendix M). (This finding is explored in detail in the next section.) This is consistent with a recent study performed by Bowie and Beaini (1985) that examined serial dexamethasone suppression test results; decreases of serum cortisol on the test along with normalization of the test were highly correlated with clinical recovery. Therefore, as Klein et al. (1984) concluded, the dexamethasone suppression test may be much more useful as a measurement of the "present state" of a patient's depression rather than as a diagnostic tool. This would also suggest that, if there is an incomplete reduction in the dexamethasone suppression test and in depressive symptomology, it would be important to continue treatment until full clinical recovery is achieved.

The dexamethasone suppression test and melancholic symptoms. Many researchers utilizing the endogenous-exogenous distinction based on the dexamethasone suppression test assert that abnormal dexamethasone suppression test results correlate with melancholia. The rationale is

based on the fact that the dexamethasone suppression test indirectly measures hypothalamus functioning and that many of the melancholic symptoms are suggestive of hypothalamic dysfunction (e.g., disturbances in sex drive, sleep, appetite, autonomic activity). Since many of the neurotransmitters implicated in the chemical pathology of depression (e.g., nonadrenaline, serotonin, acetylcholine) also regulate the hypothalamic function, deficiencies in the functional activity of these neurotransmitters would be reflected in the hormonal responses they regulate. Therefore, one would expect high correlations between abnormalities in cortisol secretion based on the dexamethasone suppression test and melancholic symptoms. Several studies have found high correlations between abnormal dexamethasone suppression test results and melancholia (Brown et al., 1979; Calloway, Fonagy, DeSouza, & Wakeling, 1984; Carroll et al., 1981; Johnson, Hunt, Kerr, & Catersan, 1984; Sachar, 1975; Zung, Mahorney, & Davidson, 1984) while others have not found a correlation (Beeber, Kline, Pies, & Manring, 1984; Coryell, Gaffney, & Burkhardt, 1982; Morphy, Fava, Perini, Molnar, Zielezny, & Lisansky, 1985; Rabkin, Quitkin, Stewart, McGrath, & Piug-Antich, 1983). In addition, it has also been suggested that depressives with melancholic symptoms respond better to antidepressant medication than depressives with nonmelancholic symptoms (Bielski & Friedel, 1976; Kiloh et al., 1962; Paykel, 1972; Rao & Coppen, 1979; Raskin & Crook, 1976). Because of the studies suggesting that melancholic symptoms covary with abnormal dexamethasone suppression test results, and that melancholic depressives may respond better to antidepressant medication, melancholia was examined in a post hoc fashion in this present investigation.

Diagnoses were made by the principal investigator using the specific criteria from the Diagnostic and Statistical Manual of Mental Disorders, third edition (see Appendix G-2). Therefore, questions asked were: (a) Did melancholic symptoms correlate highly with abnormal responses to the dexamethasone suppression test; and (b) Were there differential treatment effects, depending on the presence or absence of melancholic symptoms?

As reported earlier, a high correlation (.74) was found between abnormal dexamethasone suppression test results and melancholic symptoms based on the Diagnostic and Statistical Manual of Mental Disorder, third edition (see Appendix G-2 and Table 4, Appendix M). Therefore, melancholic symptoms were highly correlated with abnormal results, which support the above mentioned studies. The treatment validity of the presence or absence of melancholia was not supported. Similar results were found for the treatment validity of the dexamethasone suppression test and for the presence or absence of melancholia. In other words, overall subjects with or without melancholia significantly improved with regard to depressive symptoms regardless of type of treatment, with non-melancholic subjects improving significantly more than melancholic subjects. The effectiveness of cognitive therapy for melancholics was supported by Kovacs et al. (1981) and Blackburn (1981) who did not find any evidence that "endogenous" or "melancholic" symptoms in non-psychotic depression predicted any worse outcome in response to cognitive therapy. Other studies have also not found the distinction of presence or absence of melancholia useful in predicting response to chemotherapy (Raskin & Crook, 1976; Zimmerman, Coryell, & Pfohl, in press).

There are several possibilities as to why the above results were found. First, it may be that the diagnosis of melancholia in its present state is unreliable, with different researchers using different criteria. Second, it may be that the presence or absence of melancholic symptoms does not make the endogenous-exogenous distinction, and that this distinction may be based on other factors (e.g., family history, occurrence of manic symptoms). Third, the distinction of the presence or absence of melancholic symptoms may be useless in the selection of treatment. As with results from the dexamethasone suppression test, the melancholic distinction may be useful in serving other functions (e.g., such as a predictor for the course of treatment) but does not help in selecting treatment. Finally, while the presence or absence of melancholic symptoms may represent the "etiology" of a depressive illness, treatment choice may not be dependent on etiology.

Effects of Cognitive Therapy and Antidepressant Medication on the Dexamethasone Suppression Test

Of particular interest here was the impact of cognitive therapy and antidepressant medication on subjects who had abnormal dexamethasone suppression test results. It was predicted that cognitive therapy would not impact on abnormal dexamethasone suppression test results, whereas antidepressant treatment would result in a significant reduction in dexamethasone suppression test scores for abnormal responders. This prediction was based on studies suggesting that an abnormal dexamethasone suppression test result represents an "endogenous"

depression which is suggestive of a biological abnormality and treatable only by a biologically-oriented treatment (antidepressant medication), along with studies showing conversion from abnormal to normal results on the dexamethasone suppression test after administration of antidepressant medication (Bowie & Beaini, 1985; Brown et al., 1979; Brown & Shuey, 1979; Fraser, 1983; Greden et al., 1980; Spar & La Rue, 1983). Therefore, it was predicted that cognitive therapy would have no effect on the dexamethasone suppression test for abnormal responders. In actuality, there was a significant reduction in dexamethasone suppression test scores for abnormal responders after being exposed to either antidepressant medication or cognitive therapy. As was mentioned earlier, several studies have shown that treatment with antidepressant medication is associated with significant reductions in dexamethasone suppression test scores (e.g., conversion from abnormal to normal results) and clinical improvement. This is the first study to examine and show that a psychological treatment for depressed outpatients, cognitive therapy, was also associated with significant reductions on the dexamethasone suppression test (in addition to conversions from abnormal to normal results) and significant clinical improvement.

While several studies have shown a clear correlation between clinical improvement in depression and significant reductions in cortisol levels based on the dexamethasone suppression test (and the lack of reduction in cortisol without clinical improvement) (Bowie & Beaini, 1985; Brown et al., 1979; Brown & Shuey, 1979; Carroll, 1980; Greden et al., 1980; Fraser, 1983; Sachar, 1982; Spar & La Rue, 1983), none of these studies, including the present investigation, has utilized

control groups to examine the dexamethasone suppression test over time without treatment. Therefore, the only support available that the dexamethasone suppression test would not change over time is the above studies showing strong correlations between cortisol levels and clinical improvement. To illustrate, Bowie and Beaini (1985) showed that for abnormal responders on the dexamethasone suppression test, normalization either presided or coincided with good clinical response, while poor clinical response remained highly correlated with abnormal dexamethasone suppression test results.

The question must be asked, "How can a psychological treatment impact on a biological system?" The theoretical position of physical monism may be of help here. This view says that both types of treatments are effective in altering the dexamethasone suppression test because they are both dealing with the same substance, physical matter. The only difference between the two treatments is the level of intervention. Therefore, the physical system can be affected by either a biochemical level of intervention, or by an environmental level of intervention. Antidepressant medication may impact on the biological system (represented here by the dexamethasone suppression test) by directly manipulating the neurotransmitters in the brain (e.g., increases in serotonin). Cognitive therapy may indirectly affect the biological system by altering dysfunctional thoughts. Depression may be unitary phenomenon that involves a physical system. Changes resulting from treatment (be it cognitive therapy or chemotherapy) depend on the level of intervention (biochemical, cognitive, behavioral). Another view might hold that there are different "systems" that are affected in

a depressive episode (e.g., biochemical, cognitive, behavioral). These systems are interrelated, so that if one system is affected, the other systems are affected also. Therefore, if the cognitive system is impacted through cognitive therapy, corresponding changes in the biochemical system could also occur. Similarly, changes in the biochemical system may result in corresponding changes in the cognitive system. Some researchers have objected to the use of the terms levels or interactions, and instead employ the term transaction since this term does not imply priority of one type of analysis over another and does not imply that the systems are independent, non-related systems.

Effects of Cognitive Therapy and Antidepressant Medication on Dysfunctional Thoughts

Beck (1967, 1972, 1979) maintains that negative thought patterns and distorted perceptions that the depressed person holds about himself/herself, the present, and the future result in depression. Therefore, Beck's cognitive therapy of depression focuses directly on modifying dysfunctional thoughts and overt behavior in order to change the patient's negative self-perception and by doing so alleviating the patient's depression. Based on this rationale and the effectiveness of Beck's treatment for depression, it was predicted that cognitive therapy would have a significant impact on the subject's dysfunctional thoughts (i.e., significantly reduce the number of dysfunctional thoughts) while antidepressant medication would not. This prediction was not supported in that both cognitive therapy and antidepressant medication resulted in

significant reductions of dysfunctional thoughts, while cognitive therapy produced significantly greater reductions than antidepressant medication (according to the Personal Beliefs Inventory). These results parallel very closely the results from a study conducted by Rush, Beck, Kovacs, Weissenburger, and Hollon (1982) which found significant reduction in hopelessness and a significant increase in self-esteem after depressives received either chemotherapy or cognitive therapy, with cognitive therapy producing a significantly greater change than chemotherapy.

Again, the question must be asked: How can a somatic treatment result in significant reductions of dysfunctional thoughts? One answer is consistent with a growing body of research that suggests dysfunctional thinking is a "result," "symptom," or "correlate" of depression rather than a "cause" (Lewinsohn et al., 1981; Silverman et al., 1984; Simons et al., 1984). For example, Lewinsohn et al. (1981) in a longitudinal study, found that dysfunctional thoughts do not exist immediately prior to or after a depressive episode (with no treatment), but rather dysfunctional thoughts are a symptom or correlate accompanying the depression. Silverman et al. (1984) and Simons et al. (1984) used a methodology similar to that of this present study by examining directly the effect of chemotherapy on dysfunctional thoughts. Chemotherapy is believed to be treating the "depressive illness," which when alleviated ameliorates the "symptom" of dysfunctional thinking. Therefore, somatic treatments treat the underlying "depressive illness" resulting in the alleviation of the symptoms of dysfunctional thinking,

much like antibiotics treating the underlying virus which results in the alleviation of the symptoms such as nausea or fever.

Another answer would again utilize the theoretical position of physical monism (see previous section). Both treatments are effective because they are both dealing with the same substance, physical matter. The only difference between the treatments is the level of intervention. Therefore, the physical system can be affected at either a biochemical level of intervention, or an environmental level of intervention. Changes in cognitions resulting from cognitive therapy are changes in physiology, while changes in biochemistry resulting from chemotherapy result in changes in cognitions. Depression may be a unitary phenomenon that affects the whole physical system. Changes resulting from treatment (chemotherapy or cognitive therapy) depend on the level of intervention (biochemical, cognitive, behavioral).

Another view is consistent with Coyne (1980) and Blackburn and Bishop (1983) who argue that there is a circular system in depression involving mood, cognitions, and biochemical changes. Depending on the level of intervention or entry into the system, all these functions will change to the same degree depending on the efficacy of the intervention. This view is consistent with the present results in that both treatments resulted in reductions of dysfunctional thoughts (and depressive symptoms), with cognitive therapy resulting in significantly greater reductions of dysfunctional thoughts than chemotherapy. Based on this view, cognitive therapy would be a more direct intervention with regard to dysfunctional thoughts as compared to chemotherapy (therefore, the greater effectiveness of cognitive therapy). But because of the

circular system, chemotherapy results in changes in the related system of cognitions.

A final comment is warranted in response to assertions that dysfunctional thoughts are not "causes" of depression but rather are "correlates" or "symptoms" of a depressive syndrome. It could be argued that dysfunctional thoughts or abnormal biochemistry (e.g., abnormal dexamethasone suppression test) may represent "proximal" causes or maintaining factors in depression. Manipulation of these "proximal" causes result in changes in these factors and consequently in the depression, along with changes in intercorrelated factors (e.g., changes in cognition resulting in changes in biochemistry). "Ultimate" causes of depression (e.g., experiences when young, predispositions to depression) may or may not be discovered for the individual patient, and if discovered, they may not be able to be manipulated. Therefore, while dysfunctional thoughts or abnormal brain chemistry may not represent "ultimate" causes, they may represent "proximal" causes or maintaining factors.

Strengths and Limitations of the Present Investigation

The present investigation has several major methodological strengths. First, the population ware carefully selected in terms of subjects having a Major Depression as their major presenting problem. This was based on the most commonly used questionnaires and criteria for depression, along with a structured interview to arrive at a diagnosis with high and measured reliability. Subjects with normal and abnormal

dexamethasone suppression test results also did not differ with regard to severity of their depressions according to the questionnaires. Second, factors affecting the dexamethasone suppression test were ruled out, such as physical illnesses, alcohol consumption, diabetes, pregnancy, and the ingestion of certain medicines. Third, the treatments were clearly defined and therapeutic levels of antidepressant were assured by the use of blood tests measuring blood plasma levels of the medicine. Next, both the principal investigator and the staff psychiatrist were blind to the dexamethasone suppression test results. Also, the presence or absence of melancholic symptoms were recorded and analyzed, which is seldom done in most studies on depression. In addition, there was a very low attrition rate for all groups unlike the study performed by Rush et al. (1977) which showed high attrition rates for the medicine group and not for the cognitive therapy group. Related to this, the present investigation allowed for the flexible administration of the tricyclic medicines to assure maximum effectiveness and to better mirror clinical reality. This has been a criticism of previous studies comparing cognitive therapy and antidepressant medications. Finally, subjects were selected at pre-intervention to have a high level of dysfunctional thoughts to assure that the treatments would be appropriate for all subjects.

In addition to the major strengths, several limitations exist in the present investigation that may be important for future research. First, the defined population is restricted to outpatient depressives with a high level of dysfunctional thoughts. It may be that abnormal dexamethasone suppression test responders without dysfunctional thoughts

may respond to a purely biological approach (medications) and not to cognitive therapy. Further, the results may be different if this study were conducted with hospital inpatients. For example, the treatment validity of the dexamethasone suppression test may be valid for hospital inpatients. Their dexamethasone suppression test results may be more abnormal, representing a more severe biological abnormality that may render psychological interventions ineffective; biologically-oriented treatments might prove to be more expedient or cost-effective with these inpatients versus psychologically-oriented treatments. Since there are no studies directly examining similarities and differences between inpatient and outpatient depressives' with regard to the dexamethasone suppression test, this is an important area of future study. Also, some clinicians or researchers may question whether subjects in this project were "real" melancholics, and therefore question the diagnosis. Second, since different tricyclic antidepressants were prescribed by a single psychiatrist, it is difficult to generalize across tricyclics not used in this study, along with the fact that different psychiatrists may have prescribed different antidepressants than the psychiatrist in this investigation. In other words, this study cannot generalize across treatments for depression, either somatic or psychological, not utilized in this study. Further, while the psychiatrist meeting with the subjects receiving medication was instructed to focus on the biological model of depression, his sessions (and the cognitive therapy sessions) were not taped to assure compliance with the protocol (although the psychiatrist had no formal training in cognitive therapy and the cognitive therapy sessions were structured in a formal protocol).

Related to this, a confound existed in terms of different lengths of sessions for the two treatments. While the cognitive therapy sessions were one hour long, the medication sessions ranged from one-half hour to one hour in duration. Also, the principal investigator conducted the cognitive therapy rather than having unbiased therapists, in spite of taking precautions such as being blind to the dexamethasone suppression test results, having another person assign subjects to groups, score questionnaires, etc. Thirdly, there have been no studies examining the effect of time on the dexamethasone suppression test without treatment (this investigation did not employ a control group). Fourth, factors resulting in false positives on the dexamethasone suppression test may have influenced the results, such as the finding that caffeine can results in false positives (Uhde, 1985) or the influence of age on cortisol levels (Lewis, Pfohl, Schlehte, & Coryell, 1984). Finally, the finding by Bowie and Beaini (1985) that a fluctuating nonsuppressor group exists based on the dexamethasone suppression test (subjects who alternate between normal and abnormal results over time) may represent a separate population that needs to be defined in future studies based on the dexamethasone suppression test.

