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Teresa M. Edenfield

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**EXERCISE AND MOOD: EXPLORING THE ROLE OF EXERCISE IN
REGULATING STRESS REACTIVITY IN BIPOLAR DISORDER**

By

Teresa M. Edenfield

B.S. Florida State University, 2001

M.A. University of Maine, 2004

A THESIS

Submitted in Partial Fulfillment of the

Requirements for the Degree of

Doctor of Philosophy

(in Psychology)

The Graduate School

The University of Maine

August, 2007

Advisory Committee:

Sandra T. Sigmon, Professor of Psychology, Advisor

Jeffrey E. Hecker, Professor of Psychology

Geoffrey L. Thorpe, Professor of Psychology

Larry Smith, Associate Professor of Psychology

Nina Boulard, Adjunct Faculty, Department of Psychology

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EXERCISE AND MOOD: EXPLORING THE ROLE OF EXERCISE IN REGULATING STRESS REACTIVITY IN BIPOLAR DISORDER

By Teresa M. Edenfield

Thesis Advisor: Dr. Sandra T. Sigmon

An Abstract of the Thesis Presented
in Partial Fulfillment of the Requirements for the
Degree of Doctor of Philosophy
(in Psychology)
August, 2007

Bipolar Disorder (BD) is a recurrent and debilitating psychological disorder characterized by a chronic dysregulation of mood with fluctuations between extremely low (e.g., depression) and extremely elevated mood states (e.g., mania), and ranks as the 6th leading cause of disability in the world. Although research has consistently shown that exercise may have antidepressant and stress-attenuating benefits in other psychiatric illnesses (e.g., depression, anxiety), these benefits have not been directly investigated for BD. The current study represents the first known investigation to examine this relationship. Single-participant designs, with crossover and interaction treatment components (i.e., A/B/A/B/A, A/C/A/C/A, A/B/A/C/A, or A/C/A/B/A) were utilized to investigate the impact of participation in a prescribed regimen of exercise (EP) versus standard behavioral activation (SBA; i.e., non-exercise activity) has on stress perception and reactivity, and mood stability in a sample of individuals with BD. Individuals completed four total weeks of treatment, and psychophysiological measures of reactivity

were recorded during a laboratory stress task (i.e., backward counting task) prior to and following each two-week intervention phase.

No appreciable differences were found between levels of exercise participation between treatment groups. Interestingly, symptoms of depressed mood (BDI-II scores) decreased at similar rates following 4 weeks of treatment for all participants. BDI-II decreases were found to be most correlated with elective exercise participation, although this relationship was not significant. Regarding stress reactivity, *elective* participation in mild to moderate intensity exercise was found to reduce an individual's *perception* of stress reactivity to an acute stressor, while participation in a prescribed program of exercise was more effective in reducing physiological response to the same task. Utilizing multiple forms of behavioral activation simultaneously was found to be most effective in decreasing perception of stress reactivity, and may also result in a positive change in the use of adaptive versus maladaptive coping strategies. Participation in a 4-week program of exercise appeared to provide the most benefit, consistent with exercise habituation theories. Overall, current findings provide preliminary support for the prophylactic benefits of including a prescribed and monitored program of exercise as an adjunct treatment for individuals with BD. Larger scale research is needed to more clearly determine the impact of exercise on stress reactivity and mood episode relapse in individuals with BD.

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CHAPTER 1

INTRODUCTION

Bipolar Disorder (BD) is a recurrent and debilitating psychological disorder characterized by a chronic dysregulation of mood, with fluctuations between extremely low mood states (e.g., Major Depressive Episode; MDE) and extremely elevated mood states (e.g., Manic Episode; ME). Elevated mood is generally associated with behavioral activation (e.g., increased goal-directed activity, decreased need for sleep) and altered cognitive functioning (e.g., grandiose delusions, racing thoughts, distractibility), for one week or more (APA, 2000). These mood fluctuations often lead to impulsive, risky behaviors (e.g., substance abuse, sexual indiscretion, excessive spending), and interpersonal difficulties.

BD is considered to be one of the most serious disorders due to its wide-ranging impact. For example, individuals with a diagnosis of BD experience increased mortality rates from suicide, homicide, accidents, and natural causes (e.g., heart disease; Brickman, LoPiccolo, & Johnson, 2002; Murray & Lopez, 1996). Suicidal ideation is a common feature of the depressive spectrum of the disorder with completed suicide occurring in an estimated 10 – 15% of individuals with Bipolar I Disorder (BD-I; APA, 2000). BD is also noted as the third leading cause of occupational disability among medical and psychiatric disorders (Brickman et al., 2002), and the sixth leading cause of disability in the world (Murray & Lopez, 1996). Due to the considerable impact of BD, continual research on the etiology and course of BD is needed to increase our understanding of the disorder. This type of research may have implications for developing alternative interventions for BD.

Although research has identified BD as a leading cause of disability, psychological research has traditionally focused on other disorders (e.g., schizophrenia, unipolar major depression; Quraishi & Frangou, 2002). In general, investigations of BD have concentrated on biological mechanisms (e.g., endocrine system dysregulation) and pharmacologic treatment (e.g., mood stabilizers, anticonvulsants, antidepressants). BD has been widely conceptualized as a biological disorder, primarily (or maybe exclusively) treatable by pharmacological means (e.g., Miklowitz, 2001). Given the apparent biological correlates of the disorder (i.e., 72% concordance rate for monozygotic twins; Rehm, Wagner, & Ivens-Tyndal, 2001); the focus on these treatment modalities appears warranted. However, adjunct forms of intervention (e.g., psychotherapy, exercise) that may target important aspects of the disorder (e.g., medication adherence, stress adaptation responses) and reduce risk of relapse may also prove beneficial.

Previous research has found that regular exercise (i.e., regular, structured, leisure-time pursuit) or other physical activity (e.g., during domestic or occupational tasks) may alleviate depressed mood in individuals experiencing unipolar depression (e.g., Gauvin, Rejeski, & Norris, 1996; Martinsen, 1987; 1990; Steptoe, Kimbell, & Basford, 1998; Yeung, 1996). Although results are generally positive for individuals experiencing unipolar depression, the impact that exercise may have on mood fluctuation and stress adaptation responses in BD has not been investigated. Given consistent evidence that stressful life events (e.g., disruption of daily routines and “social rhythms”) contribute substantially to the onset of bipolar mood symptoms (e.g., Ehlers, Kupfer, Frank, & Monk, 1993; Miklowitz & Goldstein, 1997), this lack of attention is surprising.

Researchers have hypothesized that regular exercise may provide a more structured life system for individuals, thereby reducing vulnerability to stress (Cramer, Nieman, & Lee, 1991; Crews & Landers, 1987; Salmon, 2001) and risk of relapse. Regular exercise has been consistently shown to reduce anxiety, depression, and stress for individuals of all ages and both genders (e.g., Byrne & Byrne, 1993; Folkins & Sime, 1981; Gauvin & Spence, 1995; Plante & Rodin, 1981; Plante & Rodin, 1990). In addition, exercise has been shown to increase life structure and decrease stress reactivity (Salmon, 2001) in individuals meeting criteria for affective disorders (e.g., depression), as well as healthy controls. Regular exercise has also been associated with lower perceived stress in daily living (Steptoe, Lipsey, & Wardle, 1998). However, it remains to be seen if regular exercise may buffer the effects of stress in individuals with BD. Regular exercise may also provide a means of coping with stressors that have not yet occurred by providing increased structure and habitual routine in the life of an individual with BD.

According to Ehlers and colleagues (1988), both depressive and manic episodes may be triggered by dysregulation in routine for individuals with BD. The possibility that exercise may enhance routine regulation is also consistent with current conceptualizations of behavioral activation (BA) interventions. BA interventions focus on assisting individuals with the establishment and maintenance of a regular routine. Also consistent with routine regulation hypotheses, repeated exposure to uncontrollable (unexpected) stressors is thought to result in the manifestation of a resistance to stress (i.e., habituation to the stressor; Salmon, 2001). Therefore, repeated experiences with controllable (e.g., planned, expected) stressors (e.g., exercise; Salmon, 2001) may assist

individuals in achieving this level of stress adaptation more quickly (Maier & Seligman, 1976; Weiss & Glazer, 1975).

Several hypotheses have been offered to account for exercise's effect on mental health (e.g., decrease in negative mood) that involve biochemical, physiological, and psychosocial mechanisms. Specific mechanisms and the conditions under which they operate, however, have not yet been determined (Camacho, Roberta, Lazarus, Kaplan & Cohen, 1991; Fox, 1999). Overall, the physical and mental health benefits of exercise are widely recognized (American College of Sports Medicine, 1990; Dubbert, 2002; Pate et al., 1995; U.S. Department of Health and Human Services, 1996), and could potentially benefit individuals with BD. In addition, the high level of non-compliance for traditional forms of treatment for BD (e.g., medication) demonstrates an increased need for expanding intervention approaches to include adjunctive treatment modalities that may be considered effective, individually acceptable, and widely accessible (e.g., Salmon, 2001) to a variety of affected individuals (e.g., both inpatient and outpatient populations).

Psychopharmacological intervention (e.g., mood stabilizing medications) remains the gold standard treatment for BD. Consistent with this mainstream approach, empirically supported psychotherapeutic interventions for BD have been devised (e.g., Family Focused Therapy, Interpersonal and Social Rhythm Therapy, Cognitive Behavioral Therapy). In these approaches, BD is conceptualized as a primarily biological condition in which pharmacological intervention is warranted to stabilize mood before any other behavior is targeted for treatment. Nonetheless, an integrated treatment approach pairing these supported treatments with regular physical exercise may offer a more comprehensive and cost-effective intervention, including the provision of coping

strategies available for implementation to a wider range of individuals with BD. An integrated treatment approach to BD, including a regular physical exercise regimen, may enhance relapse prevention efforts by assisting individuals to develop more structured daily routines, thereby decreasing stress reactivity and vulnerability for relapse.

Incorporating exercise into existing models of treatment for Bipolar Disorder may promote a better understanding of the role of perceived stress, stress reactivity, and structured daily routine in BD. Empirical investigation of the role of exercise in the treatment of BD using multiple assessment methods is needed to determine whether exercise may represent a useful adjunct intervention in regulating mood. This review will examine current research criteria and features of BD, as well as current etiological and maintenance models of the disorder. The theoretical rationale underlying each model will be reviewed as well as evidence that such models may represent accurate accounts of the onset and maintenance of mood episodes. Because the extant literature primarily examines the role of exercise in alleviating depressed mood, the following sections will explore whether these models can be applied to the conceptualization of relapse into either spectrum of BD (e.g., depressed or manic phase).

It was hypothesized that mood episode relapse in individuals with BD would be maintained and exacerbated by higher levels of reactivity to perceived daily stress and decreased structure in daily routine. The current experimental investigation utilized psychophysiological measurement of participants' stress reactivity (i.e., skin conductance levels) during an evoked stress response task prior to and immediately following two forms of intervention (i.e., standard BA versus BA with exercise prescription as the focus). Participants monitored their exercise levels (via pedometer and self-report),

perceived stress levels, and mood on a daily basis. In addition, individuals monitored the onset of mood symptoms that indicate full relapse or subsyndromal symptom expression of either manic or depressive symptoms on a weekly basis.

Description, Diagnostic Criteria, and Characteristics of Bipolar Disorder

Bipolar Disorder (BD) is one of the oldest and most recognized psychiatric disorders, affecting 1 – 3% of the population (APA, 2000; Kessler et al., 1994).

Although more advanced than in previous times, current conceptualizations of the disorder continue to bear remarkable similarity to Kraepelin's (1921) original description of "manic-depressive insanity" (Rehm, Wagner, & Ivens-Tyndal, 2001). The central characteristic of Bipolar Disorder is extreme affective dysregulation. Specifically, individuals' mood may fluctuate from extremely low mood states (e.g., MDE) to extremely elevated mood states (e.g., manic episode) characterized by euphoria or irritability. Elevated mood is characterized by behavioral activation (e.g., increased goal-directed activity, excessive involvement in high-risk activities, decreased need for sleep, pressured speech) and altered cognitive functioning (e.g., grandiose delusions, inflated self-worth, racing thoughts, distractibility; APA, 2000). In addition, these fluctuations are associated with psychosocial impairment, as well as changes in energy, social behavior, cognitive functioning, attention, and impulse control.

For individuals with BD, the pattern of affective dysregulation and associated psychosocial disruption often results in a variety of adverse consequences (e.g., divorce, extreme debt from excessive spending, abusive/violent behavior that may result in legal consequences). In addition, individuals suffering from early-onset BD are more likely to report a history of alcohol or substance use problems (APA, 2000). As noted earlier,

individuals diagnosed with BD experience higher mortality rates from suicide, homicide, accidents, and natural causes (e.g., cardiovascular disease) than individuals who do not meet criteria for a BD diagnosis (Brickman, LoPiccolo, & Johnson, 2002; Murray & Lopez, 1996). Suicidal ideation is a common feature of the depressive spectrum of the disorder, with completed suicide occurring in an estimated 10 - 15% of individuals with Bipolar I Disorder (APA, 2000). Overall, individuals with BD appear to be at increased risk of injury and death secondary to both natural and self-inflicted causes than the general population (Brickman et al., 2002). In addition to increased rates of mortality, individuals with BD often experience impairments in social and occupational functioning. These impairments may last as long as five years following the primary mood episode and result in additional stressful situations (e.g., marital discord, delays in returning to work, below-average performance after returning to work; Goldberg, Harrow, & Grossman, 1995; Tohen, Waternaux, & Tsuang, 1990).

High relapse rates and breakthrough episodes, related to medication non-adherence, are also characteristic of BD. Specifically, the survival rate (i.e., absence of relapse) at a 2-year follow-up with traditional lithium carbonate treatment is estimated at 40% (Gelenberg et al., 1989), declining further to 27% survival at 5-year follow-up (Gitlin, Swendsen, Heller, & Hammen, 1995). Researchers have estimated that only 30% of individuals with BD consume prescribed mood stabilizing medications on a routinely scheduled basis, particularly if the most recently experienced mood fluctuation was a manic episode (Miklowitz, Goldstein, Nuechterlein, Snyder, & Mintz, 1988). Regardless of adherence to a medication regimen, the previously mentioned impairments are often

associated with BD. More research is needed to develop treatment protocols that may be helpful in reducing relapse rates and increasing treatment adherence.

Epidemiology of Bipolar Disorder

Prevalence of Bipolar Disorder

Researchers have questioned the 1 – 3 % prevalence estimate, given the prevalence of “softer” bipolar spectrum disorders (i.e., incorporating BD-II, BD-NOS, recurrent brief hypomania, sporadic brief hypomania, cyclothymia, antidepressant induced mania, and depression with bipolar family history; Akiskal et al., 1998; Frye, Gitlin, & Altshuler, 2004). Including the full spectrum of bipolar disorders, lifetime prevalence rates have been estimated as high as 6.5% in adults (Angst, 1998) and 13.3% in adolescents (Carlson & Kashani, 1988; Lewinsohn, Klein, & Seeley, 1995). However, adolescent reports of bipolar spectrum symptoms are not always corroborated by parental reports of symptom expression (Lewinsohn et al., 1995).

In a recent re-analysis of the Epidemiological Catchment Area Study (ECA; Judd & Akiskal, 2003), researchers noted an increased prevalence rate (5.1%) of subthreshold mania based on DSM-III criteria used in the original study. Using the Mood Disorders Questionnaire (Hirschfeld et al., 2000), Hirschfeld and colleagues (2003) reported that 3.7% of individuals ($N = 127,800$ adults representative of population in the United States) screened positive for BD-I and BD-II disorder. The discrepancy that exists in earlier and later prevalence estimates, however, may be due to recent guideline changes regarding the formulation of a Bipolar II diagnosis (i.e., DSM-IV-TR; APA, 2000). Specifically, the DSM diagnostic criteria currently state that a hypomanic formulation be considered only when symptoms persist for four or more days. However, research has shown that

hypomanic symptoms of shorter durations (e.g., 1 – 3 days) are frequently recognized as a valid subtype of the disorder (Angst, 1998).

Course of Bipolar Disorder

Bipolar Disorder appears to affect a wide variety of individuals at equivalent rates. Average age of onset for the disorder is approximately 20 years of age, for both males and females (DSM-IV-TR; APA, 2000). Even when treated with maintenance psychopharmacology (Gitlin et al., 1995), an estimated 70 – 80% of individuals with a diagnosis of BD will experience at least one recurrent episode within 5 years of the index episode (Gitlin et al., 1995; Miller, Uebelacker, Keitner, Ryan, & Solomon, 2004; Tohen et al., 1990). In addition, an estimated half of all individuals initially hospitalized with BD will be re-hospitalized within 5 years (Goldberg, Harrow, & Grossman, 1995). Impairment resulting from the experience of BD appears to be relatively consistent across individuals who develop the disorder, with no clear evidence that severity differs according to demographic characteristics.

Approximately 60 – 70% of individuals who experience a Manic Episode (ME) do so immediately preceding or following the experience of a MDE (APA, 2000). Interestingly, there have been no reported differences between depressive episodes that occur in individuals meeting criteria for Major Depressive Disorder (MDD) compared to depressive episodes that occur in BD. However, the number of lifetime episodes of mood dysregulation, regardless of polarity, is reported to be higher in individuals with Type I Bipolar Disorder (BD-I) versus recurrent MDD (APA, 2000). In particular, in the absence of treatment (e.g., lithium), it is estimated that individuals will experience an

average of four episodes of mood disturbance over the course of a 10-year time period (APA, 2000).

Some individuals with BD appear to demonstrate a pattern of cycling over the course of the disorder that may be associated with a poorer prognosis and treatment response. An estimated 13 – 20% of individuals with BD will demonstrate a pattern of rapid cycling (Goodwin & Jamison, 1990; Tondo & Baldessarini, 1998). This pattern of rapid cycling occurs more frequently in females than males (e.g., Akiskal et al., 2000; Bauer et al., 1994; Coryell, Endicott, & Keller, 1992; Shelton & Calabrese, 2000), and is more likely to occur with a first depressive episode (Goodwin & Jamison, 1990; Perugi et al., 2000). To qualify for the rapid cycling specifier, an individual must report the experience of multiple (i.e., at least 4) episodes of mood dysregulation (i.e., MDE, ME, Mixed Episode, or Hypomanic Episode) during the course of a single year. The pattern of rapid cycling has been linked to a greater number of illness episodes and hospitalizations (Avasthi, Sharma, Malhotra, Gupta, & Kulhara, 1999; Bauer et al., 1994) and a longer mean duration of illness (Bauer et al., 1994; Maj, Magliana, Pirozzi, Marasco, & Guarneri, 1994). Associated with poorer prognosis (APA, 2000), this pattern of cycling is relatively uncommon.