Conclusions

This study compliments previous research by demonstrating that exposure to cognitive therapy or tricyclic antidepressants results in a significant reduction of depressive symptoms. This investigation also supports a growing body of research that suggests that the dexamethasone

suppression test (and the presence or absence of melancholic symptoms) does not support the functional utility of the endogenous-exogenous distinction in selecting treatments for depression. The dexamethasone suppression test may actually be more appropriate as an indicator of prognosis (with abnormal responders responding less well to treatment than normal responders) and a "present state" indicator of a depressive's course in treatment. Furthermore, this study supports the existing body of literature suggesting that the changing of dysfunctional thoughts by the use of cognitive therapy alleviates depression. Because of the fact that antidepressant medication also significantly lessened dysfunctional beliefs (although not as much as cognitive therapy), it cannot be concluded that dysfunctional thoughts "cause" depression. Also, this study was the first to show that a psychologically-oriented treatment (cognitive therapy) could impact on a biological system resulting in many cases "normalization" of that system (e.g., conversion from abnormal to normal results on the dexamethasone suppression test). Finally, reanalyzing the results in terms of the presence or absence of melancholic symptoms revealed the same conclusions that were made in dividing subjects based on normal or abnormal dexamethasone suppression test results.

Future directions of research should first of all attempt to correct some of the limitations present in the current investigation. Control groups, if possible, should be utilized particularly with repeated administration of the dexamethasone suppression test. In addition, future research should examine the dexamethasone suppression test with different groups of depressives, such as hospital inpatients,

or dexamethasone suppression test responders without dysfunctional thoughts. Future research should also continue to examine other biological correlates (e.g., GH response, REM latency, EEG, Metyrapone tests) in terms of their relation to the dexamethasone suppression test and in terms of their treatment validity. Future studies should retain a diagnosis of melancholia, along with further examination of the relationship between the presence of melancholic symptoms and abnormal dexamethasone suppression test results. Finally, other uses of the dexamethasone suppression test should be examined, such as predicting suicide, prognosis, and prediction of relapse.

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APPENDICES

APPENDIX A

Dear _____:

(Physician's Name)

We are conducting a psychiatric/psychological research-treatment project to examine the differential effectiveness of a chemotheraputic approach versus a cognitively-oriented approach in treating depressed female clients. The techniques we are using have been shown to be useful with this population in the past. Since in your medical practice you are in contact with women who report that they are depressed, we are requesting that you refer to us any client whom you think is an appropriate candidate for psychiatric/psychological treatment.

We will be working only with female clients who are not suicidal and who have been free from tranquilizing or anti-depressant medications for a minimum of two weeks (or, if the patient is taking medication for depression, has your permission to discontinue drug use for the duration of the project).

We are enclosing a flyer describing the project which you may give to any patient whom you refer. In addition, we are enclosing several "Physician Statements" which acknowledge that the patient you are referring has met the medical requirements specified.

If you have any questions concerning the specific procedures that we will use, you may contact Dennis McKnight at Charter Mandala Center (768-7710) between 9:00 a.m. and 5:00 p.m., Monday through Friday.

Thank you very mcuh for your assistance.

Sincerely,

Dennis McKnight, M.A.
Doctorial Candidate
in Clinical Psychology

Jarrett Barnhill, M.D.
Staff Psychiatrist
Charter Mandala Center

Rosemery Nelson, Ph.D.
Professor of Psychology
University of North
Carolina at Greensboro

APPENDIX B

Newspaper Notice

If you have been feeling depressed and are female, at least 18 years old, you may be interested in a research-treatment project being conducted at Charter Mandala Center in Winston-Salem. If you are interested and eligible for participation in this project, three assessment sessions and eight individual treatment sessions will be available to you for only the cost of laboratory work and medication, if prescribed, along with a $50.00 deposit that is fully refundable after completion of the study.

If interested, call Dennis McKnight at Charter Mandala Center (768-7710 between 9:00 a.m. and 5:00 p.m., Monday through Friday) to schedule a "screening interview". At the screening interview, your eligibility for participation will be further assessed, and additional details of treatment will be offered.

APPENDIX C

Descriptive Flyer

If you have been feeling depressed and are female, at least 18 years old, you may be interested in a research-treatment project being conducted at Charter Mandala Center in Winston-Salem. If you are interested in and eligible for participation in this project, three assessment sessions and eight individual treatment sessions will be available to you for only the cost of laboratory work and medication, if prescribed.

In order to participate in the project you must:

1. be feeling depressed and be a female at least 18 years old
2. have been free from anti-depressant or tranquilizing drugs for a minimum of two weeks
3. if you are under a physician's care for depression, you must submit a "physician's statement" stating that you are free of anti-depressant or tranquilizing drugs and are appropriate for psychological treatment
4. if interested, call Dennis McKnight at Charter Mandala Center in Winston-Salem (768-7710 between 9:00 a.m. and 5:00 p.m., Monday through Friday) to schedule a "screening interview". At the screening interview, your eligibility for participation will be further assessed, and additional details of treatment will be offered.

Thank you for your interest

APPENDIX D

Medication Statement

This statement affirms that I, _____,
have been free from any tranquilizing drugs or anti-depressant medication for the past two weeks, and will remain free from this medication as long as I am a participant in this research-treatment project, unless medication is prescribed during this project. In addition, I am not currently under a physician's (or psychiatrist's or psychologist's) care for the treatment of depression or the symptoms relating to depression, and will not be for the duration of this project. Failure to comply with this statement will result in a discontinuation of my participation in this project.

Signed _____

Witness _____

Date _____

APPENDIX E

Physician's Statement

PLEASE RETURN TO:
Dennis L. McKnight, M.A.
Charter Mandala Center
3637 Old Vineyard Road
Winston-Salem, NC 27104

Telephone
Charter Mandala Center: 768-7710
Monday - Friday 9:00 a.m.-5:00 p.m

Date: _____

This statement acknowledges that to my knowledge, based on a medical examination, _____, is an appropriate candidate for participation in a research project which compares the differential effectiveness of a chemotheraputic (medicine) and cognitive therapy (psychological) approach to treating depression. To the best of my knowledge, she has been free from anti-depressant or tranquilizing medication for at least two weeks prior to the date listed above, or has discontinued drug use from the date indicted and is appropriate for psychological treatment. In addition, it is my opinion that her symptoms of depression are not due to medication she is receiving under my supervision.

Please check ( ) Free from drug use; minimum two weeks
( ) Discontinued drug use from date indicated

Date of last prescription for anti-depressant or tranquilizing medication: _____

Date of last medical examination: _____

_____
Physician's Signature

_____
Office Telephone Number

APPENDIX F

Consent Form I

I understand that I will be interviewed and asked to complete questionnaires to be used in selecting subjects for a research investigation involving the assessment and treatment of depression. In addition, I have been informed that I may withdraw from this screening session at any time, and that all personal information (e.g., my name) I give is completely confidential, and will only be available to the experimenters. I further understand that specific numerical scores provided by laboratory tests and by questionnaires will be used (without my identity) for research purposes and publication. I understand that if I am not eligible for participation in this program, I will be given a list of referrals for assessment and treatment in Winston-Salem that I amy contact if I so desire. However, if I am eligible for this program, I understand that experimental procedures will be explained to me more fully before I decide to continue to participate.

Signed: _____

Witness: _____

Date: _____

APPENDIX G-1

SADS Interview Outline: Shortened Version

Dysphoria:

How is it going? (Work, school, home life)

Feeling good or bad about it?

Worried?

Feeling under pressure? From where?

If things are bad, what are the prospects for improvement in the immediate or distant future?

Major happenings during the past year: best? worst?

Goals for the future? Expectations for attainment?

Self-description: good points? bad points?

Aspects of self that would be desirable to change?

Mood:

Ups and downs? How severe and long-lasting are the downs? Any highs?

Any thoughts or ideas about suicide? Previous attempts? Plans?

Describe Typical Day:

Interests and activities that are enjoyable?

Any change from previous level of activity or enjoyment?

Difficulty in initiating action?

Having to exert a lot of effort to do things?

Problems making decisions?

Social-Interactional Problems:

How involved with other people?

Number of close friends? Acquaintances?

Ability to share with friends?

Are relationships a source of discomfort, anxiety, and/or conflict?

Feelings of social adequacy/inadequacy?

Guilt:

Religious background; importance of religion at present?

Concern for welfare of family and friends?

Blame self for present condition?

Perceive self as failure in important responsibilities?

Material Burden:

Depression attributed to external problems (e.g., finances, children, demands of relatives or employers)?

If external problems could be resolved, would that affect the depression?

Somatic Manifestations: (not attributable to physical condition)

Feeling slow? Tired all the time? Without energy?

Problems sleeping? Difficulty in falling asleep? Waking frequently during the night? Sleep not restful? Problems with waking early in the morning and not being able to get back to sleep? Sleeping more than usual?

How is appetite? Any weight loss?

Gastrointestinal problems?

Headaches?

APPENDIX G-2

Criteria for Major Depression and Melancholia based on the Diagnostic and Statistical Manual of Menal Disorders, third edition.

A. Dysphoric mood or loss of interest or pleasure in all or almost all usual activities and pastimes. The dysphoric mood is characterized by symptoms such as the following: depressed, sad, blue, hopeless, low, down in the dumps, irritable. The mood disturbance must be prominent and relatively persistent, but not necessarily the most dominant symptom, and does not include momentary shifts from one dysphoric mood to another dysphoric mood, e.g., anxiety to depression to anger, such as are seen in states of acute psychotic turmoil. (For children under six, dysphoric mood may have to be inferred from a persistently sad facial expression.)

B. At least four of the following symptoms have each been present nearly every day for a period of at least two weeks (in children under six, at least three of the first four):
   (1) Poor appetite or significant weight loss (when not dieting) or increased appetite or significant weight gain (in children under six, consider failure to make expected weight gains).
   (2) Insomnia or hypersomnia.
   (3) Psychomotor agitation or retardation (but not merely subjective feelings of restlessness or being slowed down) (in children under six, hypoactivity).
   (4) Loss of interest or pleasure in usual activities, or decrease in sexual drive not limited to a period when delusional or hallucinating (in children under six, signs of apathy).
   (5) Loss of energy; fatigue.
   (6) Feelings of worthlessness, self-reproach, or excessive or inappropriate guilt (either may be delusional).
   (7) Complaints or evidence of diminished ability to think or concentrate, such as slowed thinking, or indecisiveness not associated with marked loosening of associations or incoherence.
   (8) Recurrent thoughts of death, suicidal ideation, wishes to be dead, or suicide attempt.

C. Neither of the following dominate the clinical picture when an affective syndrome is absent (i.e., symptoms in criteria A and B above):
   (1) Preoccupation with a mood-incongruent delusion or hallucination.
   (2) Bizarre behavior.

D. Not superimposed on either Schizophrenia, Schizophreniform Disorder, or a Paranoid Disorder.

E. Not due to any Organic Mental Disorder or Uncomplicated Bereavement.

F. With Melancholia. Loss of pleasure in all or almost all activities, lack of reactivity to usual pleasurable stimuli (doesn't feel much better, even temporarily, when something good happens), and at least three of the following:
   (a) Distinct quality of depressed mood, i.e., the depressed mood is perceived as distinctly different from the kind of feeling experienced following the death of a loved one.
   (b) The depression is regularly worst in the morning.
   (c) Early morning awakening (at least two hours before usual time of awakening).
   (d) Marked psychomotor retardation or agitation.
   (e) Significant anorexia or weight loss.
   (f) Excessive or inappropriate guilt.

APPENDIX I

Lubin Depression Adjective Check List (DACL)

The DACL check lists consist of 7 forms (A-G). Forms A-D consist of balanced sets of 22 positive and 10 negative adjectives from a pool of items which significantly differentiated between a group of 48 depressed female psychiatric patients and a group of 179 normal females. Forms E-G consist of balanced sets of 22 positive and 12 negative adjectives from a pool of adjectives which significantly differentiated between a group of 47 depressed male psychiatric patients and a group of 100 normal males.

Intercorrelations among the 7 forms are high, regardless of sex of subject group (Lubin, 1967). Internal consistency indices range from 0.79 to 0.90, and split-half reliabilites range from 0.82 to 0.93 for normals and from 0.86 to 0.93 for patients. All forms were cross-validated in a large study using normals, and depressed and non-depressed patient groups. Correlations with the MMPI-D are 0.31 (Nussbaum et al., 1963), 0.54-0.57 (Lubin, 1967), and 0.42-0.55 (Lubin, 1967); with teh Zung SDS are 0.53 (Levitt and Lubin, 1975), 0.63 (Levitt & Lubin, 1975), 0.58 (Lubin, 1967), 0.27-0.38 (Lubin, 1967); and with the Beck DI are 0.66 (Nussbaum et al., 1963), and 0.38-0.50 (Lubin, 1967).

One key will score all seven lists. The pattern of plus and minus adjectives on each list is the same. To score, place the stencil over the list and score one point for each (+) adjective that is checked and one point for each minus adjective (0) that is not checked. The score for each lists consists of the total number of plus (+) adjectives checked and minus (0) adjectives not checked.

APPENDIX J

Scale for Suicide Ideation

Name _____ Date _____

Characteristics of Attitude Toward Living/Dying

1. Wish to live
   0. Moderate to strong
   1. Weak
   2. None

2. Wish to die
   0. None
   1. Weak
   2. Moderate to strong

3. Reasons for living/dying
   0. For living outweigh for dying
   1. About equal
   2. For dying outweigh for living

4. Desire to make active suicide attempt
   0. None
   1. Weak

5. Passive suicidal attempt
   0. Would take precautions to save life
   1. Would leave life/death to chance
   2. Would avoid steps necessary to save or maintain life (e.g., diabetic ceasing to take insulin)

APPENDIX K

Referrals for Continued Assessment and Treatment of Depression

1. Forsyth County Mental Health — 725-7777
   725 Highland Avenue
   Winston-Salem, NC

2.** Salem Psychiatric Associates — 768-6930
   Charlois Boulevard
   Winston-Salem, NC

3.** Charter Mandala Center — 768-7710
   3637 Old Vineyard Road
   Winston-Salem, NC

4. Spectrum — 761-0650
   1111 Brookstown Avenue
   Winston-Salem, NC

5. NOTE: Practicing psychiatrists and psychologists in the Winston-Salem area can be found in the yellow pages of the phone book.