Culture, Age, and Gender Considerations in Bipolar Disorder

There appears to be little evidence for gender differences in BD prevalence rates (Akiskal et al., 2000; Gitlin et al., 1995). However, a gender difference has been reported regarding the phase of the first mood episode. Females appear to be more likely to experience a first episode of major depression than males, whereas males may be more likely to have the initial experience of a ME (APA, 2000; Goodwin & Jamison, 1990;

Perugi et al., 2000). In addition, females are more likely to experience a greater number of lifetime MDE, whereas males are more likely to experience a greater number of lifetime MEs than females (Akiskal et al., 2000; APA, 2000; Bauer et al., 1994; Gitlin et al., 1995). Regardless of these gender differences, individuals meeting criteria for BD tend to report that affective symptoms tend to abate between mood episodes. However, a small percentage of affected individuals (i.e., 20 – 30%) experience residual symptoms between episodes (Akiskal et al., 2000; APA, 2000; Gitlin et al., 1995), and the length of time between episodes appears to increase with age (Akiskal et al., 2000; APA, 2000).

Additional considerations regarding the diagnosis of BD include cultural and age factors. Regarding cultural factors, there is no empirical support for cultural (i.e., based on race or ethnicity) differences in the prevalence of BD (Boyd & Weissman, 1981; Kessler et al., 1994; Robins & Reiger, 1991). However, in some ethnic groups (e.g., African Americans) and special populations (e.g., children and adolescents), there may be a tendency to over-diagnose Schizophrenia versus BD (e.g., Akiskal et al., 2000; Faraone, Biederman, Mennin, Wozniak, & Spencer, 1997; Kafantaris, Coletti, Dicker, Padula, & Pollack, 1998). This tendency toward misdiagnosis may reflect differences in cultural standards of appearance, hygiene, and fashion (e.g., hair styles). The tendency to diagnose Schizophrenia instead of BD in younger individuals may offer support for reports that psychotic symptoms are more likely to be present in individuals exhibiting symptoms at an early age of onset (e.g., Faraone et al, 1997; Kafantaris et al., 1998; Muller-Oerlinghausen, Berghofer, & Bauer, 2002).

Researchers continue to debate the appropriateness of the BD diagnosis within childhood and adolescent populations (e.g., Cassano, McElroy, Brady, Nolen, & Placidi,

2000; Geller & Luby, 1997; Geller et al., 2002; Kim & Miklowitz, 2002). Given the lack of age-appropriate diagnostic guidelines for BD, it is more difficult to make an accurate diagnosis in younger populations than for individuals exhibiting symptoms at a more common age of onset (i.e., age 18 – 24). In addition, it is important to recognize the overlap in symptoms between the typical early-onset presentation of BD and other disorders. For example, disruptive behaviors are characteristic of several disorders in adolescence (e.g., ADHD, Oppositional Defiant Disorder, Conduct Disorder), further complicating the diagnostic picture (Biederman et al., 1998; Carlson & Kelly, 1998; Faraone et al., 1997; Geller et al., 1998).

Bipolar I Disorder (BD-I) compared to Bipolar II Disorder (BD-II)

Bipolar I Disorder (BD-I) is differentiated from other categories of Bipolar Disorders (e.g. Bipolar II Disorder, Cyclothymia) based on the occurrence of one or more Manic or Mixed Episodes, not due to the direct effects of a medication, other treatments for depression, drug abuse, toxin exposure, or because of a General Medical Condition. To meet criteria for a BD-I diagnosis, an individual must have experienced a Manic Episode that cannot be better accounted for by another major Axis I disorder (i.e., Schizoaffective, Schizophrenia, Schizophreniform, Delusional, or Psychotic Disorder NOS). In addition, manic symptoms must persist for seven or more days, and cause clinically significant impairment for the individual experiencing the dysregulation of mood (APA, 2000). Frequently, individuals meeting criteria for a BD-I diagnosis will have experienced one or more Major Depressive Episodes, although this is not essential in making a BD-I diagnosis. The criteria for Manic, Mixed, and Depressive Episodes

will be outlined in upcoming sections following the description of the four major categories of Bipolar Disorders.

Bipolar II Disorder (BD-II), or Recurrent MDE with Hypomanic Episodes (APA, 2000) can be differentiated from BD-I based on the persistence and severity of symptoms consistent with the elevated mood characteristic of BD (i.e., must meet criteria for one or more Hypomanic Episodes), and the required occurrence of one or more MDE.

Hypomania is defined by abnormally and persistently elevated, expansive, or irritable mood that lasts at least four days (APA, 2000). Generally, individuals experiencing hypomanic symptoms will demonstrate enthusiasm for social, interpersonal, or occupational interactions. According to the DSM-IV-TR (APA, 2000), symptoms of hypomania represent expansive mood that is noticeably different from an individual's mood presentation in the absence of any mood episode. Therefore, symptoms of hypomania should be carefully discriminated from euthymic periods following a depressive state in order to accurately determine whether diagnostic criteria are met.

Hypomania can typically be differentiated from a Manic Episode based on symptom severity. Specifically, hypomanic symptoms do not cause clinically significant impairment (e.g., need for hospitalization) and can be generally creative and productive periods (APA, 2000). However, an accurate diagnosis of BD-II emphasizes that the period of increased goal-directed activity characteristic of Hypomanic Episodes is observably different from the individual's normative state. Therefore, such an increase in goal-directedness may place the individual at risk for adverse consequences subsequent to impulsive behaviors (e.g., hypersexuality, reckless driving, risky financial

investments). Broadly, Hypomanic Episodes have been described as organized, less bizarre, and less debilitating than symptom expression characteristic of Manic Episodes.

Comorbidity

Comorbidity is defined as the current or lifetime experience of two or more psychiatric or medical conditions that coexist with the psychiatric disorder of primary interest (APA, 1994). Individuals meeting diagnostic criteria for a Bipolar Disorder diagnosis often present a complex picture of multiaxial comorbidity. According to McElroy and colleagues (2001), a BD diagnosis may be twice as likely to occur comorbidly with another Axis I psychiatric disorder as alone. This complex presentation may interfere with appropriate diagnosis, and ultimately may demonstrate a negative influence on acute treatment response and course of illness (McIntyre, Konarski, & Yatham, 2004).

Although the primary symptom in Bipolar Disorder is mood fluctuation, most individuals exhibiting BD symptoms are affected by comorbid conditions that may warrant simultaneous treatment (e.g., anxiety disorders, substance dependence, eating disorders). In the Epidemiologic Catchment Area study (ECA; Regier et al., 1984), researchers found substantial rates of comorbidity for bipolar disorder as defined by the DSM-III (APA, 1980). Specifically, individuals who met diagnostic criteria for a Bipolar Disorder also met DSM –III criteria for lifetime alcohol abuse (15%) or dependence (31%); a total of 41% endorsed a pattern of other substance abuse (13%) or dependence (28%). Overall, 61% of individuals included in the ECA study met criteria for substance abuse or dependence. Anxiety, eating, and personality disorders also commonly occur with BD. According to Miklowitz and colleagues (2001), the disorders that commonly

co-occur with Bipolar Disorder demonstrate an underlying causal mechanism of affective dysregulation, the primary feature of BD. Given the high rates of comorbidity for BD, the recognition of the multidimensional nature of BD may influence the formulation of a more inclusive therapeutic model. This type of comprehensive conceptualization may assist care providers in formulating more concise, realistic, and beneficial therapeutic objectives. Common comorbidities observed in individuals with BD will be reviewed below.

Substance Use/Abuse Disorders. In the past 20 years, more attention has been given to the overrepresentation of substance use disorders in individuals with psychiatric disorders (i.e., dual-diagnosis patients; Grant, 1995; Grant & Harford, 1995; Kessler et al., 1996; Regier et al., 1990; Robins & Regier, 1991). Results from the ECA study (Regier et al., 1984) indicated that individuals with BD-I (compared to other Axis I conditions) had the highest lifetime rates of alcohol use disorders (i.e., 46%) and drug use disorders (i.e., 41%). Risk of having any substance use disorder was 61% for BD-I, and 48% for BD-II. In a more recent analysis, the National Comorbidity Survey (NCS; Kessler et al, 1996) revealed that individuals with mania were 8.2 times more likely to demonstrate drug dependence in the past year, and 8.4 times more likely to have lifetime drug dependence, compared to the general population. Overall, individuals with BD demonstrate rates of drug abuse or dependence of 14 – 65% (Brown, Suppes, Adinoff, & Thomas, 2001) versus 6 – 12% of individuals in the general population (Kessler et al., 1996; Robins & Regier, 1991).

Although there is notable difficulty regarding the diagnosis of BD in individuals with substance abuse disorders, diagnostic confusion is less likely to occur for individuals

with chronic, severe BD or for those who demonstrate a clear development of BD symptoms prior to substance use (Levin & Hennessy, 2004). Individuals with an earlier onset of BD illness are thought to be more likely to exhibit current substance and alcohol use and/or abuse problems (APA, 2000). Consistent with the NCS results just described, approximately 21 – 61% of individuals with BD are estimated to struggle with alcohol or other substance abuse difficulties (APA, 2000). This rate of occurrence is substantially higher than that seen in the general population (i.e., 3 – 13%; Kessler et al., 1996; Regier et al., 1984). Substances used commonly by individuals with BD include stimulants and alcohol. Current opinions regarding stimulant use emphasize the enhancement of euphoric feelings that occur secondary to consumption (e.g., cocaine). In general, the concomitant use or abuse of alcohol or other substances may increase the number and length of hospitalizations, and generally result in poorer outcome (e.g., Cassidy, Ahearn, & Carroll, 2001; Goldberg, Garo, Leon, Kocsis, & Portera, 1999; Sonne, Brady, & Morton, 1994).

Panic Disorder. A consistent relationship of comorbidity has been established between Bipolar Disorders and Panic Disorder (Bowen, South & Hawkes, 1994; Chen & Dilsaver, 1995; Dilsaver et al., 1997; Goodwin & Hoven, 2002; Grunhaus, Pande, Brown, & Greden, 1994; Pini et al., 1997; Savino et al., 1993). Estimated prevalence rates of co-occurring Panic Disorder in adult individuals with BD range from 26 – 80% (Chen & Dilsaver, 1995; Dilsaver et al., 1997; Frank et al., 2002; Pini et al., 1997). Conversely, the rate of a co-occurring BD in those adults with Panic Disorder is estimated to occur substantially less often, at a rate of 13 – 23% (Bowen et al., 1994; Grunhaus et al., 1994; Savino et al., 1993). There is some evidence from family and

genetic investigations to support the hypothesis of a genetic relationship between Panic Disorder and Bipolar Disorder (MacKinnon et al., 2002; Rotondo et al., 2002; Wozniak, Biederman, Monuteaux, Richards, & Faraone, 2002), although mixed findings render these results inconclusive.

Additional Diagnostic Dilemmas

Depressive Episodes may appear remarkably similar in Unipolar Depression and Bipolar Disorder. Because the DSM-IV-TR diagnostic criteria (APA, 2000) require a manic, mixed, or hypomanic episode for the diagnosis of BD, a large number of individuals may be initially misclassified and treated for MDD (Akiskal, 1995; Ghaemi et al., 1999). Misdiagnosis in these cases can be quite detrimental for an individual's prognosis. For example, typical treatments for unipolar depression include antidepressant medications. Some antidepressant medications (e.g., tricyclic antidepressants such as imipramine) may actually increase the likelihood of the development of a manic or hypomanic phase, as well as produce rapid cycling (Bowden, 2001). Typical guidelines for antidepressant treatment suggest that individuals complete a year or more of the medication following the episode onset. Unfortunately, a longer duration of treatment may increase the risk of cycling in individuals who have not been correctly diagnosed (Bowden, 2001). In addition, individuals who have experienced three to eight episodes of BD are less likely to benefit from treatment with standard medications (either during acute phases of illness, or for prophylaxis) such as lithium (Gelenberg et al., 1989; Swann, Bowden, Calabrese, Dilsaver, & Morris, 1999).

Given the potential for inappropriate medical treatment, it is important to identify features of bipolar depression that can reliably distinguish such episodes from the

depressive episodes experienced by individuals with unipolar depression (e.g., Akiskal, 1995; Mitchell & Malhi, 2004). Researchers have found that depressive episodes of bipolar depression are associated with a higher degree of mood lability during the episodes (Akiskal, 1995), more psychomotor retardation (Brockington, Altman, Hillier, Meltzer, & Nand, 1982; Mitchell & Malhi, 2004), hypersomnia (Hartmann, 1968; Kupfer, Himmelhoch, Swartzburg, Anderson, Byck, & Detre, 1972; Detre, Himmelhoch, Swartzburg, Anderson, Byck, & Kupfer, 1972), weight loss (Abrams & Taylor, 1980) and agitation (Beigel & Murphy, 1971; Katz, Robins, Croughan, Secunda, & Swann, 1982) than individuals with unipolar depression. In addition, bipolar depression may account for the high rate of morbidity and mortality that occurs as a result of this illness (Mitchell & Malhi, 2004). Researchers have suggested that even subsyndromal depressive episodes that occur in BD may contribute to the high rate of disability related to this condition (e.g., Murray & Lopez, 1996). Although there is evidence to suggest symptomatic differences between unipolar and bipolar depressive episodes, there are no specific constellations of symptoms that allow for a more accurate diagnosis.

The following section outlines a variety of potential risk factors that have been consistently linked to both initial onset of BD and relapse into successive mood episodes. In addition, the section will review models that consider one or more of these risk factors in explaining the etiology of BD. Although a variety of risk factors have been identified regarding onset and maintenance of BD, there are some limitations. Of particular concern is the common tendency to collapse research participants into a single experimental group of “mood disordered” individuals. Similarly, there is a tendency to collapse all individuals endorsing bipolar characteristics into a single research group, as

opposed to collecting and/or analyzing data based on categorical guidelines (e.g., BD-I, BD-II). The risk factors described below are relevant for the recurrent mood dysregulation in individuals who generally experience polarity shifts in mood, whether diagnostic criteria are met for BD-I, BD-II, Cyclothymia, or BD-NOS.

Risk Factors for Bipolar Disorder

Family History

Although the propensity to develop Bipolar Disorder is generally grounded in a diathesis-stress framework, primary emphasis continues to focus on the importance of genetic and biological factors (Craighead & Miklowitz, 2000; Craighead, Miklowitz, Frank, & Vajk, 2002). Elevated rates of occurrence are reported for individuals with a family history of BD (APA, 2000; DelBello & Geller, 2001). More specifically, first-degree relatives of individuals with BD-I are reported to develop BD-I at a rate of 4 – 24% (APA, 2000), which is markedly higher than standard prevalence estimates. Similarly, these genetically vulnerable individuals go on to develop Type II BD at a rate of 1 – 5% (APA, 2000), and MDD at a rate of 4 – 24% (APA, 2000), still exceeding national averages for the development of such mood disorders. Moreover, age of onset for the disorder is reported to occur at an earlier age than for affected individuals without a family history of BD (Akiskal et al., 1998; APA, 2000; DelBello & Geller, 2001).

Psychosocial Stressors

Although a genetic vulnerability appears to have strong support, the importance of psychosocial stressors also needs to be considered. Research findings have consistently supported the premise that the onset of mood episodes in BD is most frequently preceded by the occurrence of one or more life stressors (e.g., social and family; Basco & Rush,

1995, 1996; Gitlin et al., 1995; Miklowitz, 2001; Miklowitz & Goldstein, 1997). Time periods prior to affective episodes appear to contain a greater number of stressful events when compared to euthymic periods of an individual's life (Miklowitz & Goldstein, 1997). Furthermore, disruption of social rhythms secondary to the occurrence of stressful life events may further increase one's risk for relapse (Miklowitz & Goldstein, 1997). Manic episodes, particularly, are likely to be precipitated by stressful life events that disrupt biological rhythms (e.g., travel across time zones, change in work load). Given the apparent connection between mood episode onset and the experience of life stressors, the role of psychosocial factors in the development and maintenance of BD needs to be further investigated.

Life Stress and Bipolar Disorder

Several social and psychological variables have been directly linked to the development and maintenance of BD (e.g., Craighead & Miklowitz, 2000; Ehlers et al., 1993; Johnson & Meyer, 2004). In particular, major negative life events such as those that involve loss, appear to be reliable predictors of depressed mood episodes and a poorer prognosis for individuals with BD (Ellicott, Hammen, Gitlin, Brown, & Jamison, 1990; Hunt, Bruce-Jones, & Silverstone, 1992; Johnson & Roberts, 1995; Kessing, Agerbo, & Mortensen, 2004). Negative life events are thought to be associated with alterations in daily life patterning, as well as changes in biological and social rhythms (Ehlers et al., 1988, 1993). For example, Frank and colleagues (1994, 2000) propose that life events may precipitate relapse by inducing disruptions in social (and biological) rhythms that are directly linked to mood episode relapse. In samples of individuals with bipolar disorder, Malkoff-Schwartz and colleagues (1988, 2000) found that major life

events result in disruptions in social routines, and are significantly associated with relapse into the manic phase of illness.

In a recent pilot study, the role of exercise in reducing stress reactivity in individuals with BD and healthy controls was examined (Edenfield, Gallant, & Sigmon, 2004). In this study, all individuals recording the highest level of exercise activity were significantly less likely to exhibit significant increases in skin conductance during the experimental stress tasks. These findings are consistent with current theories (i.e., Salmon's Stress Adaptation Theory) hypothesizing that regular exercise may effect antidepressant changes by way of improving stress adaptation responses (e.g., Salmon, 2001). In addition, results from this preliminary investigation revealed that skin conductance reactivity during the initial stress task was a significant predictor ($p < .05$) of *perceived* stress during the self-monitoring period. Specifically, reactivity to the first laboratory stress task was a significant predictor of perceived stress during the 14-day self-monitoring period. This finding is consistent with the conclusion that all participants reported a level of stress in their daily lives consistent with reactivity recorded during a stressful task. These findings are of particular interest, given previous indications that exercise may reduce the perception of stress in daily living (Steptoe et al., 1998). Further research is needed, however, to investigate the impact of increased exercise levels on stress perception and reactivity in individuals with BD.

Life Structure and Episode Relapse/Recurrence in Bipolar Disorder

Given the established relationship between life stress and relapse in BD, psychological interventions have begun to focus on routine regulation as a major component of therapy in individuals diagnosed with BD. However, individuals with

Bipolar Disorder typically experience frequent relapses and chronic subsyndromal symptoms, even when receiving validated forms of treatment (Judd et al., 2002; Miklowitz & Goldstein, 1997; Prien & Rush, 1996). Psychosocial interventions for BD often include a component focused on improving routine regulation and life structure. Some researchers hypothesize that routine regulation may increase access to available coping strategies for these individuals through various mechanisms, such as decreasing daily stress and allowing for more flexible problem-solving (e.g., Miklowitz & Goldstein, 1997; Johnson & Meyer, 2004).

Some researchers (e.g., Frank, 1999; Malkoff-Schwartz et al., 1998; Swartz & Frank, 2001) have found that routine regulation may play an important role in protecting individuals against relapse over the course of bipolar illness. In these studies, life events associated with high levels of social rhythm disruption were found to be highly predictive of manic episode onset (Malkoff-Schwartz et al., 1998). In addition, individuals with bipolar disorder demonstrated the ability to maintain stability in daily routines during acute treatment phases (Frank et al., 1997). These findings have led to predictions that psychotherapy involving the promotion of routine regulation may have a selectively prophylactic effect on episode occurrence (e.g., mania; Frank et al., 2000). Preliminary data suggest that preventative psychotherapy involving routine regulation activities may protect individuals with BD from symptoms of the disorder (e.g., Frank, 1999).

Although there appears to be relative agreement within the BD treatment literature regarding the importance of routine regulation, various methods for achieving this level of daily structure have been proposed (e.g., Frank, 2000; Miklowitz & Goldstein, 1997). For example, Frank and colleagues (2000) typically assist individuals

with bipolar disorder to devise and maintain a structured plan for daily activities. Similarly, Miklowitz and Goldstein (1997) include treatment components (e.g., mood monitoring relative to life events) aimed at increasing awareness of how life events influence mood. In general, most of the treatment studies utilizing routine regulation include a psychoeducation component that focuses on improving an individual's understanding of the disorder. Such a component often includes a focus on the identification of risk factors for relapse, the promotion of a clearer understanding of the course of bipolar disorder, and self-monitoring the occurrence of mood fluctuations (e.g., Frank, 1999; Frank et al., 1997, 2000; Miklowitz & Goldstein, 1997).

The presumed reduction in stress reactivity (Salmon, 2001) and greater use of more effective, available coping mechanisms (Johnson & Meyer, 2004) that follows increased structure in daily living may also play an important role in relapse prevention. Given the positive correlation between stressful life events and relapse in BD (Ehlers et al., 1988, 1993; Frank et al., 1994, 2000; Malkoff-Schwartz et al., 1998; Miklowitz et al., 1988), it is surprising that behavioral treatment modalities containing behavioral activation components (i.e., BA, exercise) have not been examined more extensively. For example, exercise has been implicated in a reduction in the perceived experience of stress in daily living (Steptoe et al., 1998). Behavioral activation, in the form of exercise, may protect individuals from the effects of stress, prolong euthymic periods, and aid in relapse prevention.

Exercise may also provide a coping mechanism for stressors that have not yet occurred by increasing structure and habitual routine in an individual with Bipolar Disorder. According to Salmon's Stress Adaptation Model, the *controllability* of the

exercise stressor may provide the largest benefit of all. Such control may result in quicker adaptation to stressful events, decrease the likelihood of relapse as a result of routine regulation, lead to generalized coping responses, and decrease initial reactivity to stressful life events (Salmon, 2001).

Behavioral Activation

Behavioral Activation (BA) represents a therapeutic approach that emphasizes structured attempts to increase overt behaviors (e.g., Hopko, Lejuez, Ruggiero, & Eifert, 2003). According to Hopko and colleagues (2003), these behaviors are likely to produce improvements in the frequency of negative thoughts, depressed mood, and in the overall quality of life by putting an individual in contact with potential reinforcing environmental contingencies. Lewinsohn and colleagues (1975) first proposed BA as a behavioral treatment for Major Depressive Disorder (MDD). In Lewinsohn's model, a decrease in pleasant events or an increase in aversive events in an individual's life results in the experience of depression. The model was based on earlier conceptualizations of depression (e.g., Ferster, 1973; Lewinsohn, 1974; Lewinsohn & Graf, 1973; Skinner, 1953) that emphasized the importance of a functional analysis of behavior.

According to Lewinsohn (1975), depression can be viewed as a person-behavior-environment interaction. Low rates of response-contingent positive reinforcement or high rates of aversive experiences are likely to result in the onset of depressive symptoms. Therefore, treatment involved monitoring engagement in pleasant events and using activity scheduling to increase engagement in pleasant events. In addition, when few positive outcomes (or many negative outcomes) follow an individual's behaviors, depressed mood, negative thoughts, and avoidance behaviors are increased (Grosscup &

Lewinsohn, 1980; Lewinsohn, Youngren, & Grosscup, 1979). These experiences can be rooted in a variety of causes, including a change in the number of reinforcers available, social skill deficits, or a loss in capacity to experience an event as pleasurable (i.e., *perception* of event has changed versus a change in the actual event). Regardless of the mechanism involved, evidence suggests that depressed individuals experience fewer pleasant events and more negative events than individuals in the general population (Lewinsohn, 1974; Lewinsohn & Gotlib, 1995; Lewinsohn, Munoz, Youngren & Zeiss, 1986).

Rather than prescribing broad classes of pleasant events according to a self-reported absence of particular events in an individual's life (i.e., Lewinsohn, 1975), more recent approaches to BA (i.e., Jacobson, Martell, & Dimidjian, 2001; Martell, Addis, & Jacobson, 2001) emphasize a functional analysis of contingencies of reinforcement that may operate for an individual. In these current conceptualizations, there is no assumption that engaging in 'formally' pleasant activities (i.e., activities defined as pleasurable by the individual; Lewinsohn & Graf, 1973) will function as a positive reinforcer for an individual. Instead of focusing on an indiscriminate increase in contact with events that are presumed to be pleasant, the focus shifts toward understanding the functional aspects of behavior modification (Martell et al., 2001, 2004). Generally, current BA approaches are more idiographic in nature, giving increased attention to unique environmental contingencies that may be maintaining an individual's symptoms of depression (Jacobson et al., 2001; Lejuez et al., 2001).

According to Jacobson and colleagues (2001), behavioral activation is effective in reducing depressed mood because of a fundamental increase in opportunities to engage in

behaviors that will be positively reinforced in the natural environment. In this model, the focus is on increasing engagement in meaningful behavior/activity. Reinforcement (e.g., alleviation of distress) of meaningful activity may allow greater opportunity for personal goal attainment (e.g., Martell et al., 2004), thereby reducing depressed mood.

An additional focus in current BA models is routine regulation (Lejuez, Hopko, & Hopko, 2001, 2002; Jacobson et al., 2001; Martell et al., 2001). A substantial literature suggests that mood episodes (i.e., depressive, manic, hypomanic) in BD are triggered by dysregulation in routine (which is perceived as stressful) for individuals with BD (e.g., Ehlers et al., 1988; Frank et al., 1999, 2000; Gitlin et al., 1995; Miklowitz & Goldstein, 1997). In BA treatments, individuals are encouraged to establish and maintain a regular routine to address the problem of routine disruption thought to typically precipitate mood fluctuation.

The routine regulation approach represents a core feature of BA in which individuals are encouraged to adhere to a planned routine, even in the face of major life stressors (Martell et al., 2004). Cognitive and emotional components associated with stress are indirectly targeted by bringing an individual in contact with more positive consequences for overt behaviors that are directly targeted for change. Individuals are asked to commit to changing behaviors, regardless of internal states (e.g., feeling depressed or lethargic, lack of motivation; Martell et al., 2004). This commitment allows a therapist and client to examine whether motivation to change follows the behavioral activation and change. If motivation is not a by-product of behavior modification, an examination of other positive outcomes that may be secondary to the action(s) is performed (e.g., increased time spent with friends, changes in mood). Clients are taught

that “activity breeds activity, and inactivity breeds inactivity” (Martell et al., 2004, p.160). Clients are asked to test this rule/algorithm, ultimately resulting in the implementation of a new rule/algorithm (e.g., “When feeling blue, get active” versus “When feeling blue, shut down”; Martell et al., 2004, p. 161). This new rule development and implementation parallels the BA concept of de-pathologizing thinking or behaving that is often consistent with various conditions (e.g., depression).

According to the BA approach, the downward spiral toward less productive activity (cognitive and physical) is central to the struggle often observed in mood disorders. In this treatment approach, clients are encouraged to increase engagement in productive behaviors regardless of how they are feeling. According to BA models (e.g., Jacobson et al., 2001; Martell et al., 2001), this behavior modification acts to disengage the individual from the fundamental dependence on mood that is frequently observed in the affective disorders. The current investigation will provide an analysis of the effectiveness of exercise (a specific form of BA) in regulating mood. Exercise will be compared to other forms of behavioral activation (i.e., productive behaviors that are not exercise) in the study. This approach will provide additional knowledge regarding the issue of routine regulation versus increase in specific productive behaviors (e.g., exercise) as viable means of regulating symptoms of mood disturbance.

Behavioral Activation and Major Depressive Disorder (MDD)

Over the past decade, there has been an increase in interest in BA as a comprehensive treatment for clinical depression. The revitalization of behavioral treatments for MDD has led to the development of two new interventions: Behavioral Activation (BA; Martell et al., 2001) and the Brief Behavioral Activation Treatment for

Depression (BATD; Lejuez, Hopko, & Hopko, 2001, 2002). Both approaches represent extensions of conventional behavioral models regarding the treatment of depression (e.g., Ferster, 1973; Lewinsohn, 1975; Skinner, 1953).

In order to examine the effectiveness of using BA strategies in the treatment of clinical depression, Jacobson and colleagues (1996) conducted a large scale treatment outcome study. In this study, 150 clinically depressed individuals received a comprehensive cognitive treatment (including BA and direct cognitive manipulation components), a combined BA and skills training approach aimed at modifying automatic thoughts (AT), or behavioral activation (BA) alone. Results indicated that the AT and BA interventions were as effective in reducing depressive symptoms and improving cognitive functioning as the comprehensive cognitive therapy both at post-treatment and at a 6-month follow-up. In addition, the BA approach was as effective in reducing negative thought frequency (e.g., dysfunctional attributional styles) as the comprehensive cognitive treatment.

This initial outcome study comparing a comprehensive cognitive-behavioral (CBT) program to the BA component alone demonstrated that BA is as effective regarding overall treatment outcome and alteration of dysfunctional beliefs and negative thinking styles (Jacobson et al., 1996). In a 24-month follow-up of treated individuals in the Jacobson et al., 1996 study, Gortner and colleagues (1998) found that BA alone and the comprehensive CBT protocols were equally effective in preventing relapse. In addition, higher rates of relapse were associated with lower levels of activation at post-treatment (Gollan, 2001).

Another research group (Lejuez et al., 2001, 2002) developed a brief behavioral activation treatment (BATD) for clinical depression. In this brief intervention, BATD (i.e., 8-15 sessions), researchers predicted that increased contact with reinforcers for healthy behavior (or decreased contact with reinforcers for depressed behavior) would result in a decrease in depressive behaviors and an increase in healthy behaviors.

Consistent with earlier conceptualizations of BA for the treatment of depression (e.g., Lewinsohn, 1975; Rehm, 1977), the focus in BATD is on identifying behavioral goals within major life areas. In a preliminary study with depressed individuals attending a community mental health center, BATD appears to be a promising method for reducing depressive symptoms. In addition, in a series of case studies, BATD resulted in a substantial decrease in depression index scores (i.e., BDI-II scores) for individuals with moderate levels of clinical depression (Lejuez et al., 2001).

More recently, Hopko and colleagues (2003) completed a randomized pilot trial using BATD in an inpatient setting. In this study, 25 inpatients were randomly assigned to either a BATD condition ($N = 10$) or a supportive psychotherapy condition ($N = 15$). All participants were simultaneously receiving antidepressant medication (i.e., tricyclic antidepressants or selective serotonin reuptake inhibitors). Results of this study suggest that BATD leads to significant decreases in depressive symptoms from pre- to post-treatment. Despite a small sample size utilized in the study, researchers reported a large effect size for BATD ($d = .73$). The results of this study are consistent with previous evidence that BA represents an effective treatment for individuals with comorbid anxiety and depression symptoms (Hopko, Lejuez, & Hopko, 2004) and as an adjunct to pharmacotherapy (Hopko, Lejuez, LePage, McNeil, & Hopko, 2003). In addition, this

study demonstrates the feasibility and effectiveness of a brief psychotherapeutic intervention for depressed inpatients, which has not received much attention in the empirical literature (Jarrett, 1995).

Some investigations utilizing behavioral interventions have incorporated cognitive strategies as well (e.g., reinforcing positive thoughts; Lewinsohn et al., 1986; Lewinsohn, Hoberman, Teri, & Hautzinger, 1985). The addition of cognitive strategies was thought to be important because: 1) behavioral interventions were thought to be insufficient when used as a comprehensive treatment for MDD; and 2) the absence of direct cognitive manipulations has been labeled as a limitation of behavioral interventions (Lewinsohn et al., 1986; Lewinsohn, Clarke, Hops, & Andrews, 1990; Lewinsohn & Gotlib, 1995). However, little evidence has supported the premise that the inclusion of cognitive components in the treatment of MDD is necessary for clinically significant change to occur (Lejuez et al., 2001). Rather, research suggests that cognitive changes are just as likely to occur when environment-based manipulations are performed compared to direct cognitive interventions (e.g., Simons, Garfield, & Murphy, 1984). In addition, researchers have found that benefits of cognitive-behavior therapy are most evident during the initial sessions of treatment, the phase when behavioral interventions are often more prominent (Beckham & Leber, 1995; Hollon, Shelton, & Davis, 1993; Otto, Pava, & Sprich-Buckminster, 1996).

Evidence supporting the premise that BA alone is sufficient to produce a positive treatment outcome in clinically depressed individuals (e.g., Hopko et al., 2003; Jacobson et al., 1996; Lejuez et al., 2001) offers promise for more efficient interventions that retain their effectiveness. Given that group interventions represent another method of achieving

therapeutic efficiency regardless of therapeutic modality (e.g., Shapiro, Sank, Shaffer, & Donovan, 1982), researchers have begun to investigate the effectiveness of behavioral activation applied in a group setting (Porter, Spates, & Smitham, 2004). In order to examine the effectiveness of BA in a group modality, Porter and colleagues (2004) conducted a pilot investigation of behavioral activation group therapy (BAGT). In this study, group therapy sessions were based on the Jacobson et al. (1996) BA treatment manual. The researchers devised a structured treatment manual which provided session-by-session instructions for group facilitation (Porter et al., 2004). In the study, 37 participants from four different community mental health centers met DSM-IV-TR criteria for MDD, according to the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID; First et al., 1997). Participants were assigned to either a wait-list control condition (i.e., received BAGT after 4-6 weeks) or an immediate treatment (i.e., BAGT) condition. BAGT consisted of 10-weeks of weekly sessions, lasting 95 minutes each.

Porter and colleagues (2004) found that BAGT resulted in significant reductions in depressive symptoms from pre-treatment to 3-month follow-up assessment for both groups. The researchers also found additional evidence of clinically significant change relative to the diagnostic status of participants. At the time of initial assessment (i.e., pre-treatment) 100% of participants met criteria for a diagnosis of MDD, whereas only 26.9% met diagnostic criteria for MDD at post-treatment and at a 3-month follow-up assessment (a statistically significant reduction, $p < .001$). Results of this investigation (Porter et al., 2004) support the hypothesis that BA administered in group settings may be an adequate

intervention for individuals in a community mental health setting meeting criteria for MDD.

BA appears to be an efficient and cost-effective modality of treatment for individuals meeting diagnostic criteria for MDD in a variety of settings (e.g., outpatient, inpatient), particularly when compared to traditional forms of therapeutic interventions (e.g., Cognitive Therapy, Interpersonal Therapy). Recent evidence for the effectiveness of BA administered in a group modality provides additional support for the utility of behavioral interventions for clinical depression, in that there may be potential for a “multiplier effect” (Porter et al., 2004, p. 297) regarding enhancing efficiency of the treatment. Overall, evidence suggests that BA may represent an effective adjunctive or sufficient treatment for MDD (e.g., Dimidjian et al., 2003; Jacobson et al., 1996; Lejuez et al., 2001; Porter et al., 2004). Future research is needed to determine mechanisms of change within the BA approach, and whether various forms of BA (e.g., exercise) may produce differential effects in certain populations of individuals with MDD.

Models of Exercise

Despite continuing research that demonstrates the positive effects of exercise on depressed mood, no single theory adequately explains how exercise leads to a reduction in depressive symptoms. However, a plethora of physiological and psychological theories have been proposed to explain the interaction between mood state and exercise. Many theories focus on physiological factors such as an increase in core body temperature (Thermogenic Model; Petruzzello et al., 1991) and differential neocortical activation following exercise (Kubitz & Landers, 1993). Other models focus on biochemical mechanisms such as increased secretion of amine metabolites, as well as

serotonin synthesis and metabolism (Monoamine Hypothesis; Dishman et al., 1997; Dunn & Dishman, 1991; Esler et al., 1990; Morgan, 1996). The central premise of these biochemical theories is that exercise acts on the same pathway that antidepressant medications target in the treatment of clinical depression (Ransford, 1982).