** Can request Dennis McKnight or other staff psychiatrist or psychologist.

APPENDIX M

TABLES

TABLE 1
Descriptive Data on Subjects Completing the Study

SUBJECT NUMBER	GROUP[a] NUMBER	AGE	YEARS EDUCATED	OCCUPATION	PREVIOUS TREATMENT	FAMILY HISTORY? YES or NO
1	1	30	12	Housewife	None	No
2	1	36	14	Salesperson	Counseling	No
3	1	37	14	Unemployed	None	Yes
4	1	28	12	Secretary	None	No
5	1	30	13	Beautician	None	Yes
6	1	28	15	Student	None	No
7	1	41	16	Nurse	Trial on Antidepressant	No
8	1	28	12	Housewife	Short-Term Therapy	No
9	1	63	14	Retired	None	Yes
10	1	37	13	Housewife	None	No
11	1	38	13	Secretary	Counseling	No
12	1	48	14	Unemployed	None	Yes
13	2	39	12	Secretary	None	Yes
14	2	24	13	Secretary	None	Yes
15	2	48	13	Health Professional	None	No
16	2	52	13	Manager	None	No
17	2	39	14	Unemployed	Short-Term Therapy	No
18	2	36	12	Housewife	None	No
19	2	35	14	Student	Counseling	Yes
20	2	37	12	Secretary	None	Yes
21	2	40	12	Housewife	None	No
22	2	25	15	Student	None	Yes
23	2	39	16	Salesperson	Trial on Antidepressant	No
24	3	32	12	Housewife	None	No
25	3	38	13	Clerical Worker	Analysis	No
26	3	39	13	Clerical Worker	None	No
27	3	26	14	Reservationist	None	No
28	3	33	12	Housewife	Short-Term Therapy	Yes
29	3	38	13	Secretary	None	Yes
30	3	42	13	Secretary	None	Yes
31	3	45	15	Computer Operator	Counseling	No
32	3	50	14	Salesperson	None	No
33	3	36	14	Health Professional	None	No
34	4	37	15	Reservationist	None	Yes
35	4	38	12	Clerical Worker	Trial on Antidepressant	No
36	4	32	16	Salesperson	Counseling	Yes
37	4	35	13	Housewife	None	No
38	4	37	12	Salesperson	Trial on Antidepressant	No
39	4	38	14	Computer Operator	None	No
40	4	45	14	Unemployed	None	No
41	4	48	12	Housewife	None	Yes
42	4	29	13	Secretary	None	No
43	4	36	13	Secretary	None	No

[a]Group 1 = Normal Dexamethasone Suppression Test; Cognitive Therapy
Group 2 = Normal Dexamethasone Suppression Test; Antidepressant Therapy
Group 3 = Abnormal Dexamethasone Suppression Test; Cognitive Therapy
Group 4 = Abnormal Dexamethasone Suppression Test; Antidepressant Therapy

TABLE 2

Sketch of Experimental Design

TREATMENT	GROUP	PRE	POST
COGNITIVE THERAPY	Normal Dexamethasone Suppression Test		
	Abnormal Dexamethasone Suppression Test		
ANTIDEPRESSANT MEDICATION	Normal Dexamethasone Suppression Test		
	Abnormal Dexamethasone Suppression Test		

TABLE 3

Raw Scores on Global Depression Measures and Cognitive Measure

SUBJECT NUMBER	GROUP NUMBER[a]	PRE-MMPI-D[b]	POST MMPI-D[b]	PRE-BDI[c]	POST-BDI[c]	PRE-DACL	POST-DACL[d]	PRE-PBI[e]	POST-PBI[e]
1	1	38	19	33	5	22	3	3.0	2.8
2	1	45	40	40	37	21	18	3.0	3.0
3	1	38	21	28	0	18	5	3.2	1.8
4	1	33	18	26	0	18	1	3.2	2.6
5	1	38	19	28	0	19	2	3.4	2.4
6	1	36	17	27	5	12	10	3.1	2.5
7	1	35	27	30	15	20	1	3.1	2.6
8	1	37	20	29	1	18	3	3.2	1.7
9	1	40	22	30	10	20	5	3.3	2.0
10	1	37	21	32	12	22	8	3.5	2.2
11	1	44	25	33	15	25	10	3.2	2.6
12	1	34	27	29	19	20	13	2.1	2.3
13	2	34	20	26	4	18	9	3.0	2.7
14	2	48	36	34	19	22	10	3.1	3.0
15	2	31	18	29	4	25	6	3.2	2.2
16	2	41	33	33	15	27	10	3.0	2.3
17	2	40	24	24	2	25	2	3.0	3.1
18	2	41	27	24	7	21	10	3.0	3.2
19	2	38	26	25	3	19	10	3.3	3.0
20	2	35	21	27	12	20	8	3.1	2.8
21	2	40	27	26	8	25	6	3.4	3.2
22	2	42	34	28	6	22	5	3.0	2.3
23	2	33	17	30	13	18	7	3.5	3.0
24	3	35	35	29	30	20	20	3.0	3.3
25	3	40	31	45	21	31	8	3.7	2.5
26	3	43	39	40	15	28	20	3.0	3.0
27	3	41	30	30	15	19	13	3.3	2.3
28	3	40	31	28	18	21	21	3.6	3.4
29	3	37	27	37	21	26	3	3.1	3.0
30	3	39	28	40	20	20	20	3.0	2.9
31	3	40	25	29	28	25	15	3.4	2.1
32	3	48	35	36	21	19	17	3.0	2.8
33	3	34	21	43	19	18	13	3.2	2.1
34	4	40	31	23	20	19	14	3.1	2.6
35	4	41	28	33	5	21	4	3.0	2.9
36	4	38	25	28	4	18	3	3.0	2.9
37	4	35	24	24	14	23	7	3.0	2.9
38	4	30	21	22	5	19	11	3.0	3.1
39	4	42	29	25	22	20	15	3.2	3.1
40	4	37	24	22	8	21	4	3.1	2.6
41	4	40	31	27	21	19	14	3.0	2.3
42	4	34	21	30	3	18	16	3.4	3.0
43	4	36	23	27	23	23	15	3.5	3.1

[a] Group 1 = Normal Dexamethasone Suppression Test; Cognitive Therapy
Group 2 = Normal Dexamethasone Suppression Test; Antidepressant Therapy
Group 3 = Abnormal Dexamethasone Suppression Test; Cognitive Therapy
Group 4 = Abnormal Dexamethasone Suppression Test; Antidepressant Therapy

[b] MMPI-D = Raw Scores from the Depression Scale of the Minnesota Multiphasic Personality Inventory

[c] BDI = Raw Scores from the Beck Depression Inventory

[d] DACL = Raw Scores from the Lubin Depression Adjective Checklist

[e] PBI = Raw Scores from the Personal Beliefs Inventory

TABLE 4

Diagnosis and Dexamethasone Suppression Test Score on Subjects Completing the Study

NUMBER	NUMBER	PRE-DIAGNOSIS (ALL MAJOR DEPRESSIVES WITH OR WITHOUT MELANCHOLIA)	POST-DIAGNOSIS	PRE-DST	POST-DST	ANTIDEPRESSANT MEDICATION ADMINISTERED
1	1	Without Melancholia	Major Depression Without Melancholia	0.0	0.6	None
2	1	Without Melancholia	None	0.2	0.1	None
3	1	Without Melancholia	None	0.3	0.2	None
4	1	Without Melancholia	None	0.1	0.1	None
5	1	Without Melancholia	None	0.1	0.4	None
6	1	Without Melancholia	None	0.0	0.2	None
7	1	Without Melancholia	None	0.2	0.6	None
8	1	Without Melancholia	None	1.2	0.3	None
9	1	Without Melancholia	None	1.5	1.3	None
10	1	Without Melancholia	None	0.8	0.7	None
11	1	Without Melancholia	None	1.0	0.8	None
12	1	Without Melancholia	None	0.9	0.7	None
13	2	With Melancholia	None	3.5	1.2	Elavil
14	2	With Melancholia	None	2.9	0.7	Elavil
15	2	Without Melancholia	None	0.0	3.6	Elavil
16	2	Without Melancholia	None	0.4	0.2	Desipramine
17	2	Without Melancholia	None	0.4	0.7	Elavil
18	2	Without Melancholia	None	0.0	0.2	Desipramine
19	2	Without Melancholia	None	1.2	0.0	Desipramine
20	2	Without Melancholia	None	2.1	1.9	Elavil
21	2	Without Melancholia	None	1.7	1.6	Desipramine
22	2	Without Melancholia	None	0.8	0.2	Elavil
23	2	Without Melancholia	None	0.1	0.1	Elavil
24	3	With Melancholia	Major Depression With Melancholia	6.1	2.5	None
25	3	With Melancholia	None	5.6	4.5	None
26	3	Without Melancholia	Major Depression Without Melancholia	5.1	2.8	None
27	3	With Melancholia	None	5.7	5.4	None
28	3	With Melancholia	None	5.6	5.3	None
29	3	With Melancholia	None	5.8	5.7	None
30	3	With Melancholia	None	6.1	2.7	None
31	3	With Melancholia	None	6.1	4.6	None
32	3	With Melancholia	Major Depression Without Melancholia	5.8	5.8	None
33	3	With Melancholia	None	5.9	5.7	None
34	4	With Melancholia	None	5.7	5.1	Desipramine
35	4	Without Melancholia	None	5.4	6.1	Elavil
36	4	Without Melancholia	None	5.3	0.4	Elavil
37	4	Without Melancholia	None	5.1	1.9	Elavil
38	4	With Melancholia	None	5.8	5.1	Elavil
39	4	Without Melancholia	Major Depression Without Melancholia	5.1	5.1	Elavil
40	4	With Melancholia	None	6.0	3.4	Desipramine
41	4	With Melancholia	Major Depression Without Melancholia	5.7	4.1	Desipramine
42	4	Without Melancholia	None	5.3	0.9	Elavil
43	4	With Melancholia	None	6.1	5.0	Desipramine

[a]Group 1 = Normal Dexamethasone Suppression Test; Cognitive Therapy
Group 2 = Normal Dexamethasone Suppression Test; Antidepressant Therapy
Group 3 = Abnormal Dexamethasone Suppression Test; Cognitive Therapy
Group 4 = Abnormal Dexamethasone Suppression Test: Antidepressant Therapy

TABLE 5

2 (Dexamethasone Suppression Test Result) x 2 (Treatment)

Multivariate Analysis of Variance

Using Difference Scores for the Global Measures of Depression

SOURCE	WILKS' LAMBDA	df	F	p
Treatment	.969	3,37	.39	.76
Dexamethasone Suppression Test Result	.747	3,37	4.17	.01*
Treatment x Dexamethasone Suppression Test Result	.904	3,37	1.31	.29
Subject (Treatment x Dexamethasone Suppression Test)				

* $p \leq .05$

** $p \leq .01$

*** $p \leq .001$

**** $p \leq .0001$

TABLE 6

2 (Dexamethasone Suppression Test Result) x 2 (Treatment) Univariate Analysis of Variance on Difference Scores from the Depression Scale of the Minnesota Multiphasic Personality Inventory With Least Squared Means

SOURCE	df	MS	F
Treatment	1	0.75	0.05
Dexamethasone Suppression Test Result	1	117.53	7.90**
Treatment x Dexamethasone Suppression Test Result	1	49.2	3.30
Subject (Treatment x Dexamethasone Suppression Test)	39	14.9	

Least Squared Means

Treatment	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T HO: LS MEAN 1=LS MEAN 2
Cognitive Therapy	12.2	.0001****	.9700
Antidepressant Medication	12.1	.0001****	

Dexamethasone Suppression Test Result	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T HO: LS MEAN 1=LS MEAN 2
Normal	13.8	.0001****	.0085**
Abnormal	10.5	.0001****	

TABLE 6 (Continued)

Treatment	Dexamethasone Suppression Test	Difference Scores	PROB > T HO:LS MEANS	PROB I/J	HO: LS MEAN (I) = LS MEAN (J)			
Cognitive Therapy (1)	Normal	14.9	.0001****	1	___	.0022**	.1816	.0515*
Cognitive Therapy (2)	Abnormal	9.5	.0001****	2	___	____	.0628	.2307
Antidepressant Medication (3)	Normal	12.7	.0001****	3	___	____	____	.5074
Antidepressant Medication (4)	Abnormal	11.6	.0001****	4	___	____	____	____

* $p \leq .05$
** $p \leq .01$
*** $p \leq .001$
**** $p \leq .0001$

TABLE 7

Group Means for Pre-Intervention and Post-Intervention Raw Scores from the Depression Scale of the Minnesota Multiphasic Personality Inventory

Group[a]	Pre	Post	
1	X = 37.9	X = 23.0	X = 30.45
2	X = 38.5	X = 25.7	X = 32.1
3	X = 39.7	X = 30.2	X = 34.95
4	X = 37.3	X = 25.7	X = 31.5
	X = 38.35	X = 26.15	X = 32.25

[a]Group 1 = Normal Dexamethasone Suppression Test; Cognitive Therapy

Group 2 = Normal Dexamethasone Suppression Test; Antidepressant Therapy

Group 3 = Abnormal Dexamethasone Suppression Test; Cognitive Therapy

Group 4 = Abnormal Dexamethasone Suppression Test; Antidepressant Therapy

TABLE 8

2 (Dexamethasone Suppression Test Result) x 2 (Treatment)

Univariate Analysis of Variance on Difference Scores

from the Beck Depression Inventory

With Least Squared Means

SOURCE	df	MS	F
Treatment	1	19.2	.30
Dexamethasone Suppression Test Result	1	344.9	5.42*
Treatment x Dexamethasone Suppression Test Result	1	.07	0.00
Subject (Treatment x Dexamethasone Suppression Test)	39	63.7	

Least Squared Means

Treatment	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T LS MEAN 1=LS MEAN 2 HO:
Cognitive Therapy	17.7	.0001****	.62
Antidepressant Medication	16.5	.0001****	

Dexamethasone Suppression Test Result	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T LS MEAN 1=LS MEAN 2 HO:
Normal	19.9	.0001****	.02*
Abnormal	14.2	.0001****	

TABLE 8 (Continued)

Treatment	Dexamethasone Suppression Test	Difference Scores	PROB ↘ T HO:LS MEANS	PROB I/J	HO: LS MEAN (I) = LS MEAN (J)			
Cognitive Therapy(1)	Normal	20.5	.0001****	1	___	.1092	.7348	.0503*
Cognitive Therapy(2)	Abnormal	14.9	.0001****	2	___	____	.2080	.7176
Antidepressant Medication (3)	Normal	19.4	.0001****	3	___	____	____	.1063
Antidepressant Medication (4)	Abnormal	13.6	.0001****	4	___	____	____	____

* $p \leq .05$
** $p \leq .01$
*** $p \leq .001$
**** $p \leq .0001$

TABLE 9

Group Means for Pre-Intervention and Post-Intervention Scores on the Beck Depression Inventory

Group[a]	Pre	Post	
1	X = 30.4	X = 9.9	X = 20.15
2	X = 27.8	X = 8.4	X = 18.10
3	X = 35.7	X = 20.8	X = 28.75
4	X = 26.1	X = 12.5	X = 19.30
	X = 30.0	X = 12.9	X = 21.45

[a]Group 1 = Normal Dexamethasone Suppression Test; Cognitive Therapy

Group 2 = Normal Dexamethasone Suppression Test; Antidepressant Therapy

Group 3 = Abnormal Dexamethasone Suppression Test; Cognitive Therapy

Group 4 = Abnormal Dexamethasone Suppression Test; Antidepressant Therapy

TABLE 10

2 (Dexamethasone Suppression Test Result) x 2 (Treatment)

Univariate Analysis of Variance on Difference Scores

from the Depression Adjective Checklist

With Least Squared Means

SOURCE	df	MS	F
Treatment	1	14.1	.38
Dexamethasone Suppression Test Result	1	317.8	8.46**
Treatment x Dexamethasone Suppression Test Result	1	6.5	.17
Subject (Treatment x Dexamethasone Suppression Test)	39	37.6	

Least Squared Means

Treatment	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T HO: LS MEAN 1=LS MEAN 2
Cognitive Therapy	10.8	.0001****	.48
Antidepressant Medication	12.1	.0001****	

Dexamethasone Suppression Test Result	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T HO: LS MEAN 1=LS MEAN 2
Normal	14.2	.0001****	.0061**
Abnormal	8.7	.0001****	

TABLE 10 (Continued)

Treatment	Dexamethasone Suppression Test	Difference Scores	PROB > T HO:LS MEANS	PROB I/J	HO: LS MEAN (I) = LS MEAN (J)			
Cognitive Therapy(1)	Normal	13.9	.0001****	1	___	.0229*	.8346	.1248
Cognitive Therapy(2)	Abnormal	7.7	.0003****	2	___	___	.0159**	.4482
Antidepressant Medication (3)	Normal	19.4	.0001****	3	___	___	___	.0901
Antidepressant Medication (4)	Abnormal	13.6	.0001****	4	___	___	___	___

* $p \leq .05$
** $p \leq .01$
*** $p \leq .001$
**** $p \leq .0001$

TABLE 11

Group Means for Pre-Intervention and Post-Intervention Scores on the Lubin Depression Adjective Checklist

Group[a]	Pre	Post	
1	X = 20.5	X = 6.6	X = 13.55
2	X = 22.0	X = 7.5	X = 14.75
3	X = 22.7	X = 15.0	X = 18.85
4	X = 20.1	X = 10.3	X = 15.20
	X = 21.33	X = 9.85	X = 15.59

[a]Group 1 = Normal Dexamethasone Suppression Test; Cognitive Therapy

Group 2 = Normal Dexamethasone Suppression Test; Antidepressant Therapy

Group 3 = Abnormal Dexamethasone Suppression Test; Cognitive Therapy

Group 4 = Abnormal Dexamethasone Suppression Test; Antidepressant Therapy

TABLE 12

2 (Dexamethasone Suppression Test Result) x 2 (Treatment)