Additional biochemical theories focus on specific areas of the brain (i.e., Hypothalamic-pituitary-adrenal (HPA axis; Peronnet & Szabo, 1993) or specific endogenous chemicals (e.g., endorphins, enkephalins) or systems (e.g., endocannabinoid system) to explain the antidepressant properties of exercise. Theories such as the HPA axis model propose that stress hormones are released in response to physical (e.g., exercise) and psychological stress experiences. Accordingly, habitual levels of exercise are thought to decrease the amounts of stress hormones secreted from the HPA axis, resulting in lower levels of depression and stress reactivity (Peronnet & Szabo, 1993; Salmon, 2001).

According to the Endorphin Hypothesis, endogenous opioids (e.g., endorphins, enkephalins) are released during and after exercise and result in a euphoric state frequently reported by individuals who are physically fit (Christie & Chesher, 1982). In a recent investigation, Sparling and colleagues (2003) confirmed the existence of an endogenous cannabinoid system by identifying two cannabinoid receptors (CB1 and CB2) and their naturally occurring ligands (anandamide and 2-AG). These chemicals are located predominantly in regions of the brain responsible for motor function, emotion regulation, and cognitive functioning (Glass, Faull, & Dragunow, 1997). The endocannabinoid system hypothesis addresses various weaknesses of former theories, but

needs further research to determine its adequacy in explaining the antidepressant effect of exercise.

In addition to the aforementioned physiological models, several psychological theories have been proposed to explain the effect that exercise has on depressed mood (e.g., Gauvin, Rejeski, & Norris, 1996; Martinsen, 1990; Salmon, 2001; Steptoe et al., 1998). Psychological theories of exercise focus on different constructs such as intrinsic motivation (Cognitive Evaluation Theory; Deci, 1975), self-determination (Self-Determination Theory; Deci & Ryan, 1985, 1991), and self-efficacy (Self-Efficacy Theory; Bandura, 1977). In addition, several social-cognitive and expectancy-value theories (e.g., Theory of Reasoned Action; Ajzen & Fishbein, 1980, Theory of Planned Behavior; Ajzen, 1988) address issues of intention, perception of control, and actual engagement in an activity (exercise). In general terms, these models focus on a variety of mechanisms that might explain the link between exercise and reduced depressed mood (e.g., improved accomplishments and confidence, positive distraction, improved self-esteem, environmental reinforcement, increasing positive coping skills available for use during stressful situations).

In an attempt to bridge these various psychological models regarding exercise and mood, Salmon (2001) has proposed an integrative model called the Stress Adaptation Model. In the Stress Adaptation Model, an increase in life structure occurs as a result of including exercise in one's daily routine. In addition, individuals who exercise on a daily basis may experience a simultaneous decrease in stress reactivity (Salmon, 2001). Support for this theory can be drawn from previous research that found reductions in the perceived experience of stress in daily living in individuals who exercise on a regular

basis (Steptoe et al., 1998). According to the Stress Adaptation Model, regular exercise protects individuals from the effects of stress, and ultimately prolongs euthymic periods or prevents depressive relapse. In this model, exercise is also conceptualized as a stressor, albeit one with potential positive benefits. In addition, Salmon (2001) proposes that the *controllability* of the exercise stressor may provide a substantial benefit. This controllability may result in quicker adaptation to other stressors, decrease the likelihood of relapse as a result of routine regulation, lead to the development of more effective coping responses, and decrease reactivity to stressful life events (Salmon, 2001). It may be possible that a regular exercise routine may provide a coping mechanism for stressors that have not yet occurred by increasing life structure and habitual routine in an individual with Bipolar Disorder.

Exercise as a Form of Behavioral Activation

There are widely accepted benefits (both psychological and physiological) of including a regular pattern of exercise in one's lifestyle (e.g., Biddle & Mutrie, 2001; Dubbert, 2002; Martinsen, 1994; Richardson, Faulkner, McDevitt, Skrinar, Hutchinson, & Piette, 2005). There has been a recent increase in attention to the potential benefits of exercise within the psychological and psychiatric community (e.g., Gauvin & Spence, 1995). Recent consensus statements have begun to address the importance of including a healthy regimen of physical activity in the lifestyle of individuals of all ages. For example, the National Institute for Mental Health (NIMH) released a statement asserting that regular physical exercise may be useful as an adjunct form of treatment for many psychological disorders (Morgan & Goldston, 1987), although no indication of potential benefits of such a treatment protocol was mentioned (Biddle & Mutrie, 2001). Possibly

of greater impact, in 1995 both the American Society of Sport Medicine (ACSM) and the Center for Disease Control (CDC) recommend a moderate level (i.e., 20-30 minutes) of regular physical activity (i.e., 6 or more days per week) to increase overall health and well-being (Pate et al., 1995). In addition, inactivity significantly increases the risk of all-cause mortality (Biddle & Mutrie, 2001; Dubbert, 2002).

An additional statement on health and physical activity came from the United States Surgeon General's Office in 1996, indicating that inclusion of regular physical activity is essential in maintaining physical and mental well being. Similarly, in a review of the exercise literature, Dubbert (2002) reported that moderate levels of exercise have consistently demonstrated an inverse dose-response relationship with various physical illnesses (e.g., coronary heart disease), and have shown potential for preventing or decreasing one's risk for other conditions (e.g., colon cancer, non-insulin-dependent diabetes mellitus). In addition, researchers are emphasizing the potential role that exercise may play in preventing mental illness (e.g., depression) and increasing the functional capacity of those that have already developed impairments (Biddle & Mutrie, 2001; Dubbert, 2002; Richardson et al., 2005).

Exercise (i.e., structured regimens of either aerobic or anaerobic activity) and general physical activity (i.e., activity that can be achieved through unstructured methods such as occupational endeavors and housework) have consistently been associated with positive effects on depressed and anxious mood (Biddle & Mutrie, 2001; Dubbert, 2002; Martinsen et al., 1989; Martinsen, 1994). For example, using experience sampling procedures, Gauvin and colleagues (1996) found a significant increase in positive affect after female participants endorsing various levels of feeling states engaged in an acute

bout of exercise (30 minutes at 75 – 80% heart rate recovery). In this study, the greatest improvements in positive affect were reported by females indicating higher levels of depression before the exercise regimen was completed.

In another study, Babyak and colleagues (2000) completed a 10-month follow-up of clinically depressed individuals who either completed a 16-week exercise treatment protocol, received a standard regimen of antidepressant medication (i.e., sertraline), or received a combination of exercise and medication (Blumenthal et al., 1999). Results of the follow-up study (Babyak et al., 2000) indicated significantly lower rates of relapse in the exercise-only condition compared to antidepressant medication alone or the combination treatment. Interestingly, Babyak and colleagues (2000) have suggested that a combined treatment may actually undermine the benefit of exercise for some individuals. For example, individuals may experience a sense of mastery and positive self-regard during exercise adoption and maintenance that is less likely to occur in the presence of a medication regimen. This assertion is consistent with findings that individuals suffering from a mental illness report that exercise is the “most effective” treatment component (Martinsen et al., 1989; Porter et al., 2004; Sexton, Maere, & Dahl, 1989).

Above and beyond the acute reduction of affective distress and impairment, exercise may serve a preventative role when used as an adjunct treatment. Farmer and colleagues (1988) found that females who were minimally active were two times as likely as females engaging in higher levels of exercise to meet criteria for clinical depression over the course of an 8-year follow-up investigation. Similarly, males who were inactive were more likely to maintain a state of depression. Similar results were obtained in a

study utilizing a 10- and 20-year follow-up protocol (Camacho, Roberts, Lazarus, Kaplan, & Cohen, 1991) for males and females who were initially depressed. In this study, minimally active males and females were at significantly higher risk of developing depression than those individuals engaging in a moderately strenuous regular program of exercise activity (i.e., three 30-minute sessions per week of jogging or cycling at 75 – 80% heart rate reserve). In an epidemiological survey, males engaging in three or more hours of sport activity per week were 27% less likely to become depressed than their minimally active counterparts (Paffenbarger et al., 1994).

Findings that exercise appears to have a more prominent effect on individuals reporting a higher level of depression (e.g., Gauvin et al., 1996) are consistent with current research indicating that behavioral activation (BA) alone (Jacobson et al., 1996; Lejuez et al., 2001; Martell et al., 2001) may represent an adequate treatment for MDD. In addition, evidence of exercise's effectiveness in individuals with higher levels of depression is consistent with current research suggesting that the benefits of complete psychotherapeutic interventions (i.e., combined BA and direct cognitive manipulations) are most evident during the early phase of treatment when behavioral interventions are more prominent (Beckham & Leber, 1995; Hollon, Shelton, & Davis, 1993; Otto, Pava, & Sprich-Buckminster, 1996). This information is particularly interesting, given ongoing debates regarding the effectiveness of psychotherapeutic interventions in severe cases of depression (e.g., NIMH-TDCRP; Elkin et al., 1989 findings that medication is most effective in severe cases of depression).

Individuals with psychological disorders have consistently been found to engage in little to no exercise (i.e., sedentary lifestyle; Biddle & Mutrie, 2001; Dubbert, 2002;

Richardson et al., 2005). Exercise may represent a form of BA that is capable of producing decreases in depressive symptomatology (including cognitive dysfunctions) similar to previous investigations of BA as a treatment for clinical depression (e.g., Jacobson et al., 1996). The inclusion of exercise in treatment regimens has been recommended as an adjunct to other interventions (e.g., Pate et al., 1995). Consistent with previous findings that BA alone is an adequate treatment for MDD (Jacobson et al., 1996; Lejuez et al., 2001; Martell et al., 2001), some researchers assert that exercise may be an equally effective intervention strategy (e.g., Craft & Landers, 1998; Nicoloff & Schwenk, 1995). Although there are no guidelines that propose algorithms for treatment decisions regarding if and when to use exercise in the treatment of psychological disorders, the positive effect of engaging in regular exercise is gaining increasing support and attention.

Moderate levels of exercise have consistently been shown to positively impact mood (e.g., Blumenthal et al., 1999; Martinsen et al., 1989) and prevent depression (e.g., Babyak et al., 2000; Craft & Landers, 1998). In addition, researchers have extended these investigations to include evaluations of exercise as an adjunct treatment for individuals with chronic and severe forms of mental illness (e.g., Schizophrenia). For example, exercise may be useful in decreasing psychotic hallucinations (Falloon & Talbot, 1981) and decreasing depression in chronic mental illness (e.g., Faulkner & Biddle, 1999; Pelham, Campagna, Ritvo, & Birnie, 1993).

Although exercise is widely considered an essential component of a healthy lifestyle, surprisingly few people engage in significant levels of regular exercise. Approximately 60% of adults in the United States are minimally active, and 22% engage

in no leisure-time activity at all (NHANES-III, 1996). Dubbert (2002) asserted that only 33% of adults achieve the recently recommended guidelines published by three sources (ACSM and CDC in Pate et al., 1995; Surgeon General, 1996). Levels of activity are reported to be even lower in individuals with mental illness (Goldberg & Huxley, 1992; Martinsen et al., 1989), raising a question of causality relative to the relationship between exercise and mental illness.

Treatment of Bipolar Disorder

Traditional treatment of Bipolar Disorder has focused on psychopharmacological agents (e.g., lithium carbonate; divalproex sodium, Depakote; carbamazepine, Tegretol) that are thought to stabilize and maintain mood and increase an individual's ability to function in a manner that improves quality of life (Keck & Licht, 2000; Keck & Manji, 2002; Keck & McElroy, 2002). The prophylactic value of such treatment modalities is at least two-fold: 1) to aid in the prevention of future mood episodes, and 2) to minimize the duration and severity of episodes that do occur, thereby increasing the potential for a better long-term prognosis for individuals with BD.

Psychopharmacological treatment typically involves three separate and distinct phases (e.g., Keck & McElroy, 2002). First, the *acute stage* of treatment entails efforts to control the most severe symptoms of the current mood episode. During this phase, traditional focus (both empirical and practical) has been on the administration of antipsychotic medications (e.g., imipramine, clozapine, iloperidone), often in concert with mood stabilizing agents (e.g., lithium). During the second phase of treatment (i.e., *stabilization phase*), the goal is to help the individual recover fully from the acute stage of suffering. Specifically, treatment will focus on ensuring that residual symptoms and/or

social-occupational problems have abated. Occasionally antidepressant medications are administered during this phase of treatment if (and only if) a manic episode has fully remitted and the antidepressant medication is less likely to trigger the experience of manic symptoms. Finally, during the *maintenance stage* of treatment, the focus is on treating any residual symptom expression, and ultimately on the prevention of recurrence of mood episodes. Treatment during this phase has traditionally been monotherapy, with the administration of a single mood-stabilizing agent if no residual depressive symptoms are present.

Effectiveness of Pharmacological Treatment of BD

In recent years, research has grown both in quality and quantity of clinical trials investigating pharmacological treatments for BD. This increase in attention has led to substantial advances in knowledge regarding the efficacy of various mood stabilizing and antipsychotic medications in the treatment of acute mania (e.g., carbamazepine, lithium, ziprasidone). Recent research utilizing parallel-group, double-blind, placebo-controlled designs (e.g., Bowden, Brugger, Swann, Calabrese, Janicak, Petty, et al., 1994; Pope, McElroy, Keck, & Hudson, 1991) has resulted in two new medications (i.e., divalproex and olanzapine) receiving approval from the U.S. Food and Drug Administration (FDA) for the treatment of acute mania. This expansion of available indicated treatments for acute mania represents the first since 1970, when Lithium received approval from the FDA as a treatment of “manic episodes of manic-depressive illness” (Goodwin & Jamison, 1990). In addition, research investigating potential treatments for the manic phase of BD has indicated preliminary support for the use of new antiepileptic medications (e.g., topiramate, lamotrigine, oxcarbazepine) for the treatment of acute

mania (e.g., McElroy & Keck, 2000). Although there have been significant advances in recent years regarding efficacy of pharmacological interventions for acute mania in BD, less is known about medical treatments for bipolar depression (Keck & McElroy, 2002).

Pharmacological treatments for bipolar depression continue to be understudied (Keck & McElroy, 2002), resulting in limited available information regarding how to treat the depressive phase of BD. Recent attempts have been made to expand empirical understanding of how pharmacological agents can be used to treat bipolar depression (e.g., Calabrese, Bowden, Sachs, Ascher, Monaghan, & Rudd, 1999; Nemeroff, Evans, Gyulai, Sachs, Bowden, Gergel, et al., 2001). However, no conclusive evidence is available regarding the efficacy of antidepressant medications in treating bipolar depression. Furthermore, the optimal duration of antidepressant treatment for bipolar depression remains unclear, given the risk of inducing manic symptoms with prolonged antidepressant use (Altshuler, Post, Leverich, Mikalaukas, Rosoff, & Ackerman, 1995; Keck & McElroy, 2002).

Perhaps the most important aspect of pharmacological treatment for BD is the use of long-term mood stabilizing agents. Researchers have consistently reported high rates of relapse for individuals with BD that increase with time. Specifically, rates of relapse at 1-year follow-up have been estimated between 37 and 67% (Gitlin et al., 1995; Shapiro, Quitkin, & Fleiss, 1989). In addition, at a 2-year follow-up with traditional medication treatment (i.e., lithium), only approximately 40% of individuals are episode-free (Gelenberg et al., 1989). This percentage declines to a mere 27% survival at 5-years follow-up (Gitlin et al., 1995), indicating an increased need for advancements in the understanding of the maintenance therapy for individuals with BD.

Recent studies of mood stabilizing medications (e.g., lithium, valproate, carbamazepine) indicate the potential efficacy of using these agents during the maintenance stage of therapy (e.g., Keck, Welge, Strakowski, Arnold, & McElroy, 2000). However, results from these studies also indicate that only a minority of individuals are able to maintain euthymic status when treated with these medications alone (Keck et al., 2000). Overall, more research is needed to address which pharmacological treatments are most useful for treating BD. In addition, research is needed to address whether certain medications are most efficacious during various stages of illness and treatment. Two major research initiatives (i.e., Stanley Foundation Bipolar Network; Leverich et al., 2001; NIMH STEP-BD Program; Sachs et al., 2000) have begun to help fill the substantial gaps that exist in our understanding of how to most effectively provide treatment (both pharmacological and psychological) for individuals with BD.

Psychological Treatments for BD

Psychological models of BD have also received increased attention in recent years. Efforts have been made to apply Beck's (1972) cognitive triad model of depression to the manic phase of illness, in order to increase the model's applicability to the treatment of BD (Beck, Rush, Shaw, & Emery, 1979; Beck, 1996). In Beck's (1972) cognitive triad model of depression, the depressed individual is thought to view the world through a distorted set of depressive schemata, including a pessimistic view of the self, the world, and the future. In the extended model (i.e., Linear Schematic Processing Model), mania is viewed as a mirror image of depression (i.e., positive cognitive triad of the self, the world, and the future). Cognitive vulnerability for a manic episode was conceptualized in a similar manner to the model for depression, with directionality of

dysfunctional beliefs being the primary difference (i.e., hyperpositive thinking versus hypernegative thinking). More recently, Beck (1996) proposed an Integrative Model, which includes a network of schemas (i.e., modes), including cognitive, affective, and motivational constructs that act in synchrony to produce goal-directed strategies characteristic of the manic phase of illness.

Although Beck's conceptualization of BD is consistent with dysregulation of mood relevant to the depressive spectrum of mood disorders, there are significant limitations in its ability to adequately explain the development and maintenance of BD. Researchers have argued that the cognitive triad model of mania is largely derivative (e.g., Scott, 2001). According to Scott (2001), the model fails to consider the extensive biological vulnerability critical to the development and maintenance of BD that has been consistently reported in the literature.

Over the past 20 years, anecdotal and single-case studies have provided preliminary evidence in support of the use of cognitive therapy (CT) in the treatment of individuals with BD (e.g., Chor, Mercier, Halper, 1980; Scott, 1995; Wright & Schrod, 1992). In addition, some researchers have conducted more controlled investigations of the utility of cognitive therapy in treating BD. Cochran (1984) found that CT was beneficial in increasing medication adherence (i.e., only 21% in CT group versus 57% in standard care group discontinued medication). In a larger study ($N = 68$), Perry and colleagues (1999) investigated the use of cognitive and behavioral techniques to help individuals with BD identify and manage early warning signs of relapse. In this study, individuals receiving the intervention were observed to have fewer manic relapses,

significantly fewer hospitalization days, and demonstrated higher levels of social functioning and work performance levels.

Further evidence showing preliminary support for the use of cognitive therapy in the treatment of individuals with BD comes from a pilot investigation (Scott, Garland, & Moorhead, 2001). Researchers compared cognitive therapy to a wait-list control condition, and found that individuals receiving cognitive therapy had fewer relapses, a general reduction in both depressive and manic symptoms, and increased self-esteem and medication adherence. According to the authors, the use of cognitive therapy may represent a highly acceptable form of intervention in this population (Scott et al., 2001; Lam et al., 2001).

To date, psychological theories of BD have been insufficiently investigated, and few insights are available to explain how therapy achieves its effects (i.e., mechanisms of change; Palmer & Scott, 2001; Scott et al., 2000). However, research attention relative to psychosocial interventions for BD has increased in the past decade. The discussion that follows will provide a more comprehensive review of the three most validated approaches to the psychological treatment of Bipolar Disorder (i.e., FFT, IPSRT, CBT).

Family Focused Therapy (FFT). To date, the most validated form of psychotherapeutic intervention for BD is Family Focused Therapy (FFT; Miklowitz & Goldstein, 1997). This treatment modality consists of six objectives aimed at improving an individual and his/her family's ability to cope with an episode of BD: 1) Integrating the experiences associated with mood episodes in BD, 2) Accepting the notion of a vulnerability to future episodes, 3) Accepting a dependency on mood-stabilizing medication for symptom control, 4) Distinguishing between the patient's personality and

his or her diagnosis of BD, 5) Recognizing and learning to cope with stressful life events that trigger recurrences of BD, and 6) Reestablishing functional relationships after a mood episode. FFT is a time limited treatment (e.g., 21 outpatient sessions), consisting of three interrelated modules (e.g., psychoeducation, communication enhancement training, and problem-solving skills training) directed at individuals who have experienced a recent episode of mania or depression (Miklowitz & Goldstein, 1997). Generally, the therapy targets the most relevant psychosocial factors associated with poor prognosis in BD (e.g., disrupted rhythms, interpersonal interactions).

An important aspect of FFT is acknowledging the problem of “breakthrough episodes” that occur regularly in the traditional administration of psychopharmacological treatment for BD. A primary reason for breakthrough episodes in BD is medication non-adherence. FFT aims to increase adherence to drug regimens in BD individuals and to teach skills for general symptom recognition and management. An essential element of FFT is the requirement that the individual must be concurrently seeking services of a care provider (e.g., Psychiatrist) who monitors his or her medication regimen (e.g., mood stabilizer such as lithium carbonate or divalproex sodium). Given the likelihood of relapse in the absence of standard pharmacotherapy, individuals who refuse medications are not accepted into FFT. Although this approach is warranted under the theoretical grounding of FFT, it is important to note the potential associated risks. Specifically, individuals who have recently experienced a manic episode (still possibly experiencing residual symptoms) may likely offer resistance to traditional interventions because of the enjoyment associated with productive feelings experienced during the recent state of euphoria.

Another component of FFT for BD is to assist individuals in learning skills that augment social and occupational role functioning. In particular, individuals learn more adaptive coping strategies in dealing with stressful life events. The presence of life stressors has been shown to place an individual with BD at greater risk for relapse (e.g., Ehlers et al., 1993; Johnson & Meyer, 2004). FFT targets prominent stressors (e.g., social and occupational), with a focus on teaching clients adaptive coping strategies in order to decrease the risk of relapse (Miklowitz, 2001; Miklowitz & Goldstein, 1997). In one study, researchers found that social and family stressors (e.g., job functioning, marital distress) decreased time to relapse in individuals with BD (Gitlin et al., 1995).

Given that manic episodes, in particular, are often precipitated by significant stressful life events, intervention often entails helping individuals re-establish previous support outlets, or develop new outlets in this domain. According to the FFT model, the family can be viewed as a potential outlet for social support in times of distress. However, highly emotional family members are thought to be less able to provide the necessary support, or the affected individual may be unreceptive to the nature of support offered by these family members. Therefore, FFT focuses on teaching the client how to regulate social rhythms (e.g., to increase positive communication between family members, decrease emotional reactivity among members of the available support system). In the FFT model, regulation of social rhythms may act as a buffer to stressful life events experienced by the individual with BD. Research investigating the utility of family therapy as an adjunct to pharmacotherapy for BD suggests that individuals report significant decreases in mood symptoms and increased compliance with medication regimens (Miklowitz, George, Richards, Simoneau, & Suddath, 2003), as well as fewer

relapses and longer delays before relapses (e.g., Miklowitz et al., 2000; Simoneau, Miklowitz, Richards, Saleem, & George, 1999). Additional evidence suggests that FFT may be superior to standard clinical management (Miklowitz et al., 2000) and individual therapy (Rea, Tompson, Miklowitz, Goldstein, Hwang, & Mintz, 2003) at 1-year and 2-year follow-up, respectively. Taken together, evidence suggests that FFT represents an efficacious adjunct to traditional forms of treatment for BD.

Interpersonal and Social Rhythm Therapy (IPSRT). Based on a theory of social zeitgebers and social zeitstörers (e.g., events that affect the biological clock; Frank, 2000), Interpersonal and Social Rhythm Therapy (IPSRT; Frank et al., 1994) represents a combination of traditional Interpersonal Therapy (IPT) and contemporary behavioral techniques. Research supporting Interpersonal Therapy (IPT) in the treatment of unipolar depression (e.g., Frank et al., 1990), along with studies supporting the role of a psychobiological influence (e.g., circadian rhythms, sleep-wake cycles) in the development and maintenance of BD (e.g., Ehlers et al., 1988, 1993; Monk, Kupfer, Frank, Ritenour, 1991), provide empirical grounding for the IPSRT approach. Generally, the IPSRT model posits that social rhythm disruption, either negative or positive, may be responsible for relapse into a mood episode for individuals with BD (Frank et al., 2000). Therefore, the IPSRT framework targets a variety of life issues that may disrupt social rhythms. The approach identifies the following targets: 1) regularization of daily routines and social rhythms, 2) resolution of interpersonal conflict, and 3) psychoeducation regarding development and maintenance of BD, and implications of living with the disorder, and relapse prevention (specific to bipolar depression).

In IPSRT, there are four distinct phases of treatment. Phase I involves taking an extensive history of the illness, collecting information with the interpersonal inventory, identifying interpersonal problem areas, and self-monitoring on the Social Rhythm Metric (SRM). The SRM is a measure that is used for continuous assessment throughout the course of treatment in the IPSRT framework. The second phase of treatment involves managing symptoms and stabilizing social rhythms. During this phase of treatment, a collaborative effort is made to identify appropriate strategies for social and interpersonal rhythm management. Phase III of IPSRT involves the maintenance of regular social rhythms through repeated implementation of strategies that are identified as useful for the individual to help promote consistent patterns of daily behaviors (e.g., sleeping, eating, social stimulation). Lastly, Phase IV (termination) of the framework involves managing symptom recurrences and new (or recurrent) interpersonal difficulties, and working toward discharge from treatment.

The IPSRT approach has received preliminary support from initial reports from the Maintenance Therapies in Bipolar Disorder (MTBD) program being conducted by Frank and colleagues at the University of Pittsburgh to evaluate the efficacy of IPSRT for the treatment of BD-I. In the MTBD program, IPSRT (plus medication management) is compared to clinical management (CM; plus medication management) during both acute and maintenance phases of treatment. In an initial report from the MTBD protocol, Frank and colleagues (1997) asserted that individuals with BD can successfully maintain stabilization of daily routines during acute treatment of manic or depressive episodes. In this investigation, participants were evaluated over the course of (up to) 52 weeks of acute treatment. The researchers found that individuals receiving IPSRT had

significantly greater increases in stability in daily routines than counterparts receiving CM. In addition, individuals receiving IPSRT were more likely to remain in treatment than individuals receiving standard CM.

In a second report from the MTBD protocol, Hlastala and colleagues (1997) found that IPSRT decreased time to clinical remission (i.e., 21 weeks versus 40 weeks) when compared to standard CM during acute treatment of manic and depressed episodes. In addition, individuals in acute manic phases of illness achieved remission faster than those in a depressed phase of illness. In a more recent report from the MTBD protocol, Frank and colleagues (1999) evaluated the use of IPSRT during preventative phases of treatment. In this investigation, 82 participants were found to exhibit lower rates of relapse when receiving the same treatment across acute and preventative phases of intervention (e.g., IPSRT during both phases). In addition, these researchers noted that receiving IPSRT during acute treatment, followed by CM (i.e., loss of IPSRT) during preventative treatment, resulted in substantially higher rates of relapse into depressive episodes. These preliminary studies provide support for the widely accepted link between life events that are associated with social rhythm disruption and episode relapse (e.g., Ehlers et al., 1993; Monk et al., 1991).

Although the IPSRT approach has received preliminary support, a significant limitation of the approach is that therapy is projected to last in excess of two years. In the age of managed care, particularly, this may be contraindicated of a cost-effective treatment approach. Conversely, however, the approach was formulated specifically for the treatment of BD, and may be applicable to all stages of BD symptom expression (e.g., manic, depressed, euthymic). According to the developers of IPSRT, this treatment can

assist in stabilization during acute stages of manic and depressive symptom experience, and recent reports suggest that IPSRT may promote periods of euthymia in individuals with BD (Frank & Hlastala, 2000). To date, no research has examined mechanisms of change for IPSRT.

Cognitive Behavioral Therapy (CBT). Otto and colleagues (2003) proposed CBT as a useful and validated approach to the treatment of bipolar depression, given the approach's strength and ability to address the treatment of common comorbid conditions (e.g., Panic Disorder). CBT for BD focuses on relapse prevention, following the stabilization of acute mood disruption. The focus is on early detection and intervention, management of stress and lifestyle, and treatment of co-occurring symptoms and conditions.

Unlike IPSRT, which promises the ability to treat all phases of the disorder, CBT specifically targets bipolar depression (BPD). Specific targets of the approach include emotional regulation, assertiveness training, and the treatment of any comorbid conditions. The approach also includes activity management (e.g., positive events scheduling), consistent with BA procedures mentioned earlier (e.g., Lewinsohn, 1975). Cognitive restructuring is heavily utilized in the approach, with extensive use and repetition of teaching metaphors (e.g., Coaching a Little League Baseball Player metaphor, Otto et al., 1999, pp. 167-168; compares a yelling approach to 'coaching' to a more empathic/explanatory approach to examine nature of the client's cognitions). Also, CBT for BPD includes teaching emotional and social problem-solving skills, which are likely to be difficult areas for many individuals with BD. Essentials of the approach encompass traditional concepts of CBT, including developing awareness of the

connection between thoughts, feelings, and behavior. Furthermore, the clients act as ‘experimenters’ responsible for observing their experiences with the goal of identifying the impact of thoughts on mood and behavior.

Sachs and colleagues (STEP-BD study; 2003) have initiated a large-scale multi-site investigation of these three approaches (i.e., FFT, IPSRT, CBT) to the treatment of BD. The goal of this ongoing study is to define various *menus of reasonable treatment* that would provide clinicians with validated guidelines for work with individuals affected by BD. Extensive control of identified *standard care pathways* and broad inclusion criteria allowed the researchers to address the issue of determining the best treatment approach for varying stages of BD. To date, the three aforementioned approaches to the treatment of BD are thought to represent efficacious interventions for BD, primarily for the treatment of bipolar depression. The STEP-BD study represents a large-scale and ongoing investigation that will compare these psychotherapeutic interventions, and will provide empirical clarification for a variety of questions regarding best practices for the treatment of individuals with BD (e.g., Do the approaches differ?, Which approach is best during a particular phase of illness?, Is there a difference between combination treatments?).

Summary of Psychological Treatments of BD

Attempts to explain and improve mood dysregulation patterns observed in BD have included a variety of perspectives. Miklowitz and Goldstein’s (1997) model of family therapy for BD (i.e., Family Focused Therapy, FFT), modeled after Falloon, Boyd, and McGill’s (1984) behavioral treatment of schizophrenia, has received empirical support in the literature (e.g., Miklowitz et al., 2000; Weisman, Tompson, Okazaki,

Gregory, Goldstein, & Miklowitz, 2002). IPSRT has also received preliminary support (Frank, 1999, Frank et al., 2000).

In addition to the IPSRT and FFT frameworks, Otto and colleagues (2003) have extended Cognitive Behavioral Therapy (CBT) to the treatment of Bipolar Disorder. The CBT approach has also received preliminary support (e.g., Lam et al., 2003; Otto, Reilly-Harrington, & Sachs, 2003; Patellis-Siotis et al., 2001), particularly relative to the reduction of mood episode recurrence (Hirschfeld et al., 1998; Lam et al., 2000), increases in medication adherence and decreases in hospitalization rates (Cochran, 1984), and increases in euthymic periods (Hirschfeld et al., 1998). Although these three models represent an advancement of the state of the psychological literature regarding the treatment of BD, evaluations of the utility of these interventions have not targeted the issue of stress reactivity and adaptation responses that may contribute substantially to an individual's risk of relapse.

Methodological Issues

Single-Participant Time-Series Research Designs

During the design phase of the current experiment, attention was given to a variety of methodological considerations. The issues of using a single-case versus a group design and the appropriateness of each approach relative to the empirical questions being addressed in the current study were among those considerations. A vast literature exists with a focus of comparing the usefulness of single-participant and group research designs (e.g., Barlow & Hersen, 1987; Kazdin, 1982; Lundervold & Bellwood, 2000; Morgan & Morgan, 2001). Although group methodologies allow for comparative testing of competing treatments, single-case designs produce detailed information about

mechanisms of change that may be responsible for advancements during the course of treatment. Single-participant designs have an extensive history in the behavioral sciences, particularly as a means of investigating psychological phenomena in preliminary research investigations (Morgan & Morgan, 2001).

Historical researchers (Ebbinghaus, Skinner) made extensive use of single-participant designs, and demonstrated a consistent ability to replicate results from such methodologies (Morgan & Morgan, 2003). Kazdin (1982) has argued that preliminary research of treatment effectiveness should utilize single-participant designs, with a primary focus on the process information that is produced during such investigations. According to Kazdin, large group designs should only be implemented after mechanisms of change have been identified to explain the effectiveness of a particular treatment. Generally, the argument holds that large group designs should be utilized to investigate overall efficacy of a particular treatment as compared to another established treatment (Kazdin, 1982).

Given the lack of clarity regarding mechanisms of change responsible for the effect of exercise on mood, the use of single-participant research is warranted. The current investigation will utilize a single-participant design to examine processes that change over the course of traditional (standard) Behavioral Activation (SBA) treatment approaches and Behavioral Activation that focuses on the implementation of an exercise prescription (EP) for participants. The following overview discusses characteristics of single-participant research methods, advantages and disadvantages of those methods, and issues about the scientific rigor of single-case research designs.

Single-participant research designs may be considered as a series of ‘mini-experiments’ that are directly relevant to clinical practice (Campbell, 1992). The idiographic nature of single-case designs allow for flexibility in designing treatments that are most appropriate according to an individual’s unique characteristics (e.g., behavioral manifestations, maintaining environmental variables). The structure of single-participant designs can be compared to the framework of (Hawkins, 1986) assessment funnel. A broad range of behaviors that are potential targets for treatment are assessed, followed by a narrowing of focus to specific target problems/behaviors and selection of the most appropriate treatment. A hallmark feature of single-participant designs is the inclusion of repeated and continuous assessment, which allows for strategic monitoring of treatment effectiveness, treatment outcome, and the effect of intermediate goal achievement (Hayes, 1981; Hayes, Barlow, Nelson-Gray, 1999). The primary goal is to examine changes in behavior across time, while drawing supported inferences regarding functional relationships through intra- and inter-participant replications (Morgan & Morgan, 2001).

Single-case designs do not employ traditional null hypothesis testing commonly utilized in large group methods. However, they are rooted in sound scientific method, and can produce rigorous and clinically meaningful results (e.g., Barlow, Hayes, & Nelson, 1984; Hayes, Barlow, Nelson-Gray, 1999; Morgan & Morgan, 2001). A variety of methods are available for the evaluation of single-participant data, which allow for the inference of causal relationships. Most commonly, visual inspection of graphical data is employed, in which individual data points are plotted across phases in order to evaluate the presence of reliable behavior change (Baer, 1977; Gaynor, Baird, & Nelson-Gray, 1999; Michael, 1974). Due to a serial dependency in the data produced by the frequency

of measures taken, traditional parametric analyses cannot be used with time-series investigations (Gaynor et al., 1999). Overall, the use of time-series methodology allows for the recognition of reliable small-scale changes in behavior that risk oversight during visual inspection of data (Kazdin, 1984).

Single-participant research is arguably the most effective method of tracking the effectiveness of empirically supported treatments (ESTs) for individuals with a particular set of problem behaviors that are maintained by a given set of environmental, psychological, and physiological variables (Morgan & Morgan, 2001; Sanchez & Turner, 2003). In addition, the repeated and continuous assessment that is central in single-participant designs improves scientific rigor of time-series investigations (Barlow et al., 1984; Gaynor et al., 1999; Hayes et al., 1999). Repeated measurement of a dependent variable is crucial in evaluating variability across baseline and intervention phases, and is necessary in the identification of trends relative to the behavior of interest. The inclusion of repeated measurement in anecdotal case study is argued to be sufficient for upgrading to the classification of a quasi-experimental research design (Browning & Stover, 1971). Time-series assessment also provides a means for separating treatment effect from measurement error (Barlow et al., 1984; Hayes et al., 1999).

Single-participant research designs also address ethical issues regarding withholding treatment commonly seen in large group designs that implement treatment versus no-treatment comparisons. An ethical dilemma arises when an individual is placed into a 'no-treatment' control group; although this type of comparison group is necessary to demonstrate treatment effectiveness in large group designs (Barlow, Blanchard, Hayes, & Epstein, 1977; Barlow & Hersen, 1987). Single-participant designs

allow for the examination of competing interventions at the individual level, in which behaviors are evaluated between phases (e.g., baseline, treatment). Therefore, the withholding of treatment is limited to a minimal amount of time while baseline data is collected. Hayes and colleagues (1999) argued that the comparison of an individual's behavior between baseline and intervention phases is more clinically meaningful than traditional comparisons.