Univariate Analysis of Variance on Difference Scores

from the Personal Beliefs Inventory

With Least Squared Means

SOURCE	df	MS	F
Treatment	1	1.35	6.92**
Dexamethasone Suppression Test Result	1	.42	2.17
Treatment x Dexamethasone Suppression Test Result	1	.18	.94
Subject (Treatment x Dexamethasone Suppression Test)	39	.19	

Least Squared Means

Treatment	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T LS MEAN 1=LS MEAN 2
Cognitive Therapy	.65	.0001****	.01**
Antidepressant Medication	.31	.0024****	

Dexamethasone Suppression Test Result	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T LS MEAN 1=LS MEAN 2
Normal	.58	.0001****	.1542
Abnormal	.38	.0004****	

TABLE 12 (Continued)

Treatment	Dexamethasone Suppression Test	Difference Scores	PROB > T HO:LS MEANS	PROB I/J	HO: LS MEAN (I) = LS MEAN (J)			
Cognitive Therapy (1)	Normal	.81	.0001****	1	___	.0916	.0145**	.0071**
Cognitive Therapy (2)	Abnormal	.49	.0011***	2	___	____	.4578	.2936
Antidepressant Medication (3)	Normal	.34	.0132**	3	___	____	____	.7360
Antidepressant Medication (4)	Abnormal	.28	.0517*	4	___	____	____	____

* $p \leq .05$
** $p \leq .01$
*** $p \leq .001$
**** $p \leq .0001$

TABLE 13

Group Means for Pre-Intervention and Post-Intervention Scores on the Personal Beliefs Inventory

Group[a]	Pre	Post	
1	X = 3.2	X = 2.4	X = 2.8
2	X = 3.1	X = 2.8	X = 2.95
3	X = 3.2	X = 2.7	X = 2.95
4	X = 3.1	X = 2.8	X = 2.95
	X = 3.15	X = 2.67	X = 2.95

[a]Group 1 = Normal Dexamethasone Suppression Test; Cognitive Therapy

Group 2 = Normal Dexamethasone Suppression Test; Antidepressant Therapy

Group 3 = Abnormal Dexamethasone Suppression Test; Cognitive Therapy

Group 4 = Abnormal Dexamethasone Suppression Test; Antidepressant Therapy

TABLE 14

One-Way (Treatment) Analysis of Variance With Least Squared Means on Difference Scores for Subjects With Normal Dexamethasone Suppression Test Results

SOURCE	df	MS	F
Treatment	1	.279	.22
Subjects (Treatment)	21	1.24	

Least Squared Means

Treatment	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T HO: LS MEAN 1=LS MEAN 2
Cognitive Therapy	.025	.9388	.6403
Antidepressant Medication	.245	.4729	

* $p \leq .05$

** $p \leq .01$

*** $p \leq .001$

**** $p \leq .0001$

TABLE 15

One-Way (Treatment) Analysis of Variance With Least Squared Means on Difference Scores for Subjects With Abnormal Dexamethasone Suppression Test Results

SOURCE	df	MS	F
Treatment	1	1.57	.58
Subjects (Treatment)	18	2.70	

Least Squared Means

Treatment	Difference Scores	PROB $\geq$ T HO:LS MEAN=0	PROB $\geq$ T HO: LS MEAN 1=LS MEAN 2
Cognitive Therapy	1.28	.0242*	.4565
Antidepressant Medication	1.84	.0024**	

* p $\leq$ .05

** p $\leq$ .01

*** p $\leq$ .001

**** p $\leq$ .0001

TABLE 16

Group Means for Pre-Intervention and Post-Intervention Scores from the Dexamethasone Suppression Test

Group[a]	Pre	Post	
1	X = .52	X = .50	X = .51
2	X = 1.2	X = .94	X = 1.07
3	X = 5.8	X = 4.5	X = 5.15
4	X = 5.5	X = 3.7	X = 4.60
	X = 3.25	X = 2.41	X = 2.83

[a]Group 1 = Normal Dexamethasone Suppression Test; Cognitive Therapy

Group 2 = Normal Dexamethasone Suppression Test; Antidepressant Therapy

Group 3 = Abnormal Dexamethasone Suppression Test; Cognitive Therapy

Group 4 = Abnormal Dexamethasone Suppression Test; Antidepressant Therapy

TABLE 17

2 (Presence or Absence of Melancholia) x 2 (Treatment)

Multivariate Analysis of Variance

Using Difference Scores for the Global Measures of Depression

SOURCE	WILKS' LAMBDA	df	F	p
Treatment	.966	3,37	.43	.73
Presence or Absence of Melancholia	.764	3,37	3.81	.01**
Treatment x Presence or Absence of Melancholia	.966	3,37	.43	.73
Subject (Treatment x Presence or Absence of Melancholia)				

* $p \leq .05$

** $p \leq .01$

*** $p \leq .001$

**** $p \leq .0001$

TABLE 18

2 (Presence or Absence of Melancholia) x 2 (Treatment) Univariate Analysis of Variance on Difference Scores from the Depression Scale of the Minnesota Multiphasic Personality Inventory With Least Squared Means

SOURCE	df	MS	F
Treatment	1	.75	.04
Presence or Absence of Melancholia	1	75.3	4.49*
Treatment x Presence or Absence of Melancholia	1	16.9	.32
Subject (Treatment x Presence or Absence of Melancholia)	39	16.8	

Least Squared Means

Treatment	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T HO: LS MEAN 1=LS MEAN 2
Cognitive Therapy	12.1	.0001****	.9210
Antidepressant Medication	11.9	.0001****	

Melancholia	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T HO: LS MEAN 1=LS MEAN 2
Without	13.6	.0001****	.0473*
With	10.7	.0001****	

TABLE 18 (Continued)

Treatment	Melancholia	Difference Scores	PROB > T HO:LS MEANS	PROB I/J	HO: LS MEAN (I) = LS MEAN (J)			
Cognitive Therapy(1)	Without	14.1	.0001****	1	___	.0314*	.3690	.1541
Cognitive Therapy(2)	With	10.1	.0001****	2	___	___	.1560	.5726
Antidepressant Medication (3)	Without	11.3	.0001****	3	___	___	___	.4784
Antidepressant Medication (4)	With	11.3	.0001****	4	___	___	___	___

* $p \leq .05$
** $p \leq .01$
*** $p \leq .001$
**** $p \leq .0001$

TABLE 19

2 (Presence or Absence of Melancholia) x 2 (Treatment)

Univariate Analysis of Variance on Difference Scores

from the Beck Depression Inventory

With Least Squared Means

SOURCE	df	MS	F
Treatment	1	19.2	.33
Presence or Absence of Melancholia	1	532.6	9.05**
Treatment x Presence or Absence of Melancholia	1	.63	.01
Subject (Treatment x Presence or Absence of Melancholia)	39	58.8	

Least Squared Means

Treatment	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T LS MEAN 1=LS MEAN 2
Cognitive Therapy	17.3	.0001****	.4265
Antidepressant Medication	15.4	.0001****	

Melancholia	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T LS MEAN 1=LS MEAN 2
Without	19.9	.0001****	.0046**
With	12.7	.0001****	

TABLE 19 (Continued)

Treatment	Melancholia	Difference Scores	PROB $\geq$ T HO:LS MEANS	PROB I/J	HO: LS MEAN (I) = LS MEAN (J)		
Cognitive Therapy (1)	Without	20.8	.0001****	1	___	.0314*	.5676	.0138**
Cognitive Therapy (2)	With	13.7	.0001****	2	___	____	.1097	.5714
Antidepressant Medication (3)	Without	19.1	.0001****	3	___	____	____	.0393*
Antidepressant Medication (4)	With	11.6	.0003****	4	___	____	____	____

* p $\leq$ .05
** p $\leq$ .01
*** p $\leq$ .001
**** p $\leq$ .0001

TABLE 20

2 (Presence or Absence of Melancholia) x 2 (Treatment)

Univariate Analysis of Variance on Difference Scores

from the Lubin Depression Adjective Checklist

With Least Squared Means

SOURCE	df	MS	F
Treatment	1	14.1	.37
Presence or Absence of Melancholia	1	275.8	7.12**
Treatment x Presence or Absence of Melancholia	1	3.29	.09
Subject (Treatment x Presence or Absence of Melancholia)	39	38.7	

Least Squared Means

Treatment	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T HO: LS MEAN 1=LS MEAN 2
Cognitive Therapy	10.6	.0001****	.6509
Antidepressant Medication	11.5	.0001****	

Melancholia	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T HO: LS MEAN 1=LS MEAN 2
Without	13.6	.0001****	.0117**
With	8.4	.0001****	

TABLE 20 (Continued)

Treatment	Melancholia	Difference Scores	PROB > |T| HO:LS MEANS	PROB I/J	HO: LS MEAN (I) = LS MEAN (J)			
Cognitive Therapy (1)	Without	13.4	.0001****	1	___	.0380*	.8931	.1468
Cognitive Therapy (2)	With	7.6	.0007****	2	___	___	.0268*	.6405
Antidepressant Medication (3)	Without	13.8	.0001****	3	___	___	___	.1151
Antidepressant Medication (4)	With	9.1	.0004****	4	___	___	___	___

* $p \leq .05$
** $p \leq .01$
*** $p \leq .001$
**** $p \leq .0001$

TABLE 21

2 (Presence or Absence of Melancholia) x 2 (Treatment)

Univariate Analysis of Variance on Difference Scores

from the Personal Beliefs Inventory

With Least Squared Means

SOURCE	df	MS	F
Treatment	1	1.34	6.60**
Presence or Absence of Melancholia	1	.08	.41
Treatment x Presence or Absence of Melancholia	1	.16	.78
Subject (Treatment x Presence or Absence of Melancholia)	39	.20	

Least Squared Means

Treatment	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T HO: LS MEAN 1=LS MEAN 2
Cognitive Therapy	.65	.0001****	.0276*
Antidepressant Medication	.32	.0038**	

Melancholia	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T HO: LS MEAN 1=LS MEAN 2
Without	.53	.0001****	.5643
With	.44	.0004***	

TABLE 21 (Continued)

Treatment	Melancholia	Difference Scores	PROB $\geq$ T HO:LS MEANS	PROB I/J	HO: LS MEAN (I) = LS MEAN (J)			
Cognitive Therapy(1)	Without	.75	.0001****	1	___	.2914	.0128*	.0594*
Cognitive Therapy(2)	With	.54	.0008***	2	___	____	.2126	.3811
Antidepressant Medication (3)	Without	.30	.0173**	3	___	____	____	.8386
Antidepressant Medication (4)	With	.34	.0515*	4	___	____	____	____

* p $\leq$ .05
** p $\leq$ .01
*** p $\leq$ .001
**** p $\leq$ .0001

TABLE 22

One-Way (Treatment) Analysis of Variance With Least Squared Means on Difference Scores from the Dexamethasone Suppression Test for Subjects Without Melancholia

SOURCE	df	MS	F
Treatment	1	1.8	.65
Subjects (Treatment)	25	2.7	

Least Squared Means

Treatment	Difference Scores	PROB > T HO:LS MEAN=0	PROB > T HO: LS MEAN 1=LS MEAN 2
Cognitive Therapy	.20	.6664	.4269
Antidepressant Medication	.71	.1185	

* $p \leq .05$

** $p \leq .01$

*** $p \leq .001$

**** $p \leq .0001$

TABLE 23

One-Way (Treatment) Analysis of Variance With Least Squared Means on Difference Scores from the Dexamethasone Suppression Test for Subjects With Melancholia

SOURCE	df	MS	F
Treatment	1	.69	.49
Subjects (Treatment)	14	1.42	

Least Squared Means

Treatment	Difference Scores	PROB $\geq$ T HO:LS MEAN=0	PROB $\geq$ T HO: LS MEAN 1=LS MEAN 2
Cognitive Therapy	1.2	.0108**	.4966
Antidepressant Medication	1.6	.0034**	

* p $\leq$ .05

** p $\leq$ .01

*** p $\leq$ .001

**** p $\leq$ .0001

APPENDIX N

Consent for Use of Audio-Tapes

Dennis L. McKnight, M.A., has my permission to use audio-tapes that will be made while I am participating in the research involving the assessment and treatment of depression for purposes of psychiatric/ psychological research, professional training, or professional consultation. I understand that undergraduate or graduate studens enrolled in psychology courses at UNC-G may view or listen to my tapes for these purposes.

I further understand that other than for the purposes above, these recordings will be treated as strictly private and confidential material. In addition, I also understand that at no time will these audio-tapes be identified by my name.

I hereby expressly waive any possible claim on my part to damages in any form in connection therewith.

Signature: _____

Witness: _____

Date: _____

APPENDIX O

COGNITIVE-BEHAVIORAL APPROACH TO TREATING DEPRESSION

Session #1

Steps

1. Therapist introduces himself and reviews treatment contract that was agreed on during the last screening interview.

2. The agenda for the first session is outlined, and includes the following:

   a. Description of the therapy used in this project.

   b. Allowing the client an opportunity to describe the problems which were involved in his/her decision to participate in this project (e.g., problems related to their depression).

   c. Emphasizing in this therapy the learning of a skill (detecting, monitoring and correcting dysfunctional thoughts) to cope with depression.

   d. Preparing the subject to do the assigned homework.

3. The therapist gives a general rationale and description of cognitive-behavioral therapy, focusing on the following points:

   a. The treatment to be received in this project is called cognitive-behavioral therapy.

   b. Main idea is: What people think influences the way they feel and the way they behave.

c. This therapy assumes that as depressed people develop, they learn to take a negative view of themselves (e.g., "I'm no good"), of the world (e.g., "The world's unfair"), and of the future (e.g., "Things won't work out").

d. Within this negative view, a depressed person has certain assumptions they utilize when stressed, and these assumptions influence the way depressed people deal with the world and what they think of themselves (e.g., "I'm not good at anything", "I can't get along with anybody").

e. These assumptions are unique to each depressed individual.

f. Although depression is a serious disorder, research has suggested that cognitive-behavioral therapy is an effective treatment approach.

g. Finally, the therapist again covers issues of confidentiality, as was done in the screening sessions.

4. The therapist provides a rationale for homework:

a. Homework is a vital part of therapy, and there is some suggestion that homework is instrumental in maintaining client's improvement after termination.

b. Homework allows clients to practice what they learn in the session in their every day world.

c. Homework provides useful information for the sessions, such as the client's weekly activities and how the therapy is progressing.

d. Completing the homework is vital to the therapy and the research project. The contract concerning the deposit and payback for homework and attendance is stressed.

5. Subject is asked to describe problems associated with their depression, and their decision to participate in the research project.

6. The therapist introduces the concepts "cognition" and "automatic thoughts" by stating that treatment will begin by learning a new skill (e.g., to detect and to self-monitor automatic thoughts). The therapist notes that the first two sessions will be spent learning to detect and monitor automatic thoughts. The last six sessions will be spent learning skills to cope with negative automatic thoughts.

a. A "cognition" is defined as either a thought or a visual image that you may not be very aware of unless you focus your attention on it. In depression, these cognitions are called "automatic thoughts" and have a negative theme. Some characteristics of automatic thoughts are as follows:

1. They are automatic, in other words they just seem to occur.
2. They are based on a low opinion of oneself.
3. They are unreasonable, inaccurate, and dysfunctional although they seem plausible at the time--the more one believes them, the more discomfort they cause.
4. They are involuntary, in other words one has difficulty turning them off.

b. The therapist further elaborates the relationship between thoughts, feelings, and behavior.

   1. Therapist illustrates relationship by contrasting differences between thoughts and feelings when one is at home alone in the evening and hears a noise and thinks, "It's a burglar" vs. "It's my spouse".
   2. The therapist asks client to shut eyes and imagine an unpleasant scene and note her emotional response. Therapist gives some instruction with pleasant scene and stresses contrast.
   3. Other examples of negative automatic thoughts may be:
      a. "Being depressed means I'm weak."
      b. "I should be able to solve this alone."
      c. "I'll never meet all the requirements of the project."
      d. "The therapist probably won't like me."

c. The therapist gives the following to aid in identifying automatic thoughts:

   1. Increases in negative and positive emotions.
   2. Troublesome life situations or events.

d. Therapist attempts to elicit automatic thoughts from client by asking, "Would you share the thoughts you had prior to the session today?" (Can be related to the session today or the client's depression.)