The most commonly noted criticism of single-participant research designs refers to the external validity of results. In particular, the relevance of responses from one client in the treatment of other individuals is questioned, along with the reliability of findings from such methodologies (e.g., Barlow et al., 1977). Although these points are valuable considerations, large group designs utilizing between groups comparisons do not eliminate this difficulty. External validity of large group designs is limited by various factors, including method of participant recruitment and compensation, and individual characteristics that make an individual more likely to participate in research. In addition, difficulties generalizing findings to an individual client are present in large group comparison designs (Barlow et al., 1977; Hersen & Barlow, 1976).

The current investigation utilized a combined series crossover single-participant methodology (i.e., A/B/A/C/A versus A/C/A/B/A design) in combination with an interaction design (i.e., A/B/A/B/A). The crossover design involved the measurement of baseline responses, prior to the introduction of two concurrent phase changes. Phase changes in the interaction design occurred at the same phase of study participation, although the treatment was not altered during the second phase. Following baseline assessment, an intervention was conducted, and measures of the same dependent variable

were made. Response variations subsequent to treatment may then be compared to baseline behavioral manifestations (Gaynor et al., 1999). The design corrected for the methodological weaknesses of a simple phase change design, in which responses are measured during a single phase of treatment. In such a design, there is no protection against extraneous events that may result in behavior change. This lack of protection renders the researcher unable to confidently state that changes in behavior are due to the intervention being implemented in the study.

The use of a crossover design added the ability to assess the interaction of different components of an intervention and to determine whether two treatments were effective (Gaynor et al., 1999; Hayes et al., 1999; Hecker, Fink, Vogeltanz, Thorpe, & Sigmon, 1998). In crossover designs, conditions are synchronized so two phase changes occur concurrently, with order of presentation reversed for half of the participants. This allows for coordinated sets of comparisons to be made between, as well as within, a series of measurements (i.e., combined series design).

Participants in the current study received one of four possible treatment orders. In one condition, participants received a prescription of an exercise regimen followed by a prescription of behavioral activation procedures (e.g., scheduling of non-exercise activities). Individuals in a second condition received the same interventions, in reverse order (i.e., standard behavioral activation followed by prescribed exercise regimen). In the two remaining conditions, individuals received either an exercise prescription only, or the standard behavioral activation prescription only, for the entire 4-weeks of treatment. Crossover designs allow for control of external factors through phase change after standard exposure to a given treatment (e.g., number of days). Therefore, effects

demonstrated within each condition are more convincing than when unsynchronized phase changes are utilized.

A primary criticism of crossover research designs involves the presence of order effects in this type of methodology. Specifically, the implementation of one treatment followed by another results in a degree of uncertainty related to treatment effects. It becomes unclear whether effects demonstrated during the second phase of treatment are due to the second intervention, delayed effects of the initial intervention, or the effects of combined treatment. Although this is a valid criticism to be considered when utilizing crossover designs, findings from single-participant research designs provide important information relative to the effectiveness of the first-order and combined treatment effects. Two additional treatment orders were included in the current investigation (i.e., EP or SBA only) so that a more clear effect of exercise on stress reactivity and mood lability could be assessed, in the absence of questions regarding combined treatment effects.

Statement of Purpose

The current study addressed the utility of including exercise (as a form of behavioral activation) in treatment protocols for individuals with Bipolar Disorder (BD). Previous research suggests that regular exercise may alleviate depressed mood in individuals with unipolar depression (Gauvin et al., 1996; Martinsen, 1987, 1990; Salmon, 2001; Steptoe et al., 1998; Yeung, 1996), but the possibility that exercise can regulate mood fluctuation in BD has not been investigated. Stressful events are thought to place an individual with BD at increased risk for relapse because of the resulting disruption in daily routines and social rhythms (Frank et al., 2000; Miklowitz & Goldstein, 1997). Regular exercise may provide a more structured life system for

individuals (Salmon, 2001), resulting in less vulnerability to stress (Cramer et al., 1991; Crews & Landers, 1987; Salmon, 2001). Regular exercise has also been linked to a decrease in perceived stress in daily living (Steptoe, Lipsey, & Wardle, 1998) and may provide a more adaptive method of coping with the disorder and its associated symptoms, buffering the impact of future life stressors.

Much of the psychological literature investigating the development and maintenance of BD has reflected the assumption that stressful life events, greater perceived stress, and a higher level of reactivity to such events precipitate mood fluctuation over the course of illness (e.g., Ehlers et al., 1993; Johnson & Meyer, 2004). Though regular exercise is thought to provide a mechanism for stress adaptation for individuals with MDD and non-depressed controls (e.g., Salmon, 2001), the role of exercise in regulating stress reactivity in individuals with BD has not been investigated prior to the current study. In the current investigation, the role of exercise (i.e., walking) and standard BA (i.e., non-exercise-related activity) was examined relative to their effects on stress reactivity and mood in individuals with BD.

There are several suggested hypotheses for this improvement in mental health related to maintenance of a standard exercise regimen involving biochemical, physiological, and psychosocial mechanisms, although specific mechanisms and the conditions under which they operate have not yet been determined (Camacho et al., 1991; Fox, 1999). The current methodology allowed for evaluation of a stress adaptation hypothesis relative to exercise (specifically) and behavioral activation (generally), within a BD sample. The question of whether exercise and standard behavioral activation will

reduce levels of stress reactivity in individuals with BD was addressed, and the two interventions were evaluated for differential effectiveness within subjects.

A structured routine of physical exercise may act to regulate mood for a variety of reasons, including decreased stress reactivity, improvements in routine regulation, increased consistency of biological rhythms, and increased self-efficacy and social functioning. Traditional non-exercise-specific forms of BA have received consistent support for alleviating symptoms of mood dysregulation (e.g., depression). Current findings address the utility of including exercise-focused BA in the standard recommendations for treatment of individuals with BD.

Research Hypotheses

1. It was expected that individuals would exhibit lower reactivity (i.e., skin conductance responses, depressed mood ratings) to laboratory stress tasks following completion of the Exercise Prescription intervention compared to the Standard BA intervention. For individuals receiving the crossover treatment protocol the largest within-subject reduction in stress response was expected to occur following Exercise Prescription as a second-order intervention due to combined treatment effects. It was expected that individuals receiving the EP only intervention would demonstrate the largest decrease in physiological reactivity to the laboratory stress task compared to response in crossover or SBA only treatments. In addition, it was expected that participants would report a lower level of depressed mood and anxiety (i.e., depressed and anxious subscale scores on POMS) following completion of the Exercise Prescription compared to Standard BA, regardless of treatment order. Depressed mood and anxiety

responses were expected to decrease most significantly for individuals completing the Exercise Prescription only intervention.

2. It was predicted that participants would report a lower level of perceived daily stress (via daily stress ratings) during completion of the Exercise Prescription compared to Standard BA, regardless of treatment order. For individuals receiving the EP only intervention, it was expected that reported daily stress would be less during the final two weeks of treatment, due to habituation effects (Salmon, 2001).
3. Given consistent evidence that exercise is effective in alleviating depressed mood, it was expected that individuals would endorse fewer depressive symptoms (e.g., weekly BDI-II scores) following the Exercise Prescription intervention than Standard BA. It was predicted that individuals completing the EP only intervention would report the largest improvement in BDI-II score, and that this improvement would be maintained over time with continued exercise participation. It was expected that all participants would demonstrate the largest improvement in BDI-II score following completion of the 4-week treatment program, regardless of treatment order, due to combined treatment effects.
4. Regarding symptoms of mania, it was predicted that individuals endorsing manic symptoms at baseline (i.e., Altman Self-Rating Mania scores) would report fewer symptoms after completing four weeks of treatment, due to combined treatment effects. In addition, it was predicted that individuals receiving the EP as the first-order treatment of a crossover protocol would endorse a slight elevation in manic symptom reporting due to a defense reaction (Folkow, 1993) to a new stressor.

This reaction was expected to decrease over time due to habituation effects (Salmon, 2001). Finally, it was predicted that individuals receiving EP as a first-order, or sole, treatment approach would exhibit the greatest level of decline and overall stability related to manic symptom reporting at the end of the study due to habituation effects.

5. It was expected that individuals receiving the EP treatment would report the largest decreases in the use of maladaptive coping strategies on the Brief COPE. It was expected that individuals participating in the EP only intervention would report the greatest decrease in the use of maladaptive coping strategies, and related increase in use of adaptive strategies, following study completion. With maintained exercise participation at follow-up, use of adaptive and maladaptive strategies was expected to remain stable.
6. It was predicted that individuals participating in the EP only intervention would demonstrate the greatest improvement in perceived experiences of daily hassles, as measured by the Survey of Recent Life Events (SRLE), due to habituation effects of exercise. In addition, it was expected that individuals participating in the 4-week exercise intervention would maintain exercise participation, and, therefore maintain their report of less stressful experiences, compared to individuals in alternative treatment orders.

CHAPTER 2

METHOD

Participants and Recruitment

Adult volunteers (18 years of age or older; male and female) with Bipolar Disorder (BD) were recruited from the community via flyers (see Appendix C) posted in public settings and health care offices around the area. Flyers indicated that a psychology graduate student was conducting a research project investigating the relationship between activity, stress, and mood, and was seeking individuals who felt that they experienced mood fluctuations consistent with Bipolar Disorder. Individuals who responded to advertisements were contacted via telephone and asked to respond to questions during a brief screening (see Appendix A) to determine whether a more thorough interview was warranted.

All individuals who appeared to meet research criteria (e.g., engaged in minimal levels of exercise, appeared to have experienced a manic episode or hypomanic episode plus major depressive episode in the past) were invited to complete an interview session, during which the Structured Clinical Interview for DSM-IV Axis I Disorders – Clinical Version (SCID-CV; First, Spitzer, Gibbon, & Williams, 1995) was administered.

Twelve participants were recruited (males and females age 18 or older) who met diagnostic criteria for Bipolar Disorder (Type I or II), and were matched on age, level of education, gender, and exercise level (as defined below). Community volunteers were compensated \$10 for the initial interview and completion of questionnaires, \$43 for daily self-monitoring (43 total days, \$1 per day), and \$5 for each of the two additional experimental sessions (\$5 for each session completed).

Each participant was asked to read and sign an informed consent document (see Appendix B), and was debriefed at the end of the third experimental session. Participants were asked to complete three follow-up sessions after conclusion of the final intervention phase, occurring at 1-, 3-, and 6-months following the final laboratory visit. All participants (community and student volunteers) who completed follow-up procedures were compensated \$5 for responding to follow-up assessment questionnaires (a total of \$15 possible).

Inclusion and Exclusion Criteria

Eight participants, who met DSM-IV-TR (APA, 2000) criteria for a Bipolar Disorder (Type I or II) according to the Structured Clinical Interview for DSM-IV Axis I Disorders – Clinical Version (SCID-CV; First, Spitzer, Gibbon, & Williams, 1995), were enrolled in the study. Individuals who endorsed a history of manic, hypomanic, or mixed episodes were eligible for study participation. For those participants who endorsed symptoms consistent with diagnostic criteria for BD-II (e.g., hypomanic episodes), a history of one or more major depressive episodes (MDE) was present. All individuals who met criteria for a BD-I diagnosis had also experienced at least one MDE.

Exclusion criteria for the study included presence of a thought disorder, psychotic disorder, and/or active alcohol or substance dependence. In addition, respondents who indicated a pattern of regular exercise (i.e., 3 or more sessions of structured exercise per week that lasted 30 minutes or longer) were not enrolled in the study. This criterion was based on reports in the current literature that consistently suggest that individuals suffering from chronic forms of mental illness such as BD are minimally active (e.g., Richardson et al., 2005). Four prospective participants were excluded from study

participation based on the aforementioned criteria. One participant was excluded who met criteria for an active psychotic disorder. Two participants were excluded who reported participating in a regular (i.e., three or more days per week) program of exercise. A final participant was excluded from the study who reported a pre-existing cardiac condition (as indicated on the PAR-Q) and was unable to provide documentation from a physician that the prescribed program of exercise included in the current study was safe to initiate.

Experimenters

The principal investigator conducted the majority of research procedures (e.g., screening, interviewing, data collection). An advanced graduate student in clinical psychology assisted in conducting brief telephone screenings. Another advanced graduate student assisted with data collection for some participants.

Screening Measures

Structured Clinical Interview for DSM-IV Axis I Disorders – Clinician Version

To establish a diagnosis of BD (Type I or II) and to assess for the presence of other Axis I disorders, the Structured Clinical Interview for DSM-IV Axis I Disorders – Clinician Version (SCID-CV; First, Spitzer, Gibbon, & Williams, 1995) was administered to each individual who agreed to participate after meeting pre-screening criteria. Reliability and validity data from a previous version of the SCID-CV (SCID for DSM-III-R) demonstrated high kappa values (i.e., .70 – 1.00) when videotaped interviews were used (e.g., Segal et al., 1993, 1995; Strakowski, Stoll, Tohen, Faedda, & Goodwin, 1993). Interrater reliability (using kappa coefficients) for the SCID-I based on

84 pairs of raters using videotaped interviews revealed kappa coefficients ranging from .57 – 1.00 for all Axis I disorders.

The Mood Disorders Questionnaire (MDQ)

The Mood Disorders Questionnaire (MDQ; Hirschfeld et al., 2000; see Appendix D) was used to assess for a lifetime history of manic or hypomanic symptoms. The MDQ consists of 13 yes/no items (e.g., “Has there ever been a period of time when you were not your usual self and you were so irritable that you shouted at people or started fights or arguments?”; “... You had much more energy than usual?”) that correspond with DSM-IV criteria and clinical experience (Hirschfeld et al., 2000). If an individual indicated that more than one symptom of elevated mood had occurred in his or her lifetime, he or she is asked to indicate whether these symptoms have ever occurred simultaneously. Finally, individuals were asked to indicate how problematic the occurrence of manic/hypomanic symptoms was for them (e.g., “no problem, minor problem, moderate problem, serious problem”).

In a sample of 198 outpatients being treated primarily for mood disorders, the MDQ yielded good sensitivity (.73) and very good specificity (.90) when individuals endorsed a screening score of 7 or more items (Hirschfeld et al., 2000). An interviewer blind to diagnostic category administered the bipolar module of the SCID for DSM-III-R (Spitzer, Williams, Gibbon, & First, 1992) to each participant in order to establish the presence of a bipolar spectrum disorder (i.e., BD-I, BD-II, Cyclothymia, or BD-NOS). Information from this module was then used as the criterion standard for comparison of diagnostic impressions. The MDQ is considered to be a useful screening instrument for

bipolar spectrum disorder in an outpatient population (e.g., Mulrow, Williams, Gerety, Ramirez, Montiel, & Kerber, 1995).

Physical Activity Readiness Questionnaire (PAR-Q)

The Physical Activity Readiness Questionnaire (PAR-Q; Thomas, Reading, & Shephard, 1992; Appendix E) is a 7-item (yes/no format) questionnaire that was used to assess participants' readiness to begin an exercise program. Individuals who answered "yes" to one or more items on the measure were considered to be at risk of physical harm related to initiation of an exercise program, and consultation with a physician was recommended prior to enrollment in the study. In the current investigation, the measure was used to screen for potential immediate physical risk factors that may have placed an individual at risk for physical harm. Two participants responded in the affirmative to item number one on this measure. One participant indicated that she was diagnosed with hypertension, and produced written documentation that her primary care physician approved the level of exercise prescribed as a treatment in the current study. This individual was enrolled in the study. A second participant reported the presence of a cardiac condition on this measure, and was unable to provide documentation from a physician that the prescribed exercise program was safe to initiate and was, therefore, excluded from study participation.

Dependent Measures

Altman Self-Rating Mania Scale (ASRM)

The Altman Self-Rating Mania Scale (ASRM; Altman, Hedeker, Peterson, & Davis, 1997; Appendix F), designed as a brief self-rating scale compatible with DSM-IV criteria (APA, 1994), was used to assess the presence and severity of manic symptoms

over the previous seven days. Originally, the measure constituted eleven groups of 5 statements representing major symptoms of mania (e.g., elevated mood, increased self-esteem, decreased need for sleep, pressured speech, psychomotor agitation). Analyses revealed three subscales for the measure: mania, psychosis, and irritability. Subscale 1 (mania subscale) represents the final version of the ASRM, resulting in 5 groups of statements. Respondents were asked to endorse only one statement in a group, rated in increasing severity from 0 (symptom not present) to 4 (symptom is present and severe) to describe their mood or behavior over the past seven days.

The ASRM is considered to be a reliable and valid scale for assessing self-reported mania symptom presence and severity. It has demonstrated good test-retest reliability on a sample of depressed and manic inpatients (i.e., $r = .86$; $p < .001$ for Mania Subscale). The measure also assesses symptom severity of manic symptoms in individuals currently experiencing mania, and is sensitive to change and improvement following treatment. Subscale scores (derived by taking the sum of rated items) exceeding 5 demonstrates 85.5% sensitivity and 87.3% specificity. The ASRM was used to assess manic symptom experience during intervention phases of the current investigation (i.e., days 7 and 14 of each intervention phase). In addition, the ASRM was used to assess manic symptom experience during the 1-, 3-, and 6-month follow-up assessments.

Beck Depression Inventory-II (BDI-II)

The Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996; see Appendix G) is a 21-item self-report inventory designed to assess cognitive, affective, and somatic symptoms commonly reported in depression. Respondents were asked to

indicate the severity of their depressive symptoms on a 0 (neutral severity) to 3 (maximum severity) scale. The BDI-II has demonstrated adequate internal consistency, short-term test-retest reliability, and convergent validity (Beck et al., 1996). The BDI-II is often used in mood disorders research and may be useful to assess the common depressive symptoms experienced by individuals with a BD diagnosis. The BDI-II was used to assess depressive symptom experience during intervention phases of the current investigation (i.e., days 7 and 14 of each intervention phase). In addition, the BDI-II was used to assess depressive symptom experience during the 1-, 3-, and 6-month follow-up assessments.

Survey of Recent Life Events (SRLE)

The Survey of Recent Life Events (SRLE; Kohn & Macdonald, 1992a; See Appendix H) is a 51-item measure formulated to assess exposure to a variety of daily hassles. This measure was developed as an alternative to earlier measures (e.g., Daily Hassles Scale (DHS); Kanner, Coyne, Schaefer, & Lazarus, 1981), which were criticized for being contaminated by items, and a response format, that were thought to reflect subjective distress, rather than predict it (Dohrenwend, Dohrenwend, Dodson, & Shrout, 1984; Dohrenwend & Shrout, 1985; Green, 1986; Kohn & Macdonald, 1992a). The SRLE utilizes a Likert scale format for respondents to indicate the extent to which an item was part of his or her life during the past month (*1 = not at all, 4 = very much*). This response format provides an alternative for individuals who have not recently experienced a particular item as distressing, unlike earlier measures. The sum of responses is calculated, with higher scores indicating a greater experience of daily hassles over the past month.

The SRLE is composed of 6 subscales: 1) Social and cultural difficulties ($\alpha = .78$; e.g., *“Being let down or disappointed by friends”*); 2) Work ($\alpha = .82$; e.g., *“Conflict with supervisor(s) at work”*); 3) Time pressure ($\alpha = .81$; e.g., *“Too many things to do at once”*); 4) Finances ($\alpha = .76$ e.g., *“Financial conflicts with family members”*); 5) Social acceptability ($\alpha = .68$; e.g., *“Being ignored”*); and 6) Social victimization ($\alpha = .76$; e.g., *“Being taken advantage of”*). The SRLE has been found to demonstrate high internal consistency ($\alpha = .91$). In addition, the measure has been shown to correlate significantly with perceived stress, trait anxiety, psychiatric symptomatology, and minor physical ailments (e.g., Kohn, Gurevich, Pickering, & Macdonald, 1994; Kohn & Macdonald, 1992b). In the current investigation, the SRLE was used to assess participants’ experience of daily hassles at each laboratory session, and at follow-up assessments (i.e., 1-, 3-, and 6-months following second-order intervention phase).

Brief COPE

The Brief COPE is a 28-item inventory designed to identify adaptive and problematic coping reactions (Carver, 1997; see Appendix I). Adaptive responses are defined by Carver (1997) as those that, when used by individuals, are found to be more predictive of lower distress and fewer physiological endpoints (e.g., slower disease progression in studies of HIV). Conversely, problematic responses are those more likely to predict higher rates of distress and greater presence of undesirable physiological effects. This measure represents an abbreviated version of the full COPE (Carver, Scheier, & Weintraub, 1989), a 60-item measure with 15 subscales. The Brief COPE is composed of 14 subscales, each with two items: 1) Active coping ($\alpha = .68$; *“I’ve been concentrating my efforts on doing something about the situation I’m in.”*; *“I’ve been*

taking action to try to make the situation better.”); 2) Planning ($\alpha = .73$; “I’ve been trying to come up with a strategy about what to do.”; “I’ve been thinking hard about what steps to take.”); 3) Positive reframing ($\alpha = .64$; “I’ve been trying to see it in a different light, to make it seem more positive.”; “I’ve been looking for something good in what is happening.”); 4) Acceptance ($\alpha = .57$; “I’ve been accepting the reality of the fact that it has happened.”; “I’ve been learning to live with it.”); 5) Humor ($\alpha = .73$; “I’ve been making jokes about it.”; “I’ve been making fun of the situation.”); 6) Religion ($\alpha = .82$; “I’ve been trying to find comfort in my religion or spiritual beliefs.”; “I’ve been praying or meditating.”); 7) Using emotional support ($\alpha = .71$; “I’ve been getting emotional support from others.”; “I’ve been getting comfort and understanding from someone.”); 8) Using instrumental support ($\alpha = .64$; “I’ve been trying to get advice or help from other people about what to do.”; “I’ve been getting help and advice from other people.”); 9) Self-distraction ($\alpha = .71$; “I’ve been turning to work or other activities to take my mind off things.”; “I’ve been doing something to think about it less, such as going to movies, watching TV, reading daydreaming, sleeping, or shopping.”); 10) Denial ($\alpha = .54$; “I’ve been saying to myself ‘this isn’t real’.”; “I’ve been refusing to believe this has happened.”) 11) Venting ($\alpha = .50$; “I’ve been saying things to let my unpleasant feelings escape.”; “I’ve been expressing my negative feelings.”); 12) Substance use ($\alpha = .90$; “I’ve been using alcohol or other drugs to make myself feel better.”; “I’ve been using alcohol or other drugs to help me get through it.”); 13) Behavioral disengagement ($\alpha = .65$; “I’ve been giving up trying to deal with it.”; “I’ve been giving up the attempt to cope.”); 14) Self-blame ($\alpha = .69$; “I’ve been criticizing myself.”; “I’ve been blaming myself for things that happened.”).

Similar to the full COPE, the Brief COPE has demonstrated acceptable internal reliability, with all subscales exhibiting alpha values ranging from .50 to .90. Moreover, exploratory factor analyses yielded a factor structure consistent with the full COPE. Participants completed this measure during each laboratory assessment visit and at each follow-up assessment (i.e., 1-, 3-, and 6-months following second-order intervention phase). Carver (1997) identified the following subscales as adaptive: adaptive coping, planning, positive reframing, acceptance, humor, religion, using emotional support, using instrumental support, self-distraction, and venting. Conversely, the subscales of denial, substance use, behavioral disengagement, and self-blame) are representative of problematic coping strategies on the measure.

Profile of Mood States (POMS)

Prior to and following each experimental stress task, participants were asked to complete the depression and anxiety subscales of the Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1971; see Appendix J). The POMS is a self-report inventory that evaluates transitory mood states (e.g., lonely, guilty, discouraged). The POMS has been used widely in research, and has demonstrated good reliability and validity (McNair et al., 1971). The POMS has often been used in mood disorders research because it allows for specification of particular mood states that may be associated with precursors to mood episodes. Given its focus on current and transient mood states, the specific reliability and validity of the POMS in mood disorders research has not been investigated. In the current study, the POMS was used to assess transitory anxiety and depressed mood states prior to and following a laboratory stress task (i.e., backward counting task).

Self-Monitoring

In order to examine the relationship between daily exercise levels, mood, and perceived level of stress, prospective recording of physical activity patterns (i.e., duration, intensity, and type of exercise, pedometer reading) and average daily mood, stress, sleep length and quality were collected for the full 43 days of active study participation. Each participant received a self-monitoring packet that included: a) daily self-monitoring forms for 43 days of reporting (see Appendix K; participants record their mood and stress ratings, as well as daily exercise and physical activity including prescribed activities); b) four copies of the Altman Self-Rating Mania Scale (ASRM; Altman et al., 1997; see Appendix F; to be completed at days 7 and 14 of each intervention phase of recording); c) four copies of the Beck Depression Inventory – II (BDI-II; Beck et al., 1996; see Appendix G) to be completed at days 7 and 14 of each intervention phase of recording); and d) one NewLifestyles pedometer (model SW-200) in order to track daily step-count totals, which were reported on the self-monitoring form (Appendix K) by participants one to two hours before retiring to bed each evening. All participants were provided with instructions for how to complete daily monitoring activities, including information about how to use and wear the pedometer.

	Phase I Intervention					Phase II Intervention				
Lab 1	D 1-6	D 7	D 8-13	D 14	Lab 2	D 1-6	D 7	D 8-13	D 14	Lab 3
<i>All</i>	SM	BDI-II	SM	BDI-II	<i>All</i>	SM	BDI-II	SM	BDI-II	<i>All</i>
<i>Forms</i>		ASRM		ASRM	<i>Forms</i>		ASRM		ASRM	<i>Forms</i>
		SM		SM			SM		SM	

Table 1. Summary of questionnaires completed according to phase of study. *Note: Form abbreviations are as follows: Beck Depression Inventory (BDI-II); Altman Self-Rating Mania scale (ASRM); daily self-monitoring form (SM). Additional forms completed during laboratory visits included: Brief COPE, Mood Disorder Questionnaire (MDQ), and Survey of Recent Life Events (SRLE).*

In order to increase accuracy of recording and compliance with the self-monitoring process, a brief instructional period was conducted prior to self-monitoring. To enhance compliance, self-monitoring records were reviewed after completion of the form. Participants were provided with instructions for how to use and wear the pedometer. Participants were provided with business reply envelopes to return daily monitoring forms for each of the monitoring days. Participants were contacted at regular intervals to discuss upcoming phase changes (e.g., day before each intervention phase begins), in addition to the three laboratory visits attended.

Experimental Tasks

Stress Induction

To explore the impact that exercise may have on regulating reactivity to stressful situations (i.e., skin conductance response), individuals in the current study underwent an experimental stress task. During this task, each participant was asked to perform mental

arithmetic (i.e., serial 7 backward counting task). The serial 7 task has been shown to produce a stress response in laboratory settings, and has been successfully utilized in previous research (e.g., Choi & Salmon, 1995; Engebretson & Matthews, 1992; Kelsey, Soderlund, & Carlotta, 2004; Matthews, Manuck, Stoney, Rakaczky, McCann, Saab, et al., 1988). The procedure involves asking participants to count backward in increments of 7 (beginning at 500) for a 3-minute period. After 90 seconds elapsed, participants were informed that they had “90 seconds left.” The task was described as a test of math performance abilities, with emphasis on speed and accuracy. Participants were instructed to provide maximum effort during the task. After the 3-minute stress induction task was completed, the depression and anxiety subscales of the POMS were re-administered to assess transient changes in depressed mood and anxiety.

This type of stress task has been found to elicit stress responses (e.g., elevated heart rate, elevated blood pressure, elevated cardiovascular activity) in previous research (e.g., Choi & Salmon, 1995; Kelsey et al., 2004; Plante, Chizmar, & Owen, 1999; Sharpley & Gordon, 1999). Skin conductance measures were collected throughout experimental procedures to make comparisons between baseline and induction periods and between laboratory testing occasions prior to and following both intervention phases of the experimental procedures.

Psychophysiological Recording

For the duration of the experimental task (i.e., serial 7 stress tasks), skin conductance recordings were obtained. Physiological assessment has been utilized in previous studies with psychiatric samples (e.g., Cramer, 2003; Plante et al., 1999), including mood disordered samples. In the current investigation, skin conductance levels

were assessed using Contact Precision Instruments psychophysiological equipment (London, UK) with PSYLAB software (Dow, 2005). Consistent with previously established methods of monitoring skin conductance, two Ag-AgCl electrodes filled with electrode paste were attached to the medial phalanxes of the two middle fingers of the participant's non-dominant hand (Fowles et al., 1981). The skin conductance transducer supplied a constant voltage (0.6V) throughout the experimental procedures.

Participants sat in a quiet, sound attenuated, windowless laboratory room during a 2-minute adaptation and a 5-minute baseline recording period. Average baseline skin conductance levels were recorded for each participant. Then, skin conductance responses (SCR) were recorded during the experimental stress task. Significant SCR represent changes of .05 microsiemens or greater from baseline skin conductance levels (SCL). The total number of significant skin conductance responses that occurred during the 3-minute stress induction task was calculated.

Interventions

Exercise Prescription (EP)

During the EP intervention, participants were asked to engage in a program of exercise composed of 30-minute walking sessions, prescribed to occur on 4 separate days during a single week. A total of 8 exercise sessions (i.e., 4 sessions of 30 minute walking bouts each week) were scheduled with the participant. The exercise bouts were expected to occur during the 2-week EP phase of the experiment, and were scheduled to be spaced evenly throughout each of the two weeks (e.g., Monday, Wednesday, Friday, and Saturday of each week). The EP phase of the experiment was followed by 5-days of baseline recording, when no activities were prescribed to occur.

Standard Behavioral Activation (SBA)

Similar to the EP phase, individuals receiving this treatment were asked to perform a prescribed activity (i.e., 4 days of performing 30-minute sessions of a chosen activity other than exercise) during the 14-days of the SBA phase of intervention. A sedentary activity to be completed during this phase of intervention was agreed upon by the participant and experimenter prior to initiation of this treatment phase. Activities during the SBA phase were allowed to vary between participants, but a single participant was asked to perform a consistent activity during the SBA intervention phase. Participants were instructed to choose an activity that did not include a standard regimen of exercise so that treatment effects could be differentiated more clearly. The experimenter worked with each participant during the laboratory visit preceding onset of this intervention phase to determine what activity he or she would be prescribed. Again, 5 days of baseline monitoring followed the SBA intervention phase.

Procedure

Following recruitment efforts outlined above, individuals who met screening criteria were invited to attend the first laboratory session. During this meeting, individuals read and signed an informed consent document prior to the administration of the SCID-CV interview. Those meeting exclusionary criteria following SCID-CV and screening procedures were debriefed, thanked for their participation, and compensated for their time. Those individuals who did not endorse current substance dependence or thought disorder, and who endorsed a history of Manic Episode(s), or Hypomanic Episode(s) and past or current Major Depressive Episode(s), were asked to participate in the remainder of the initial session and follow-up procedures.

Individuals were asked to report their current weight and height so that a Body Mass Index (BMI) could be calculated. Following these procedures, participants completed the experimental stress tasks, and a self-report of depressed mood and anxiety (i.e., POMS measure). These individuals were asked to sit in a sound attenuated, windowless laboratory room, and were provided with an overview of upcoming experimental procedures. Electrodes for psychophysiological monitoring were attached to the medial phalanxes of the two middle fingers of the participant's non-dominant hand and baseline skin conductance was recorded. Additionally, baseline mood and anxiety ratings were obtained. Next, participants completed the experimental stress induction task (i.e., serial 7 task). Following stress tasks, participants provided a second self-rating of depressed mood and anxiety (second POMS administration). After the participant completed the final POMS scale, he or she was disconnected from the physiological recording equipment and asked to complete a questionnaire packet. After questionnaires were completed, participants were instructed about on how to complete daily self-monitoring activities. In addition, participants were informed of expectations related to completion of the appropriate behavioral activation procedures (i.e., EP or SBA protocol).

After completing the laboratory stress task, during which psychophysiological measurements were taken, individuals were asked to complete self-report and self-monitoring data so that the relation between exercise, mood, and stress (e.g., stress reactivity and perception of stress) could be assessed. This consisted of a series of self-report questionnaires, including the Beck Depression Inventory-II (BDI-II), Brief COPE, the Altman Self-Report Scale of Mania (ASRM), the Mood Disorder Questionnaire

(MDQ), and the Survey of Recent Life Events (SRLE). Further, all participants were instructed to record the number of steps taken per day (e.g., with a pedometer provided by the experimenter), average daily mood (i.e., 1 = worst possible mood to 8 = best possible mood), average daily stress level (i.e., 1 = least possible stress to 8 = most possible stress), perceived exertion during any reported exercise, context of any reported exercise (e.g., activity, who is present, where you are), and outcome of engaging in any reported exercise (e.g., exercise makes me feel less stress, no benefit, etc.). If no exercise was performed on a given day, participants were asked to indicate the reason that no exercise was performed. Finally, participants were asked to report any medication taken on a given day, so that a reasonable level of monitoring relative to medication compliance can be conducted.

Self-monitoring was completed for a total of 43 days, including the day of the initial laboratory visit. Daily recording was estimated to take approximately two minutes per day, except for days 7 and 14 of the intervention phases of the experiment when participants completed two additional measures (i.e., BDI-II and ASRM). On days 7 and 14 of each intervention phase (a total of 4 days), self-monitoring procedures were expected to take approximately five to seven minutes to complete. Questionnaires were returned on a daily basis in postage-paid envelopes provided to each participant by the experimenter during the initial visit.

During the 43-day self-monitoring period, participants (who each initially reported minimal engagement in exercise activities) underwent a series of baseline recording days. Specifically five days of baseline recording occurred at three distinct periods: 1) prior to first order treatment, 2) between treatment phases, and 3) following

second order treatment phase of the experiment. This resulted in collection of 15 total baseline days of recording. Baseline recording involved completing the daily monitoring form described above, in the absence of any activity prescription.

Intervention phases (described above) of the experiment were classified as either Exercise Prescription (EP) or Standard Behavioral Activation (SBA), and each phase lasted 14-days. A total of eight sessions of activity were scheduled with the participant for each phase of the experiment. These activities (i.e., walking for EP phase, non-exercise activity during SBA phase) were expected to occur during the associated 2-week phase of the experiment. Each intervention phase of the experiment was followed by 5-days of baseline recording, when no activities were prescribed to occur.

At the end of each 2-week self-monitoring period, participants returned to the laboratory to complete stress tasks and questionnaires described above. During all laboratory visits (i.e., pre-intervention, post-intervention 1, post-intervention 2), participants completed the experimental stress task and self-report measures (i.e., BDI-II, ASRM, Brief COPE, SRLE) in counterbalanced order. At the end of the final session, participants were debriefed, thanked for their efforts, and compensated for self-monitoring and the final experimental session. Additionally, a list of treatment referral information was provided to all participants. Participants were compensated for their participation during the 43-day phase of the study at the third visit.

Follow-up assessments were conducted 1-, 3-, and 6-months following the final laboratory and baseline recording periods. During follow-up procedures, participants completed questionnaires assessing mood (i.e., BDI-II, ASRM), life stress (SRLE), and exercise and physical activity behaviors (i.e., report of weekly frequency, duration, and

type of activity). All participants (i.e., community and student volunteers) were compensated \$5 for each of the follow-up sessions completed (\$15 for completing all sessions).

CHAPTER 3

RESULTS

Demographics

Demographic information for all participants is presented in table and graph format (see Tables 2, 3, 4, and 5; see figures 27-34). Participants were primarily Caucasian females. One female participant identified as African American. One male participant completed the study (EP only intervention). Most participants were employed on either a full or part-time basis upon enrollment in the study. Seven of the eight enrolled participants met criteria for Bipolar Disorder, Type I. The eighth participant (who received the SBA only treatment) met criteria for Bipolar Disorder, Type II. Individuals in all treatment groups reported symptoms consistent with various comorbid Axis I psychiatric disorders, including Panic Disorder with Agoraphobia, history of alcohol or substance abuse or dependence, a history of eating disorder (e.g., Bulimia Nervosa), social anxiety disorder, generalized anxiety disorder, and specific phobias. In addition, many participants acknowledged psychotic features associated with mood episodes (see Table 3). With the exception of participant 8 (P8), who denied any current use of psychotropic medication, participants were stabilized on a psychotropic treatment regimen. Participant 4 (P4) was taking only an antidepressant agent upon enrollment, although this individual reported that a regimen of mood stabilizing medication (i.e., Geodon) had been introduced at 3-months follow-up (see Table 4).