7. Therapist provides rationale for the following homework assignment (e.g., self-monitoring automatic thoughts) and passes out Daily Record of Dysfunctional Thoughts--Form I (one record for each day; one completed sample record).

a. Automatic thoughts are the core of cognitive-behavior therapy, so it is important to identify them. The Daily Record of Dysfunctional Thoughts--Form I will aid in meeting this goal.

b. This form should be completed every day each time your emotions change (e.g., feel happy or sad, calm or anxious) or each time you experience depression. Ideally the form should be completed when the automatic thoughts occur; however, if this is impossible, you need to have a standard time each day (e.g., 15 minutes after supper) to complete the form. You will need to make several entries each day since we will use this information during the next session.

c. Therapist explains how to complete all parts of the Daily Record of Dysfunctional Thoughts--Form II by referring to sample form he passes out.

1. A positive or negative change in emotion or a depressed mood is a cue to complete the form. Therefore, complete the "emotion" column first (e.g., describe emotion and rate its degree).

2. Fill in the date.

3. Complete the "situation" column (e.g., describe event and thoughts preceding the emotion).

4. Complete the "automatic thoughts" column (e.g., describe the negative thoughts that preceded the emotion and rate its believability).

d. Therapist answers questions and has client practice several examples.

8. The therapist raises the issue that negative automatic thoughts can occur during treatment. For example, negative thoughts may occur in relation to the treatment sessions, the therapist, or the homework. If such automatic thoughts occur, it is important that you record them and bring them and bring them up for us to discuss.

a. The therapist attempts to elicit examples from the client.

b. The therapist provides typical examples taken from Beck, Rush, Shaw, and Emery et al., 1979, Chapter 14. See Handout entitled "Examples of Negative Automatic Thoughts Regarding Therapy."

c. The client discusses negative automatic thoughts concerning therapy from both sources a and b.

9. The therapist asks each client to describe her thoughts regarding the homework assignment.

Session #2

1. The therapist reviews the last session along with the homework and praises her completion of the task. (If a client did not complete the task, she is instructed to make at least three entries relevant to dysphoric mood or positive or negative change in affect.) The therapist administers the Depression Adjective Checklist. (Allow 5 minutes.)

2. The therapist outlines the following agenda:

   a. Introduction to new concepts--depressive assumptions.

      1. What they are.

      2. How to identify them.

   b. Review of homework.

      1. Individual identified themes.

      2. Client learns how to identify logical errors in an effort to identify depressive assumptions.

      3. Individual identifies assumptions.

   c. Assign homework. (Allow 5 minutes.)

3. The therapist defines, describes, and stresses the importance of depressive assumptions: Faulty assumptions appear to be involved in the likelihood that a person will become depressed. It is important that we detect these faulty assumptions to decrease the chance that you will become depressed in the future. In order to identify these depressive assumptions, we will pay particular attention to the automatic thoughts which you have recorded. Often common "themes" can be identified from the automatic thoughts. Yet, every person has her own set of assumptions which they probably learned during childhood from their parents or peers. For example, a parent may say to the child,

"Be nice or Nancy won't like you." After repeating such phrases the child may develop a more general rule: "My worth depends on what others think of me." Examples of faulty assumptions that increase the chance that a person will become depressed include (from Beck et al., 1979):

a. "In order to be happy, I have to be successful in whatever I undertake."

b. "To be happy, I must be accepted by all people at all times."

c. "If I make a mistake, it means that I am inept."

d. "I can't live without you."

e. "If somebody disagrees with me, it means he doesn't like me."

f. "My value as a person depends on what others think of me." (Allow 10 minutes.)

4. The therapist introduces aids for identifying depressive assumptions: "In identifying depressive assumptions it helps to use the following steps:

a. Monitor automatic thoughts.

b. Identify them.

c. Infer the primary assumption or rule.

Therapist provides illustration). For example, one client reported these automatic thoughts. "My work is of poor quality. I can't fix the bicycle. I can't cut the grass. I can't make a sale. The wallpaper wasn't lined up well."

d. What are the themes? (Performance and perfectionistic standards.)

e. What is a possible primary assumption? (My worth depends on the quality of my work.) (Allow 5 minutes.)

5. The therapist introduces the next exercise. "We will use these steps (4a-c) to help you identify your depressive assumptions." It is important to be looking for "signals" that depressive assumptions may be occurring. Helpful signals include:

a. The frequent use of global, vague words (e.g., stupid, silly, dumb).

b. "Absolute words" (e.g., never, always, should).

c. "Logical errors" or "thinking errors."

Therapist passes out hand-out entitled, "Logical Errors or Thinking Errors" and discusses it. (Allow 15 minutes.)

6. The therapist suggests that the client share her Daily Record of Dysfunctional Thoughts using the following framework:

a. Look back over your homework and identify any common themes and/or assumptions. (If necessary, self-monitoring from Session 1 and 2 can also be reviewed.)

b. Try to identify signals of depressive assumptions.

c. On a piece of paper, we'll fill in this diagram for each person:

1. emotions

2. automatic thoughts

3. themes

4. depressive assumptions

d. Subjects are instructed to copy their diagram on the back of a Daily Record of Dysfunctional Thoughts.

If a client gets "stumped," the following questions may be helpful:

1. What made you particularly happy or unhappy about this event? (e.g., "I did well because someone praised me.")
2. How do you look at the behavior of others? (e.g., "Mary is happy because she has a husband.")
3. How are you justifying your feelings? (e.g., "Anyone who always makes mistakes would feel this depressed.")

(Allow 45 minutes.)

7. The therapist collects Daily Record of Dysfunctional Thoughts--Form I and passes out blank records. The therapist instructs the client to continue to complete the forms as usual, but at the end of each day, on the back of the form, identify:

a. Common themes.

b. Depressive assumptions. (Allow 5 minutes.)

Session #3

1. The therapist collects the client's homework from the previous session. The therapist praises the completion of this task. The therapist reviews the client's homework (also praising completion). If a client did not complete the homework assignment, she is instructed to make at least three entries relevant to dysphoric mood or positive or negative change in affect. The therapist administers the Depression Adjective Checklist. (Allow 15 minutes.)

2. The therapist outlines the following agenda for this session:

   a. Description of next stop in therapy--evaluating and correcting dysfunctional thoughts.

   b. Group discussion of alternative explanations using negative expectations about therapy as an example.

   c. Review homework looking for alternative explanations or for negative thoughts.

   d. Assign homework. (Allow 5 minutes.)

3. The therapist describes the next step in treatment: We have been practicing and will continue to practice detecting automatic thoughts and depressive assumptions because we think that there is a relationship between feeling depressed and looking at the self, the world, and the future in a negative manner. However, just as important as the skill of identifying depressive thoughts and assumptions is the skill of correcting them. The goal of this step in therapy is for you to examine the evidence for and against your thoughts, using standards which a nondepressed person would use. Some of the steps which are important in <u>correcting negative automatic thoughts</u> include:

   a. Recognizing that thoughts and beliefs are inferences about the would rather than facts.

   b. Examining the logical evidence for and against the thought or belief.

   c. Providing an alternative response to the negative cognition. (Allow 10 minutes.)

4. The therapist begins discussion of some of the negative thoughts which may occur in relation to therapy: In Session 1 we noted that negative automatic thoughts can occur in relation to therapy, the therapist, or homework.

a. What were some of the examples we raised? (If needed, the therapist refers to the Handout entitled "Examples of Negative Automatic Thoughts Regarding Therapy.")

b. What evidence is there to support and to refute the thought?

c. What are some alternative explanations for each thought? (See Chapter 14 in Beck et al., 1979, for alternative explanations.)

For example, regarding the following negative automatic thought: "You are more interested in doing research than in helping me":

1. Evidence to support--the project does involve research. Evidence to refute--the research and the treatment are not incompatible.

2. Alternative response--"My participation in this research- treatment project stands to help me and to help others as researchers learn more about depression, its assessment, and treatment." (Allow 15 minutes.)

5. The therapist suggests that the client share her Daily Record of Dysfunctional Thoughts using the following framework:

a. Identify your negative automatic thoughts.

b. Describe the evidence you have to support and to refute the thoughts.

c. Suggest an alternative interpretation for your negative automatic thoughts.

If you get "stumped" in suggesting an alternative response, the following questions may aid you:

1. What part of this situation is a fact and what part is my belief?

2. How would a nondepressed person evaluate this event?

3. Even if it is true, is it as bad as it seems?

Note: Again the therapist's major activity is asking questions rather than making statements. (Allow 30 minutes.)

6. The therapist collects Daily Record of Dysfunctional Thoughts--Form I and passes out the Daily Record of Dysfunctional Thoughts--Form II. Therapist passes out a completed sample of Form II. Therapist instructs clients to:

a. Complete this form every day each time you feel sad and depressed or each time your emotions change. Ideally the form should be completed when the automatic thoughts occur; however, if this is impossible you need to have a standard time each day (e.g., 15 minutes after supper) to complete the form. You need to make several entries each day since we will use these data in the next session.

(See Session #1 for directions on how to complete the first four columns of the Daily Record of Dysfunctional Thoughts.)

b. Provide a "RATIONAL RESPONSE" to each automatic thought and to rate the believability of the response. (Therapist reminds group of questions to aid alternative, rational response.)

c. Write the "OUTCOME" of the automatic thought (i.e., re-rate believability and emotion.)

d. Therapist explains how to complete all parts of the form by reviewing the sample; answers questions; has the group practice one entry. (For a - c allow 15 minutes.)

Session #4

1. The therapist reviews the client's homework and praises her completion of the task. (If a client did not complete the task, she is instructed to make at least three entries relevant to dysphoric mood or positive or negative change in affect and to supply the rational responses to go with each negative automatic thought.) The therapist administers the Depression Adjective Checklist. (Allow 5 minutes.)

2. The therapist outlines the following agenda:

a. Review the steps of and rationale for providing alternatives to automatic thoughts.

b. Review depressive assumptions acknowledging the fact that they are difficult to give up, but suggesting skills for coping with depressive assumptions.

c. Review homework; identify depressive assumptions from homework, their pros and cons and long-term and short-term consequences; supply alternatives.

d. Assign homework. (Allow 5 minutes.)

3. The therapist begins review of the skills covered in Session #3: During the last session we focused on correcting negative automatic thoughts. As a brief review, I wonder if you could tell me:

a. Why is it important to evaluate and to correct negative automatic thoughts?

b. What are some of the steps involved in correcting negative automatic thoughts?

c. What types of questions might you ask yourself if you have difficulty providing an alternative response to a negative authomatic thoughts? (Allow 10 minutes.)

4. The therapist begins a review of depressive assumptions and the importance of evaluating them and providing alternative responses to them: During this session, we will apply the skills that we have been practicing to depressive assumptions. You may remember that depressive assumptions are important because their presence and use increases the likelihood that a person will become depressed. Some examples of the depressive assumptions we talked about included: "To be happy, I must be accepted by all people at all times." "If I make a mistake, it means that I an inept." We mentioned that the following cues often signal the presence of depressive assumptions:

a. The frequent use of global, vague words (e.g., stupid, silly, dumb).

b. The frequent use of "absolutes" (e.g., should, ought, never).

c. "Logical errors" (e.g., overgeneralization, magnification).

In identifying depressive assumptions, we examined the common themes of negative automatic thoughts and inferred the depressive assumptions. (Allow 5 minutes.)

5. The therapist provides rationale to be used in this session: Since depressive assumptions are important in the reoccurrence of depression, we are going to practice evaluating the logical evidence for and against the assumptions, and reevaluate the depressive assumptions. However, it is first important to recognize that it is difficult to "give up" an assumption or rule you have used your entire life which you may have learned from someone very significant to you. In order to cope with this reluctance we will examine the pros and cons, and the long-term and short-term consequences of each of the depressive thoughts that you identify.

The therapist applies the above to the following depressive assumptions: "I'm only as good as my work."

a. Short-term consequences: work hard, promoted.

b. Long-term consequences: looses job, thinks he is a loser.

c. Pros: encourage effort.

d. Cons: insecure when job is insecure; effort seems motivated by fear. (Allow 5 minutes.)

6. The therapist suggest that each client share her Daily Record of Dysfunctional Thoughts using the following framework:

a. Look back over your homework and identify any common themes and/or assumptions.

b. The therapist and client fill in this diagram:

1. emotions
2. automatic thoughts
3. themes
4. depressive assumptions
5. advantages of keeping this assumption
6. disadvantages of keeping this assumption
7. short-term effects of operating under this assumption
8. long-term effects of operating under this assumption
9. alternative assumption that is more useful than the depressive assumption.

c. Subjects are instructed to copy their diagram on the back of a Daily Record of Dysfunctional Thoughts Form.

Note: Again, the therapists asks many questions during this section and makes few statements. (Allow 55 minutes.)

7. The therapist collects Daily Record of Dysfunctional Thoughts--FormIi and passes out blank forms. The therapist instructs the client to continue to complete the forms as usual, but at the end of each day, on the back of the form, identify:

a. Common themes.

b. Depressive assumptions.

c. Alternatives to the depressive assumption. (Allow 5 minutes.)

Session #5

1. The therapist reviews each client's homework, and praises her completion of the task. (If a client did not complete the task, she is instructed to make at least three entries relevant to dysphoric mood or positive or negative change in affect, to note the common themes and to infer the depressive assumptions, and to provide alternative rational responses to each automatic thought and depressive assumption.) The therapist administers the Depression Adjective Checklist. (Allow 10 minutes.)

2. The therapist outlines the following agenda:

a. Review logical errors.

b. Describe the skills one can use to cope with logical errors.

c. Review homework; look for logical errors; apply skills to cope with logical errors in offering alternative to negative automatic thoughts and depressive assumptions. (Allow 5 minutes.)

3. The therapist begins a review of "logical errors" or "thinking errors": In our early sessions, we discussed "logical errors" or "thinking errors" as signals of depressive assumptions. During this

session we will review these logical errors and will practice skills designed to cope with them or decrease their likelihood. (Allow 5 minutes.)

4. The therapist destributes handout entitled, "Skills to Cope with Logical Errors." For each of the seven cognitive errors, the therapist:

   a. Describes the error.

   b. Gives an example of the error.

   c. Elicits examples from the client.

   d. Describes the skill used to cope with the cognitive error. (Allow 20 minutes.)

5. The therapist suggest that the client share her Daily Record of Dysfunctional Thoughts--Form II using the following framework:

   a. Look back over your homework and identify any of the logical errors we have discussed.

   b. We will fill in this diagram as we go:

      1. emotions
      2. automatic thoughts
      3. themes
      4. depressive assumptions and logical errors
      5. skills to cope with logical errors
      6. an alternative assumption that is more useful than the depressive assumption

   c. Subjects are instructed to copy their diagram on the back of a Daily Record of Dysfunctional Thoughts--Form II.

   Note: Again the therapist asks many questions during this section and makes few statements. (Allow 45 minutes.)

6. The therapist collects Daily Record of Dysfunctional Thoughts--Form II and passes out blank forms. The therapist instructs the client to continue to complete the form as usual and at the end of each day, on the back of the form, identify:

a. Common themes.

b. Depressive assumptions.

c. Alternatives to the depressive assumptions. (Allow 5 minutes.)

Session #6

1. The therapist collects the client's homework forms. (If a client did not complete the homework assignment, she is instructed to make at least three entries relevant to dysphoric mood or positive or negative change in affect.) The therapist administers the Depression Adjective Checklist. (Allow 5 minutes.

2. The therapist outlines the following agenda:

a. Description of the next step in therapy--evaluating and correcting dysfunctional thoughts and assumptions by designing experiments.

b. Steps involved in designing experiments.

c. Examples of experiments.

d. Discuss new homework assignment.

e. Practice new homework assignment. (Allow 5 minutes.)

3. The therapist describes the next step in treatment: We have been practicing and will continue to practice detecting automatic thoughts and depressive assumptions because we think that there is a

relationship between feeling depressed and looking at the self, the world, and the future in a negative manner. To review, you may remember that depressive assumptions are important because their presence and use increases the likelihood that a person will become depressed. Some examples of the depressive assumptions we talked about included: "To be happy, I must be accepted by all people at all times." "If I make a mistake, it means that I am inept."

We mentioned that the following cues often signal the presence of depressive assumptions:

a. The frequent use of global, vague words (e.g., stupid, silly, dumb).

b. The use of "absolutes" (e.g., should, ought, never).

c. "Logical errors" (e.g., overgeneralization, magnification).

In identifying depressive assumptions, we examined the common themes of negative automatic thoughts and inferred the depressive assumptions. However, just as important as the skill of identifying depressive thoughts and assumptions is the skill of correcting them. Since we have stated earlier that there is a difference between a thought and a fact, we will try now to subject thoughts to an experimental test. We will look at thoughts as hypotheses to be treated empirically and will gather data to refute and/or to support the hypotheses. (Allow 5 minutes.)

4. The therapist illustrates: For example, one depressed personl used the assumption--"If I assert myself (express myself openly and honestly), I will be rejected." The negative automatic thoughts which went along with this assumption were--"If I tell my supervisor I want to take the day off she will think that I am lazy and that I'm trying to avoid work." The experiment consisted of actually talking with the supervisor, recording what happens, and comparing these results with the predictions.

A depressed student predicted that she would be a failure in college because her English professor suggested many revisions on her essay. One of her automatic thoughts included--"The professor probably wishes I wasn't in his class since I am donig so poorly." The experiment consisted of going to talk with the professor, who said that the student's paper was very creative, and it needed revising. He pointed out that he had written a lot to guide her revisions and make them easier. (Allow 5 minutes.

5. The therapist mentions that there are several types of experiments. Some automatic thoughts are examined best by taking data on oneself (like the two outlined above). Other automatic thoughts are tested best by "surveying" others. For example, one depressed woman assumed: "Only unattractive women go out alone." When this client actually counted the numbers of attractive women who went out alone vs. the number of unattractive women who went out together, she found the numbers were approximately equal. (Allow 5 minutes.)

6. The therapist outlines the steps involved in testing assumptions:

   a. Identify the depressive or faulty assumption to be tested.

   b. Deduce a specific prediction from this general rule (often it helps to look at the automatic thoughts in order to deduce a specific prediction).

   c. State this prediction in a form that can be tested. Define vague terms and list behaviors necessary to carry out the test. Look at the situation in which corresponding negative, automatic thoughts occur for ideas about how to specify the hypothesis.

   d. Record the results from the experiment in an objective manner. That is, record the outcomes of the experiment in terms of what happened, rather than in terms of what you think about what happened.

   e. Compare the results you got to the prediction that you made.

   f. Ask yourself if other experiments are necessary. (Allow 10 minutes.)

7. The therapist introduces the new homework assignment Daily Record of Dysfunctional Thoughts--Form II as an aid in learning to test assumptions and/or negative, automatic thoughts.

a. The therapist points out that the first four columns (e.g., date, situation, emotions, automatic thoughts) are identical to Forms I and II. The therapist remonds the client that the cues for completing the form are dysphoria or a change in emotion. If it is impossible to complete the form at that moment, go back to the form at a standard time each day.

b. The therapist mentions that column five, "WAYS TO TEST," (the negative thought or depressive assumption) involves creating a method which would support or refute the thought. This column is used to specify how you will collect your data.

c. The therapist mentions that column six "OUTCOME OF TEST" involves recording the results of the experiment. Clients are encouraged to record the results of your experiment like "you would like for a newspaper report to report the news."

d. The therapist mentions that column seven "THOUGHTS AND BELIEFS" involves re-rating the belief in the initial automatic thought or assumption and specifying and rating the new emotion. (Allow 10 minutes.)

8. The therapist suggests that the client uses the new Form III to review her homework from the last session. The following format is used:

a. What depressive assumption would you like to test? (If client can't identify a depressive assumption, the therapist reviews. Such review is accomplished by listing emotions, automatic thoughts, themes, and deducing the assumptions.)

b. What specific prediction can you deduce from this general assumption? (Aids: Look at corresponding situations and automatic thoughts.)

c. How can we state this prediction in a testable form? (Define vague terms. List behaviors necessary to carry out the test.)

d. Why type of data would you record? Are there any precautions you might take to make sure these data are objective?

e. If any applicable examples arise, the therapist has group members conduct the experiment in group setting. In so doing the client practices:

   1. Recording data objectively.
   2. Comparing the results with the prediction.
   3. Asking if other experiments are necessary.

When this is done, the therapist makes sure that the client has another or similar experiment to conduct as homework. (Allow 40 minutes.)

9. The therapist assigns homework:

   a. Carry out the experiments which you designed and record the results.

b. Complete the Daily Record of Dysfunctional Thoughts--Form III. Therapist gives each client a completed sample of Form III. Complete Columns 1-5 each time you feel dysphoric or your emotions change. Complete Columns 6 and 7 (i.e., actually perform an experiment) once a day. (Allow 5 minutes.)

Session #7

1. The therapist reviews each client's homework and praises her completion of the task. (If a client did not complete the task, she is instructed to make at least three entries relevant to dysphoric mood or positive or negative change in affect, completing Columns 1-5, Form III. Then the therapist stresses the importance of actually carrying out the experiments and attempts to get the subjects to agree to carry out one of these experiments as her new homework.) The therapist administers the Depression Adjective Checklist. (Allow 5 minutes.)

2. The therapist outlines the following agenda:

   a. Review the rationale for and steps for hypothesis testing.

   b. Review homework (Form III).

   c. Learn a new skill which is particularly useful in testing hypotheses regarding problems (e.g., graded task assignment).

   d. Apply graded task assignment to a problem/hypothesis relevant to you.

   e. Assign homework. (Allow 5 minutes.)

3. The therapist begins review of the skills covered in Session #6: During the last session we focused on correcting negative automatic thoughts or depressive assumptions by hypothesis-testing or by setting up experiments. As a brief review, I wonder if you would tell me:

a. Why it is important to set up experiments to evaluate automatic thoughts and depressive assumptions? (Automatic thoughts and depressive assumptions are beliefs, not facts. Experiments help in establishing or refuting their validity.)

b. What are the steps involved in testing assumptions?

1. Identify the depressive or faulty assumption to be tested.

2. Deduce a specific prediction from this general rule (often it helps to look at the automatic thoughts in order to deduce a specific prediction).

3. State this prediction in a form that can be tested. Define vague terms and list behaviors necessary to carry out the test. Look at the situation in which corresponding negative, automatic thoughts occur for ideas about how to specify the hypothesis.

4. Record the results from the experiment in an objective manner. That is, record the outcomes of the experiment in terms of what happened, rather than in terms of what you think about what happened.

5. Compare the results you got to the prediction that you made.

6. Ask yourself if other experiments are necessary. (Allow 10 minutes.)

4. The therapist suggests that each client share her Daily Record of Dysfunctional Thoughts--Form III. Therapist asks each client to review one experiment, beginning with Column 1 through Column 7. If a client has not carried out an experiment, the therapist helps her design an experiment that she can carry out in the session, at this time. (Allow 30 minutes.)

5. The therapist introduces the rationale for and the steps involved in graded task assignment:

a. Rationale:

Graded task assignment offers one way of testing hypotheses that have to do with problems or doubts. This strategy is designed to help test automatic thoughts or assumptions like: "I can't do anything" or "I'll never be able to solve this problem." Graded task assignment will offer you a method of solving problems through your own effort and skill.

b. Steps:

1. Identify the problem (i.e., belief) on which you would like to work (e.g., "I can't accomplish my goals.").

2. Formulate a project. That is, write down the behaviors which are involved in the task. Start with the simplest and move to the more complicated.
3. Perform these behaviors. Check off the parts of the task as you do them.
4. Compare the results with the prediction that you made. (Allow 5 minutes.)

6. The therapist suggests that the client practice using graded task assignment to test hypotheses using this format:
   a. The therapist gives the client a chance to ventilate and to express any cynical doubts that they have regarding the utility of this task. (The therapist responds with, "This is an experiment. We can test your automatic thoughts.")
   b. The therapist suggests that clients refer to the Daily Record of Dysfunctional Thoughts--Form III during this exercise. The therapist suggests that the client write down the "plan" in the following places.
   c. Identify the assumption which can be tested through the use of graded task assignment. (Write in Column 4.)
   d. Write down the steps involved in the task, Column 5.

   Note: The therapist aids the client in setting <u>modest</u> goals. (Allow 30 minutes.)
7. For homework the therapist instructs the client to:
   a. Perform the behaviors listed in 6d, checking off the tasks as they are accomplished.

b. Complete Column 6 and 7.

c. Use graded task assignment to test at least one other belief before the next session.

d. Continue to complete Daily Record of Dysfunctional Thoughts--Form III, Columns 1-5, at least. (Allow 5 minutes.)

Session #8

1. The therapist reviews each client's homework and praises her completion of the task. (If a client did not complete the task, then she is instructed to make at least three entries relevant to dysphoric mood or positive or negative change in affect, completing Columns 1-5, Form III. Then the therapist stresses the importance of actually carrying out one of these experiments as her new homework.) The therapist administers the Depression Adjective Checklist. (Allow 5 minutes.)

2. The therapist outlines the following agenda:

a. Review the rationale and steps involved in using graded-task assignment to test hypotheses.

b. Review homework, Form III.

c. Learn a new skill which is particularly useful in testing hypotheses regarding fulfilling daily goals (e.g., activity scheduling).

d. Apply activity scheduling to a hypothesis relevant to you.

e. Assign homework. (Allow 5 minutes.)

3. The therapist begins a review of the skills covered in Session #7: During the last session we focused on correcting negative automatic thoughts related to problems or doubts by graded task assignment. As a brief review, I wonder if you would tell me:

a. What are the steps involved in graded task assignment?

1. Identify the problem (i.e., belief) on which you would like to work.

b. Formulate a project. That is, write down the behaviors which are involved in the task. Start with the simplest and move to the more complicated.

c. Perform the behaviors. Check off the parts of the task as you do them.

d. Compare the results with the prediction that you made. (Allow 5 minutes.)

4. The therapist suggests that each client share her Daily Record of Dysfunctional Thoughts--Form III. Therapist asks each client to review one experiment in which she used graded task assignment to test a hypothesis. The therapist instructs the members to review what they place in Columns 1 through 7 on the Daily Record of Dysfunctional Thoughts--Form III. If a client has not carried out an experiment using graded task assignment, the therapist helps her design an experiment that she can carry out in the session, at this time. (In some cases this may not be possible; therefore, the client is encouraged to implement the experiment as homework.) (Allow 20 minutes.)

5. The therapist introduces the rationale for and steps involved in activity scheduling:

a. Rationale:

Activity scheduling offers one way of testing hypotheses that have to do with not accomplishing enough, being unable to carry out, and not doing anything pleasurable. Activity scheduling offers a method for collecting data on these hypotheses.

b. Steps: (The therapist hands out Activity Schedules and blank Form III, asking the client to complete the steps involved in planning activities as she describes them.)

1. Identify a hypothesis you use or have used which is related to inability to accomplish daily activities and not doing anything pleasurable (e.g., "I can't get anything done" or "I don't do anything fun."). Write this hypothesis in Column 4 of the Daily Record of Dysfunctional Thoughts--Form III.
2. In Column 5 write that activity scheduling will be your method of testing the hypothesis.
3. On the Activity Schedule, go through and write down all the standing appointments you have made (e.g., go to work, come to group meeting).
4. On the Activity Schedule, go through and write down something for each day that you <u>want</u> to do (e.g., watch the evening news, play with my pet, write a letter, etc.).

5. Leave some time each day unscheduled. Right now what's more important than actually accomplishing the activity is planning the activity. Nobody accomplishes everything that she plans. Even if you don't carry out every activity, trying to carry them out and carrying out some of the activities is very important.
6. For homework on the Activity Schedule, check off the tasks as you complete them.
7. For homework on the Daily Record of Dysfunctional Thoughts--Form III in Column 6, write down the outcome of the experiment which involved scheduling activities.
8. For homework, complete Column 7 of the Daily Record of Dysfunctional Thoughts--Form III. (Allow 45 minutes.)

6. For homework the therapist instructs the client to:
   a. Carry out their experiments on Activity Scheduling (review steps 1-7 above).
   b. Continue to complete Form III, Columns 1-5, at least. (Allow 10 minutes.)

Examples of Negative Automatic Thoughts Regarding Therapy*

1. "Cognitive therapy is a rehash of 'the power of positive thinking'."
2. "I'm not depressed because I distort reality, but because things really are bad. Anyone would become depressed."
3. "I know I look at things in a negative way, but I can't change my personality."
4. "I believe what you are saying intellectually, but not emotionally."
5. "Since I don't like these negative thoughts, the reason they come must be that I want to be depressed."
6. "I'm afraid once I'm over being depressed, I'll become anxious like I was before."
7. "I want a guarantee this therapy will cure my depression."
8. "Cognitive therapy is concerned with mundane things in life and not with the serious problems that make me depressed."
9. "If negative cognitive distortions make me unhappy, does that mean that positive cognitive distortions make me happy?"
10. "I have been coming to therapy for several weeks, and I'm not any better."
11. "You can't treat my depression without seeing my spouse, too. He/she caused the depression."
12. "I'm smarter than the therapist. How can she help me?"
13. "You are more interested in doing research than in helping me."
14. "Cognitive therapy won't work because my depression is biological."
15. "I have to assert my independence by not letting the therapist get the best of me."

*Beck, Shaw, Rush, and Emery, 1979.

Skills to Cope with Logical Errors

Cognitive Error	Assumption	Skill
1. Overgeneralizing	If it's true in one case, it applies to any case which is even slightly similar.	Exposure of faulty logic. Establish criteria of which cases are "similar" and to what degree.
2. Selective abstraction	The only events that matter are failures deprivation, etc. Should measure self by errors, weaknesses, etc.	Use "log" to identify successes patient forgot.
3. Excessive responsibility (Assuming Personal Causality)	I am responsible for all bad things, failures, etc.	Disattribution technique.
4. Assuming Temporal Causality (Predicting without sufficient evidence	If it has been true in the past, then it's always going to be true	Expose faulty logic. Specify factors which could influence outcome other than past events.
5. Self-references	I am the center of everyone's attention--especially my bad performances. I am the cuase of misfortunes.	Establish criteria to determine when patient is the focus of attention and also the probable facts that cause bad experiences.
6. "Catastrophizing	Always think of the worst. It's most likely to happen to you.	Calculate real probabilities. Focus on evidence that the worst did not happen.
7. Dichotomous thinking	Everything either is one extreme or another (black or white; good or bad)	Demonstrate that events may be evaluated on a continuum.

Taken from Beck, Rush, Shaw, and Emery, 1979, p. 261.

Daily Record of Dysfunctional Thoughts--Form II

DATE	SITUATION Describe 1. Actual event leading to unpleasant emotion, or 2. Stream of thoughts, daydream, or recollection, leading to unpleasant emotion.	EMOTION(S) 1. Specify sad, anxious, angry, etc. 2. Rate degree of emotion, 1:100	AUTOMATIC THOUGHT(S) 1. Write automatic thought(s) that preceded emotions(s). 2. Rate belief in automatic thought(s). 0:100%	RATIONAL RESPONSE 1. Write rational response to automatic thought(s). 2. Rate belief in rational response. 0:100%	OUTCOME 1. Re-rate belief in automatic thought(s), 0:100% 2. Specify and rate subsequent emotions. 0:100

EXPLANATION: When you experience an unpleasant emotion, note the situation that seemed to stimulate the emotion (If the emotion occurred while you were thinking, daydreaming, etc., please note this.) Then note the automatic thought associated with the emotion. Record the degree to which you believe the thought: 0% = not at all; 100% completely. In rating degree of emotion: 1 = a trace; 100 = the most intense possible.

Adapted from Beck, Rush, Shaw, and Emery, 1979, p. 403.

APPENDIX P

Consent Form II

I understand that I am going to be interviewed by the primary researcher (Dennis L. McKnight, M.A.) and by a psychiatrist (Dr. Jarrett Barnhill), will be administered questionnaires, and will be asked to have a blood test done at a local laboratory to be used in selecting subjects for a psychological-psychiatric investigation involving the assessment and treatment of depression. I have also been informed that if I am selected for this study that my treatment may be with anti-depressant medication or a psychological treatment, whichever I am assigned. I understand that I do not have a choice as to what treatment I would receive, but I may withdraw from the study at any time. Also, I will be required to have two more blood tests taken after my treatment beings. While there is no charge for the treatment sessions, I will be asked to pay a total of $60.00 for the laboratory work and will be required to pay for medication if I receive the treatment using anti-depressant medication. Furthermore, I have been informed that I am participating in research and alternative treatment for my problem is available through my local mental health clinic or through psychologists/psychiatrist in private practice.

I understand that if I am not eligible for participation in this program I will be given a list of referrals for assessment and treatment in Winston-Salem and that I may contact if I so desire. Finally, all personal information (e.g., name) that I give is completely confidential, and will only be available to the experimenters. I further understand that specific numerical scores provided by laboratory tests and by questionnaires will be used (without my identity) for research purposes and publication. If I am eligible to participate, I understand that experimental procedures will be explained to me more fully before I decide to continue to participate.

Signed: _____

Witness: _____

Date: _____

APPENDIX Q

Consent Form III: Treatment Contract

I, _____, hereby agree to participate in research to be conducted under direction of Dr. Rosemery O. Nelson, Professor of Psychology, and Dr. Jarrett Barnhill, Psychiatrist, involving assessment and treatment for depressive disorders. As explained to me, for the next 9 weeks, I will be required to attend weekly group assessment and treatment sessions involving receiving

anti-depressant medication/psychologically-based therapy
(circle one)

treatment (groups will consist of three subjects in each). I understand that I do not have a choice in my treatment, but that I may withdraw at any time.

Although I am not paying for the treatment I receive, I have agreed to pay for laboratory tests (e.g., three blood tests), to pay for medication if necessary, and make a $50.00 "data deposit." I have agreed to have my money refunded, gradually and fully, if I come to all the sessions and participate in the treatment fully. I have also agreed to forfeit the percentage of money that matches the commitments I fail to keep. Specifically, I understand that my data deposit will be returned according to the following plan:

> If I come to all scheduled appointments, and participate in the treatments fully, my data deposit will be returned as follows:

Session #1:	$ 3.00
Session #2:	4.00
Session #3:	4.00
Session #4:	5.00
Session #5:	6.00
Session #6:	6.00
Session #7:	7.00
Session #8:	7.00
Session #9:	8.00
Total:	$50.00

I understand that if I miss a session, I may call Dennis McKnight in advance to reschedule the appointment. The rescheduled appointments should be within four days of my previous appointment. If I attend the rescheduled appointment, I will not forfeit any percentage of my data deposit.

I understand that if I become dissatisfied with this program, withdrawal can be arranged and my data deposit can be returned in full. However, I must contact Dennis McKnight before I miss a treatment session in order for my $50.00 to be returned.

I understand that the purpose of this investigation is to evaluate approaches to assessing and treating depressive disorders, approaches which have shown to be useful for certain cases in the past. However, I also realize that there can be no guarantee that I will not be depressed because I participate in this research. However, hopefully, my participation will contribute to the development of effective assessment and treatment for others, as well as for myself. In addition, if at the end of this investigation, I am not satisfied with my progress here, I will receive a referral for continued assessment and treatment.

Signed: _____

Witness: _____

Date: _____

APPENDIX R

Debriefing Statement

The general question which stimulated the investigation in which you have participated is: Is it worthwhile to view depression as two general subtypes, one subtype that results from a chemical imbalance in the brain, and the other subtype that results from faulty ways of thinking about situations? Furthermore, can a chemically based depression significantly improve using a psychological treatment (e.g., changing the way one views different situations that arise) or must medication be used? Likewise, can a supposedly reactive depression (e.g., depression resulting from faulty ways of thinking about the world) improve using medications, or must it be treated with a psychological treatment?

As you may remember, I asked you to take the Dexamethasone Suppression Test (DST test for short) at the beginning and end of this study. This test is believed to differentiate between the subtypes of depression mentioned above. Positive suppressors are believed to be indicative of a chemical depression, while negative suppressors are indicative of a reactive depression. Some subjects in this study received an anti-depressant medication as their main form of treatment, while other subjects received a psychological treatment. Therefore, this study examined whether positive suppressors responded better, equally well, or not as well to medications or to a psychological treatment. It also examined whether negative suppressors responded better, equally well, or not as well to medications or to a psychological treatment.

Each of the treatment approaches used in this study have shown to be effective in the treatment of depression. Treatment effectiveness in this investigation was assessed through the questionnaires and laboratory tests that you took.

Although you may have been told in this study that your data deposit would be refunded only under certain circumstances, the data deposit was refunded, in full, to <u>all</u> subjects.

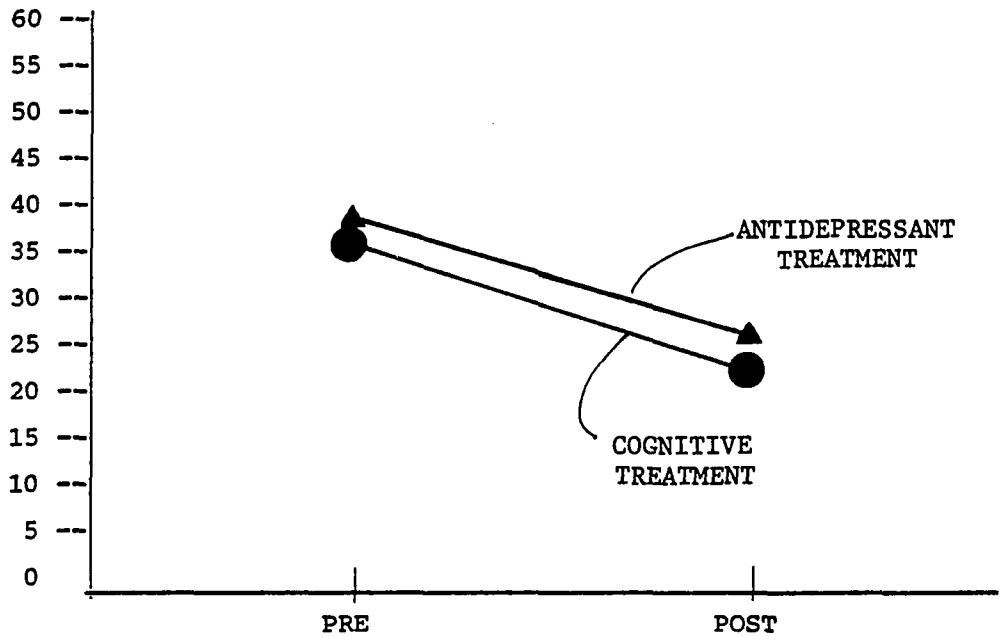
Following the termination of this study, you were given a referral list for possible further evaluation and treatment, if you so desired. Feel free to call any of these telephone numbers or Charter Mandala Center if further treatment is necessary.

APPENDIX S

FIGURES

FIGURE 1

DEPRESSION SCALE OF MMPI: NORMAL DEXAMETHASONE SUPPRESSION TEST



DEPRESSION SCALE OF MMPI: ABNORMAL DEXAMETHASONE SUPPRESSION TEST

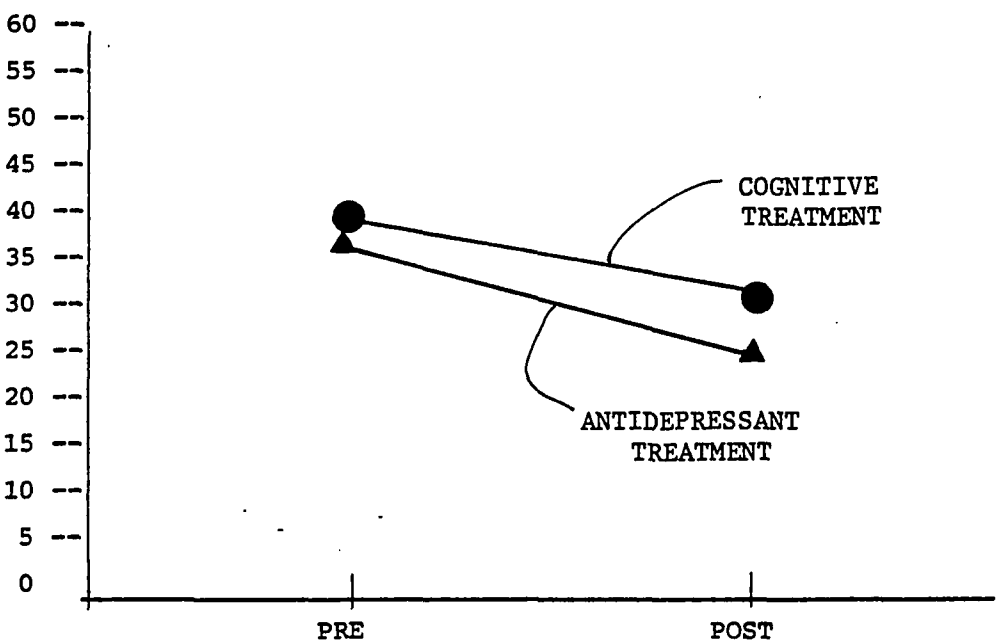
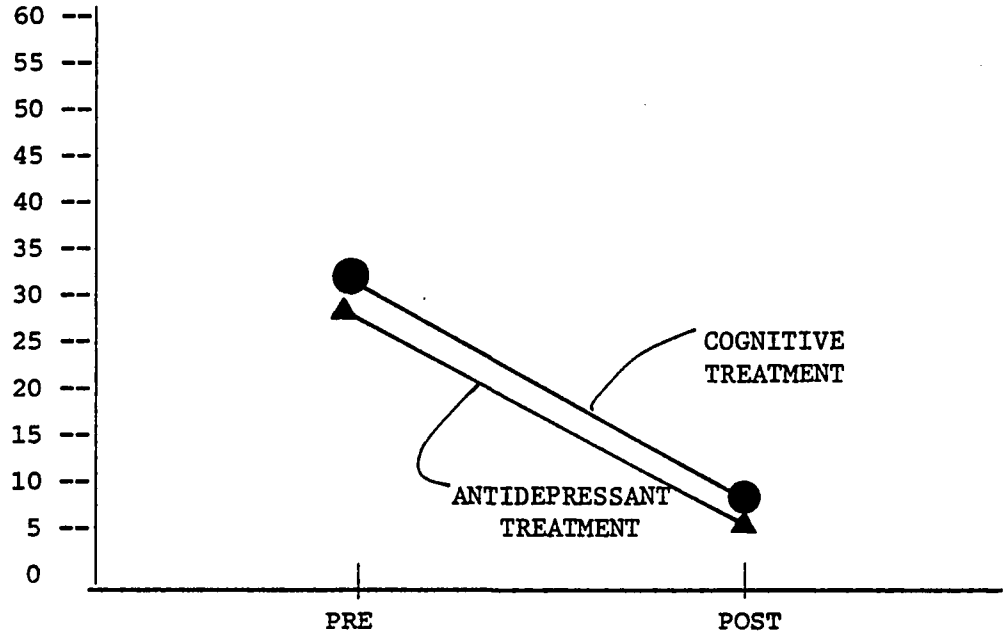


FIGURE 2

BECK DEPRESSION INVENTORY: NORMAL DEXAMETHASONE SUPPRESSION TEST



BECK DEPRESSION INVENTORY: ABNORMAL DEXAMETHASONE SUPPRESSION TEST

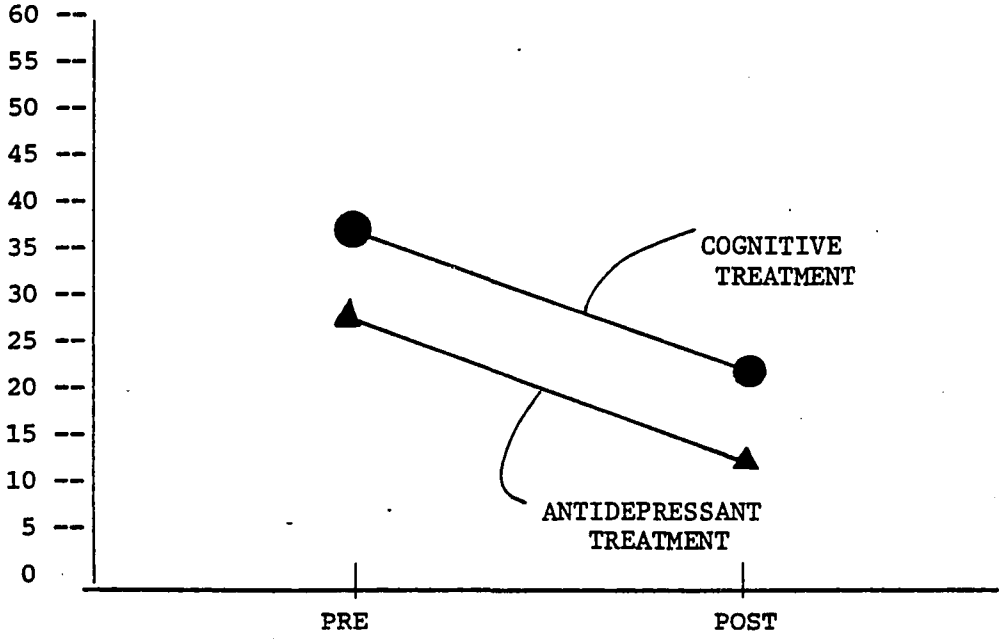
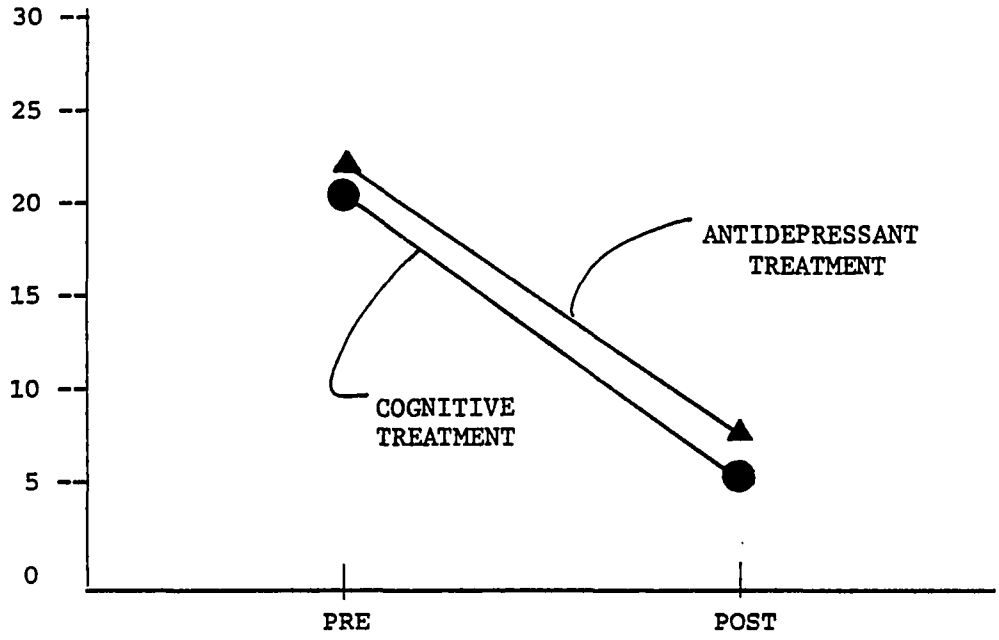


FIGURE 3

LUBIN DEPRESSION ADJECTIVE CHECKLIST:
NORMAL DEXAMETHASONE SUPPRESSION TEST



LUBIN DEPRESSION ADJECTIVE CHECKLIST:
ABNORMAL DEXAMETHASONE SUPPRESSION TEST

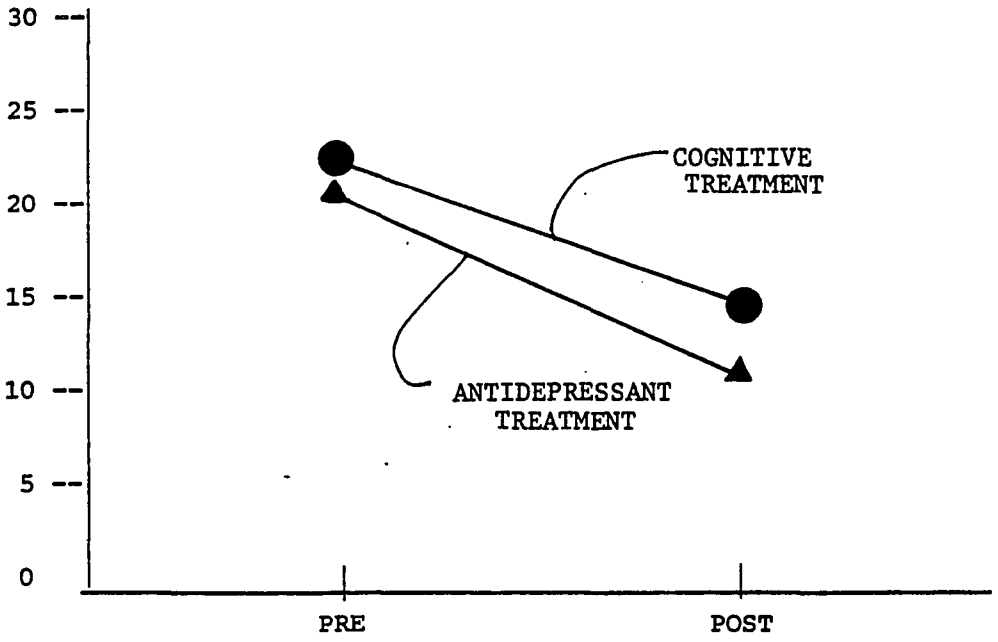
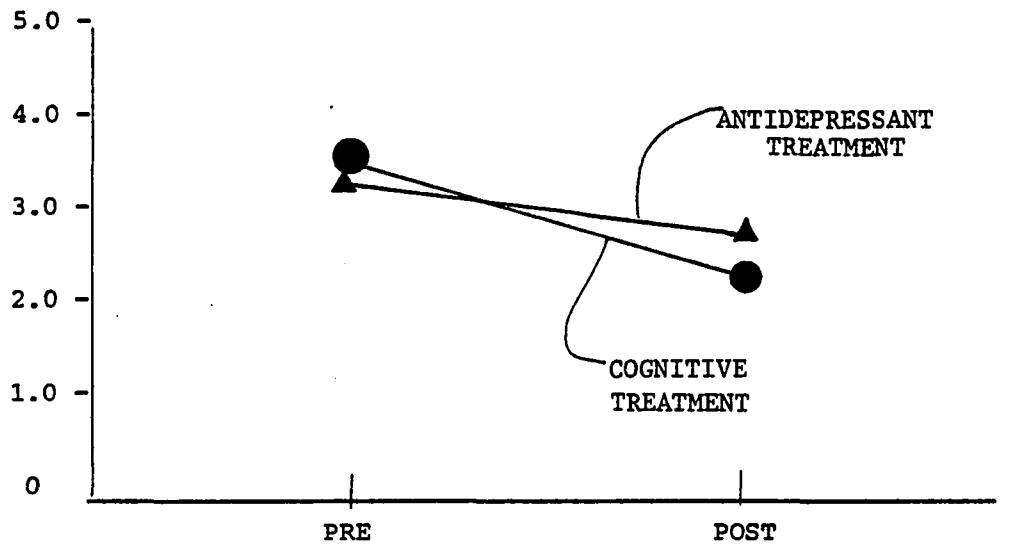


FIGURE 4

PERSONAL BELIEFS INVENTORY: NORMAL DEXAMETHASONE SUPPRESSION TEST



PERSONAL BELIEFS INVENTORY: ABNORMAL DEXAMETHASONE SUPPRESSION TEST

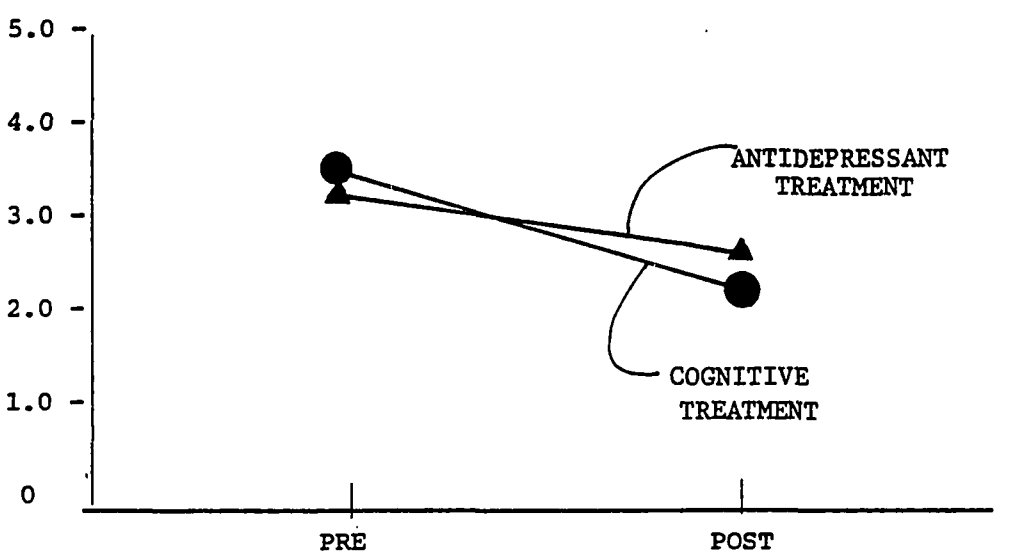
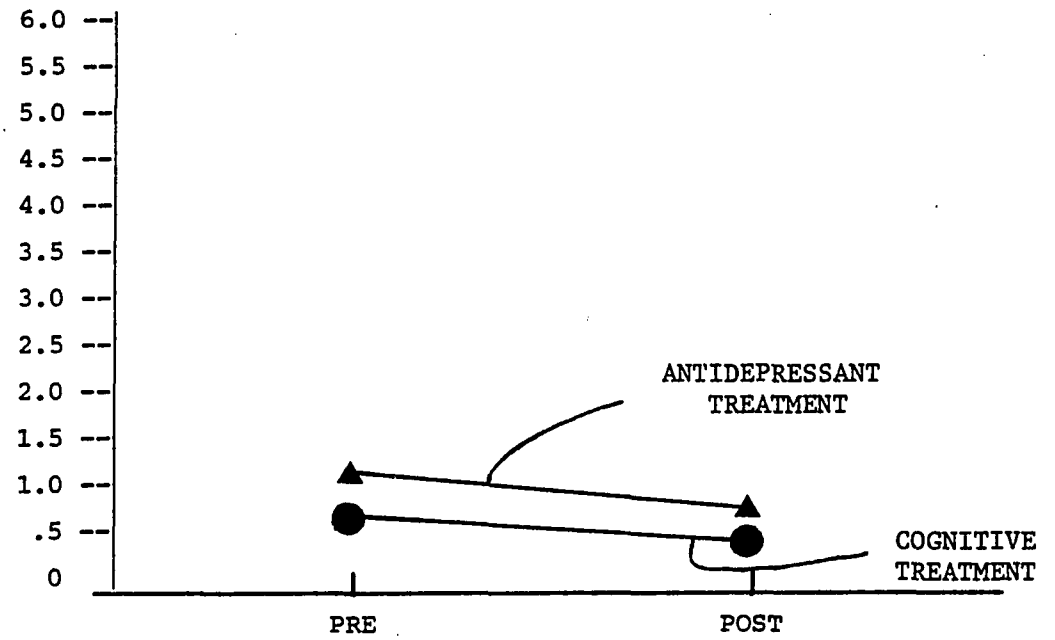


FIGURE 5

NORMAL DEXAMETHASONE SUPPRESSION SUBJECTS:

DEXAMETHASONE SUPPRESSION TEST SCORES



ABNORMAL DEXAMETHASONE SUPPRESSION SUBJECTS:

DEXAMETHASONE SUPPRESSION TEST SCORES

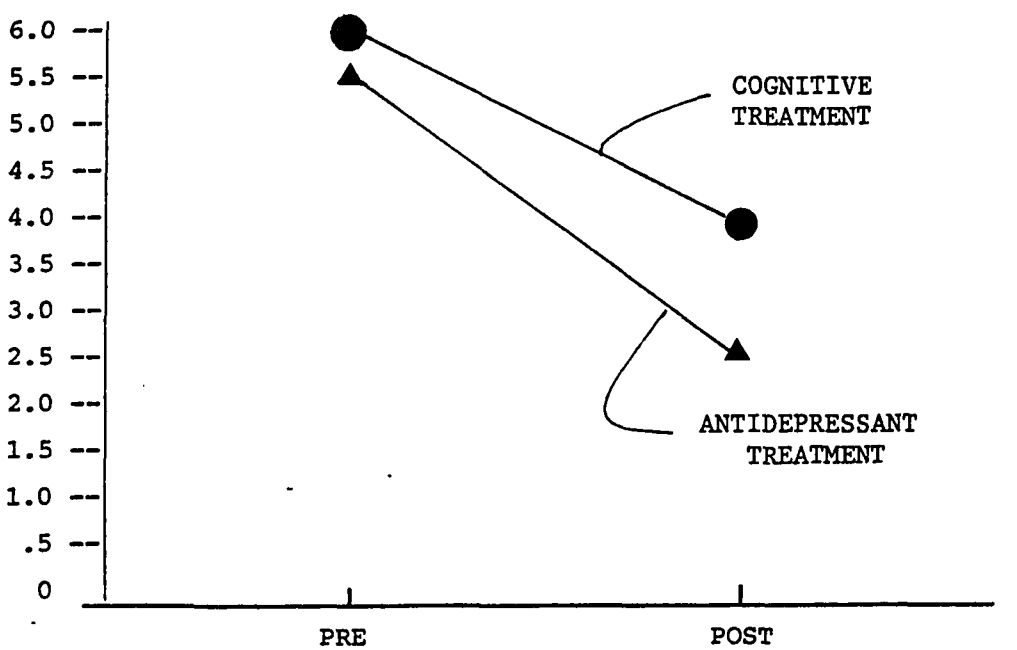
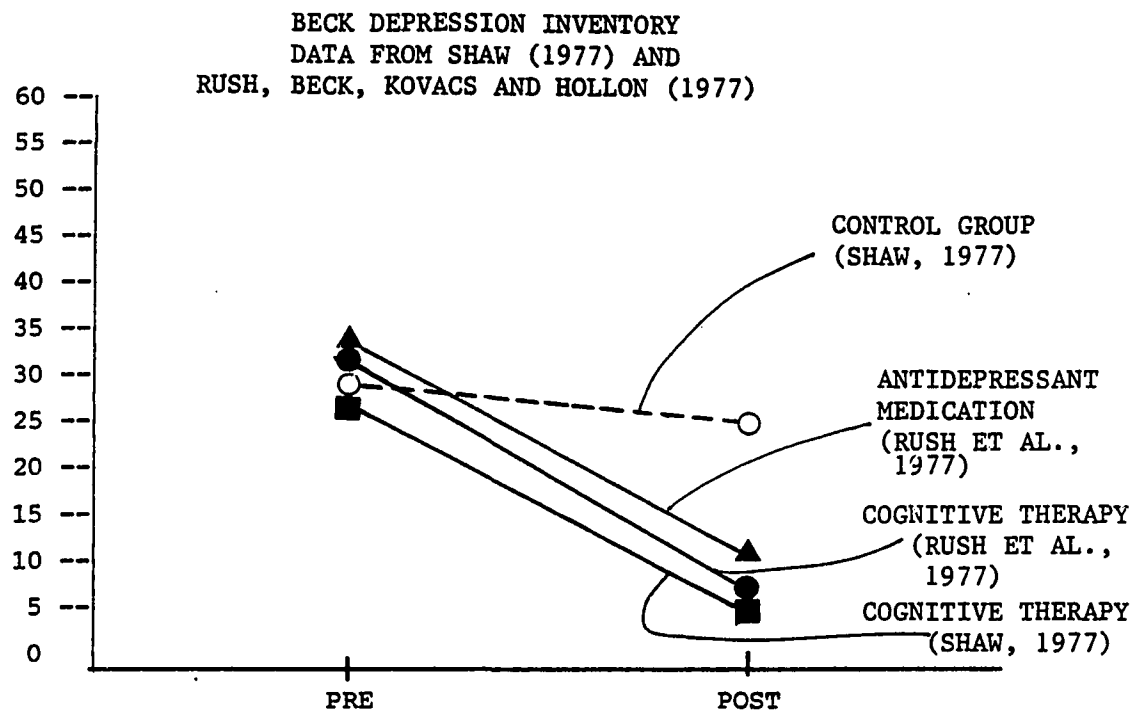
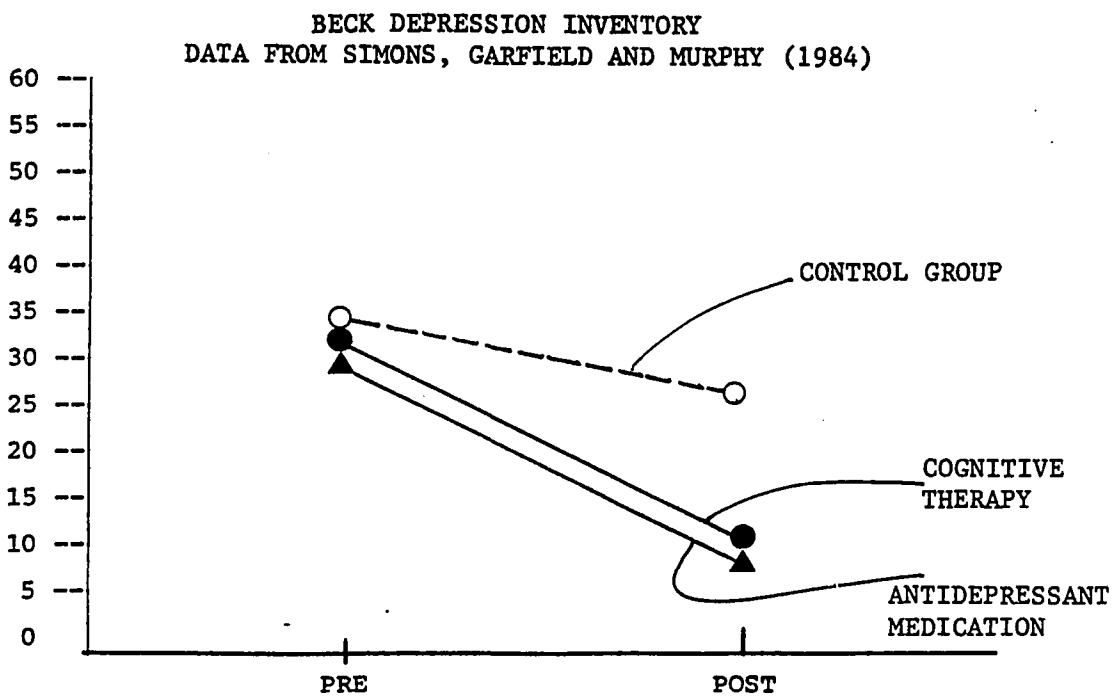


FIGURE 6


BECK DEPRESSION INVENTORY
DATA FROM SHAW (1977) AND
RUSH, BECK, KOVACS AND HOLLON (1977)
60
55
50
45
40
35
30
25
20
15
10
5
0
CONTROL GROUP
(SHAW, 1977)
ANTIDEPRESSANT
MEDICATION
(RUSH ET AL.,
1977)
COGNITIVE THERAPY
(RUSH ET AL.,
1977)
COGNITIVE THERAPY
(SHAW, 1977)
PRE
POST


BECK DEPRESSION INVENTORY
DATA FROM SIMONS, GARFIELD AND MURPHY (1984)
60
55
50
45
40
35
30
25
20
15
10
5
0
CONTROL GROUP
COGNITIVE
THERAPY
ANTIDEPRESSANT
MEDICATION
PRE
POST