Regarding exercise participation, all participants were leading a sedentary lifestyle prior to baseline. All participants reported initiating and maintaining a level of exercise consistent with that prescribed during the EP treatment protocol, regardless of

treatment condition (see Figures 23-26). Data collected for this study were visually analyzed according to published guidelines for single-participant research designs (Parsonson & Baer, 1978; Appendix L).

Exercise Prescription (EP) to Standard Behavioral Activation (SBA) Crossover					
	Age	Race	Gender	Marital Status	Employment Status
P1	26	African American	Female	Single	Employed Full-time
P3	35	Caucasian	Female	Single	Student and Employed Full-time
Standard Behavioral Activation (SBA) to Exercise Prescription (EP) Crossover					
	Age	Race	Gender	Marital Status	Employment Status
P2	32	Caucasian	Female	Married	Full-time Student and Mother
P4	26	Caucasian	Female	Single	Employed Full-time **Unemployed @ FU 2
Exercise Prescription (EP) Only Treatment					
	Age	Race	Gender	Marital Status	Employment Status
P5	55	Caucasian	Male	Divorced	Employed Full-time **Retired @ FU 1
P6	45	Caucasian	Female	Married	Employed Full-time
Standard Behavioral Activation (SBA) Only Treatment					
	Age	Race	Gender	Marital Status	Employment Status
P7	28	Caucasian	Female	Single	Ψ Disability
P8	52	Caucasian	Female	Married	Employed Full-time

Table 2. General demographic information for all participants.

Exercise Prescription (EP) to Standard Behavioral Activation (SBA) Crossover Treatment					
	Primary Dx	Psychotic Features?	Last Episode	Comorbid Dx	# Hosp.
P1	BD-I	Yes (with ME)	Mixed	Panic Disorder w/Agoraphobia	3
				Specific Phobia	
				h/o Bulimia Nervosa	
P3	BD-I	No	MDE	Panic Disorder w/Agoraphobia	5
				ETOH dependence, full remission	
Standard Behavioral Activation (SBA) to Exercise Prescription (EP) Crossover Treatment					
	Primary Dx	Psychotic Features?	Last Episode	Comorbid Dx	# Hosp.
P2	BD-I	Yes (with MDE)	MDE	PD w/A	3
				ETOH dependence, full remission	
P4	BD-I	Yes (with ME)	MDE	Bulimia Nervosa	0
				Panic Disorder w/Agoraphobia	
Exercise Prescription (EP) Only Treatment					
	Primary Dx	Psychotic Features?	Last Episode	Comorbid Dx	# Hosp.
P5	BD-I	Yes (with ME)	MDE	Substance Dependence, full remission	13
P6	BD-I	Yes (with ME)	MDE	GAD	5
Exercise Prescription (EP) Only Treatment					
	Primary Dx	Psychotic Features?	Last Episode	Comorbid Dx	# Hosp.
P7	BD-I	Yes (with MDE)	Hypomanic	Specific phobia (spiders)	2
				Panic Disorder with Agoraphobia	
				Polysubstance Dependence, early, partial remission	
P8	BD-II	No	MDE	Panic Disorder with Agoraphobia	1

Table 3. Diagnostic profiles for all participants.

Exercise Prescription (EP) to Standard Behavioral Activation (SBA) Crossover Treatment						
	Baseline	Phase I	Phase II	1-mo FU	3-mo FU	6-mo FU
P1	Depakote (1000 mg)	Depakote (1000 mg)	Depakote (1000 mg)	Depakote (1000 mg)	Depakote (1000 mg)	Depakote (1000 mg)
	Lexapro (10 mg)	Lexapro (10 mg)	Lexapro (10 mg)	Lexapro (10 mg)	Lexapro (10 mg)	Lexapro (10 mg)
	Lexapro (10 mg)	Lexapro (20 mg)	Lexapro (20 mg)	No data available	No data available	No data available
P3	Trileptyl (300 mg)	Trileptyl (300 mg)	Trileptyl (300 mg)			
	Geodon (40 mg)	Geodon (40 mg)	Geodon (60 mg)			
Standard Behavioral Activation (SBA) to Exercise Prescription (EP) Crossover Treatment						
	Baseline	Phase I	Phase II	1-mo FU	3-mo FU	6-mo FU
P2	Klonopin (1.5 mg)	Klonopin (1.5 mg)	Klonopin (1.5 mg)	Klonopin (1.5 mg)	No data available	No data available
	Seroquel (100 mg)	Seroquel (100 mg)	Seroquel (100 mg)	Seroquel (100 mg)		
			Wellbutrin (150 mg)	Wellbutrin (150 mg)		
P4				Lamictal (50 mg)		
	Carbatrol (600 mg)	Carbatrol (600 mg)	Carbatrol (600 mg)	Carbatrol (600 mg)	Carbatrol (600 mg)	Carbatrol (600 mg)
						Geodon

Table 4. Psychotropic medication profiles for crossover participants.

Exercise Prescription (EP) Only Treatment						
	Baseline	Phase I	Phase II	1-mo FU	3-mo FU	6-mo FU
P5	Lithium (1200 mg)	Lithium (1200 mg)	Lithium (1200 mg)	Lithium (1200 mg)	Lithium (1200 mg)	Lithium (1200 mg)
	Trazadone (25 mg)	Trazadone (25 mg)	Trazadone (25 mg)	Trazadone (25 mg)	Trazadone (25 mg)	Trazadone (25 mg)
P6	Lithium (1500 mg)	Lithium (1500 mg)	Lithium (1500 mg)	Lithium (1500 mg)	Lithium (1500 mg)	Lithium (1500 mg)
Standard Behavioral Activation (SBA) Only Treatment						
	Baseline	Phase I	Phase II	1-mo FU	3-mo FU	6-mo FU
P7	Zyprexa (20 mg)	Zyprexa (20 mg)	Zyprexa (20 mg)	Zyprexa (20 mg)	No data available	No data available
	Wellbutrin (300 mg)	Wellbutrin (300 mg)	Wellbutrin (300 mg)	Wellbutrin (300 mg)		
	Xanax (2 mg)	Xanax (2 mg)	Xanax (2 mg)	Xanax (2 mg)		
	Methadone (150 mg)	Methadone (150 mg)	Methadone (150 mg)	Methadone (150 mg)		
P8	None	None	None	None	None	None

Table 5. Psychotropic medication profiles for interaction group participants.

Treatment Conditions

There were four possible treatment orders included in the study protocol. Participants 1 and 3 (P1 and P3) received the exercise prescription (EP) for the first treatment phase, and the standard behavioral activation treatment (SBA; i.e., reading for both participants) for the second phase of the study. A second treatment order (i.e., P2 and P4) involved completing the SBA prescription (i.e., sedentary gardening activities for P2, and reading for P4), with a subsequent crossover to complete the EP protocol during the second phase of the study. Participants 5 and 6 (P5 and P6) completed a third treatment protocol, receiving the EP for the duration of the study. Finally, two participants (P7 and P8) received the SBA prescription for the duration of the study, and chose reading as an activity.

Data are presented in graphical format, following participants over the course of intervention phases. Due to order effects, hypotheses regarding the effects of standard behavioral activation (SBA) or exercise prescription (EP) intervention protocols are discussed in terms of participants who received the respective treatment as a first-order intervention.

Hypothesis 1: Reactivity to Laboratory Stress Task

Reactivity to a laboratory stress task (i.e., 3-minute serial 7 backward counting task) was measured objectively (i.e., skin conductance recordings) during completion of the task, as well as by self-report of depressed mood and anxiety prior to and immediately following completion of the task (i.e., POMS data). Significant skin conductance responses (SCR) are presented in terms of raw frequency count that is provided by PSYLAB software. For participants completing crossover treatment protocols, it was

predicted that reactivity (i.e., skin conductance response frequency, depressed mood ratings) to the stress task would be lower for individuals following the completion of a 2-week EP protocol, with the largest reduction in stress response occurring after EP as a second-order treatment. In addition, it was predicted that individuals receiving the EP only intervention (i.e., 4 weeks of exercise treatment) would demonstrate a decrease in stress reactivity at each recording period (i.e., after completion of 2 and 4 weeks of exercise), with the largest change occurring after completion of all 4 weeks of exercise treatment.

Skin Conductance Response (SCR) Results

SCR data were only available for the EP only and SBA only treatment groups due to a flaw in the original data collection program used to collect data for individuals enrolled in the crossover treatments. The data collection program was revised upon recognition of this error, prior to enrolling participants in the interaction groups.

EP Only. Both participants (P5 and P6) demonstrated an increased number of significant SCR frequency in response to the stress task compared to baseline, following the 2 weeks of exercise participation (see Figure 1). However, after completing 4 weeks of EP, both participants exhibited decreased frequency of significant SCR frequency to a level at or below baseline level.

SBA Only. Individuals receiving the SBA only intervention (P7 and P8) demonstrated an inverse response to stress tasks, compared to those receiving EP only (see Figures 1 and 2). More specifically, both P7 and P8 exhibited a decreased frequency of significant SCR following two weeks of SBA, with a return to an SCR frequency level similar to above baseline recordings following the fourth week of SBA treatment (see

Figure 2). Both of these participants reported participating in a level of exercise consistent with that prescribed for individuals in the EP only group.

Based on the current findings, participation in a prescribed, monitored regimen of exercise may experience an initial increase in physiological responding to an acute stressor. Conversely, participation in a prescribed non-exercise activity (i.e., reading) paired with self-initiated participation in a mild to moderate program of exercise, may result in an initial decrease in physiological reactivity to the same acute stressor. These findings are consistent with the “defense-defeat” theory of stress-induced changes (Folkow, 1993). Participants in both treatment groups demonstrated what appears to be a habituation response to a mild to moderate program of exercise, and returned to a level of physiological responding that was at or below their SCR frequency level at baseline.

SCR Data for EP/SBA and SBA/EP Crossover Participants. Due to an error in the data recording program, baseline data, and data following the first-order treatment for P1, P2, P3, and P4 were inaccurate and could not be interpreted.

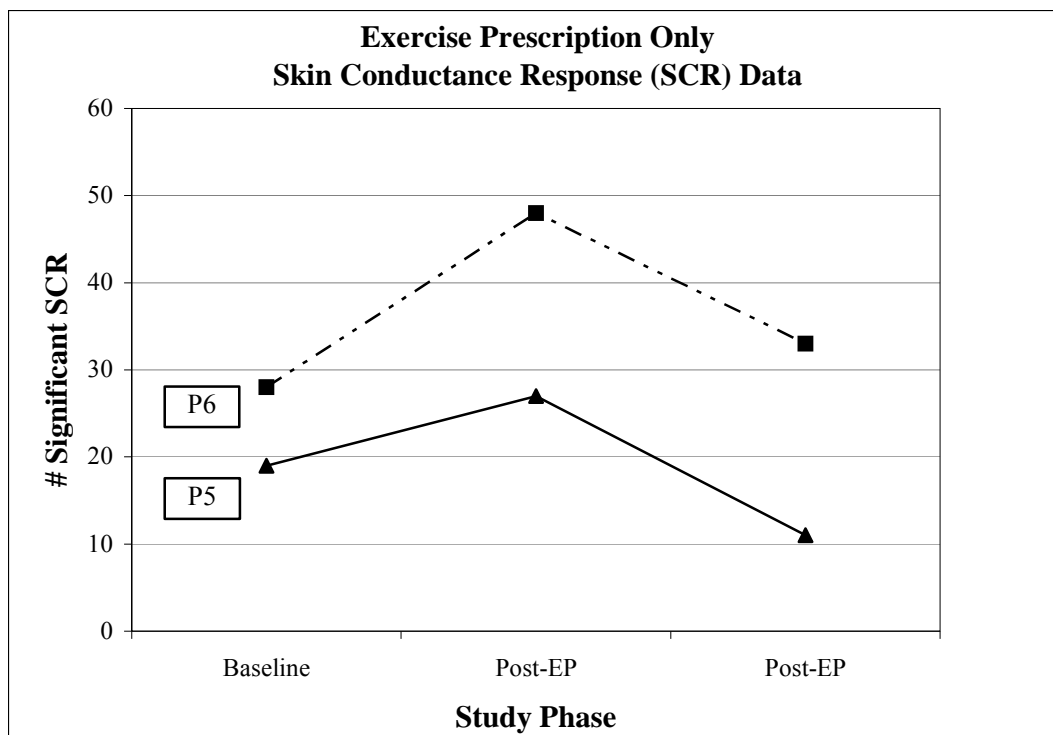


Figure 1. Skin Conductance Response (SCR) frequency for EP only. SCR exhibited during stress tasks for P5 and P6.

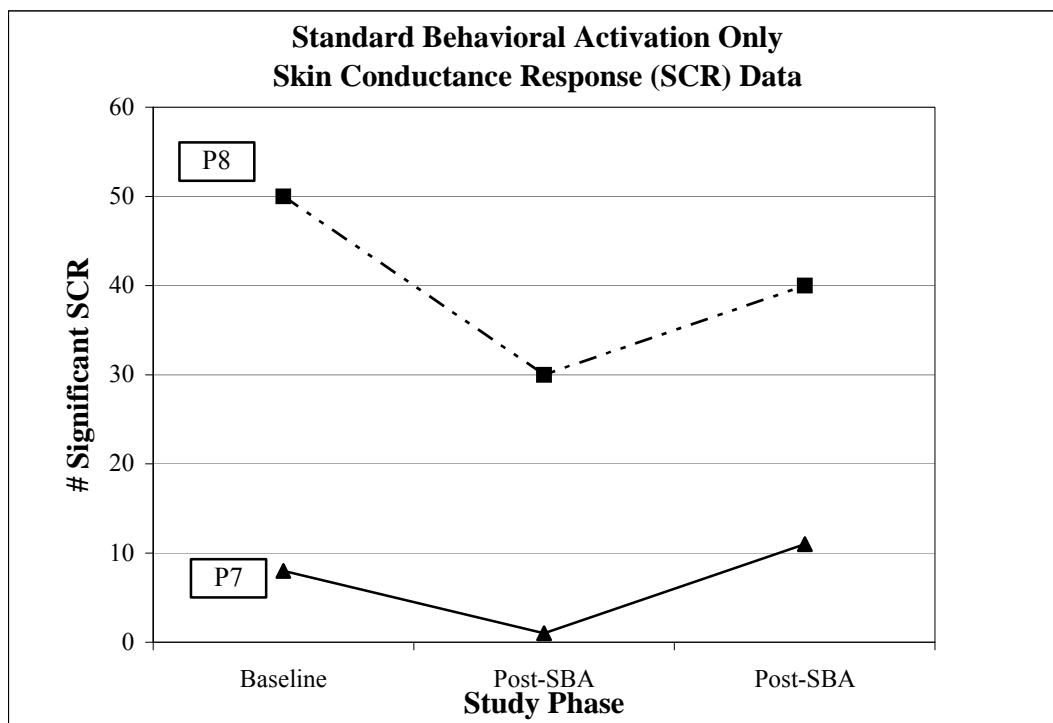


Figure 2. Skin Conductance Response (SCR) frequency for SBA only. SCR exhibited during stress tasks for P7 and P8.

Subjective Reactivity to Stress Task (i.e., POMS results)

EP Only. Depressed mood and anxiety data (i.e., POMS subscales) collected before and after stress tasks remained fairly consistent for P5 (see Figure 5). P6 showed a slight decrease in depressed mood following the stress task during the second laboratory visit (after 2 weeks of EP). A slight increase in depressed mood was observed for P6 following 4 weeks of EP, although the total response remained at a level below that reported at initial baseline. Interestingly, both participants showed an increase over time in anxiety subscale response to the stress task, with the report after 4 weeks of treatment indicating a more anxious response to the task compared to baseline (see Figure 5).

SBA Only. Depressed mood and anxiety data (i.e., POMS subscales) collected before and after stress tasks showed an interesting trend for participants completing 4 weeks of SBA (i.e., P7 and P8). Specifically, P7 showed a marked decrease in depressed mood following the first-order treatment (i.e., 2 weeks of reading); while P8 reported a marked increase in depressed mood response to the task (see Figure 6). However, P8 showed a steady increase in depressed mood responses over time, whereas P7 showed a subsequent decrease in depressed mood following completion of the second-order treatment (see Figure 6). Both participants reported a similar pattern regarding anxious responding to the stress task. Specifically, each participant showed an increase in anxious symptom endorsement following 2 weeks of treatment, with a subsequent decrease in reporting of anxious symptoms to a level at or below baseline level following 4 weeks of SBA treatment. Interestingly, both participants reported engaging in a program of exercise at or above that prescribed during the EP protocol.

EP/SBA Crossover. Although SCR frequency data for the stress tasks were not available for cross-over participants, depressed mood and anxiety (i.e., POMS ratings) data were available. These data revealed a slight decrease in depressed mood (i.e., subjective reactivity) to the stress task for both participants following the second-order treatment SBA (see Figure 3). Interestingly, both participants reported steady increases in anxious response to the stress task over time, with the most anxious response seen following 4 weeks of treatment. Both of these participants reported continued participation in a level of exercise consistent with that prescribed during the first-order (EP) treatment (see Figure 23). P3 reported less depressed mood after two weeks of EP completion.

SBA/EP Crossover. Compared to baseline, P2 and P4 reported less depressed mood in response to the stress task after completing the SBA treatment (see Figure 4). Interestingly, a marked increase in endorsement of depressed mood is observed for P2 following the stress task administered after the second-order intervention (EP). P4 showed a similar pattern, with an increase in depressed mood endorsements following the EP intervention, although less significant. Regarding anxiety in response to the stress task, a similar pattern of responding is seen for P2 and P4. Specifically, both participants reported a marked decrease in anxiety in response to the task following the SBA intervention, with a return to baseline levels of responding following the second-order (EP) intervention (see Figure 4).

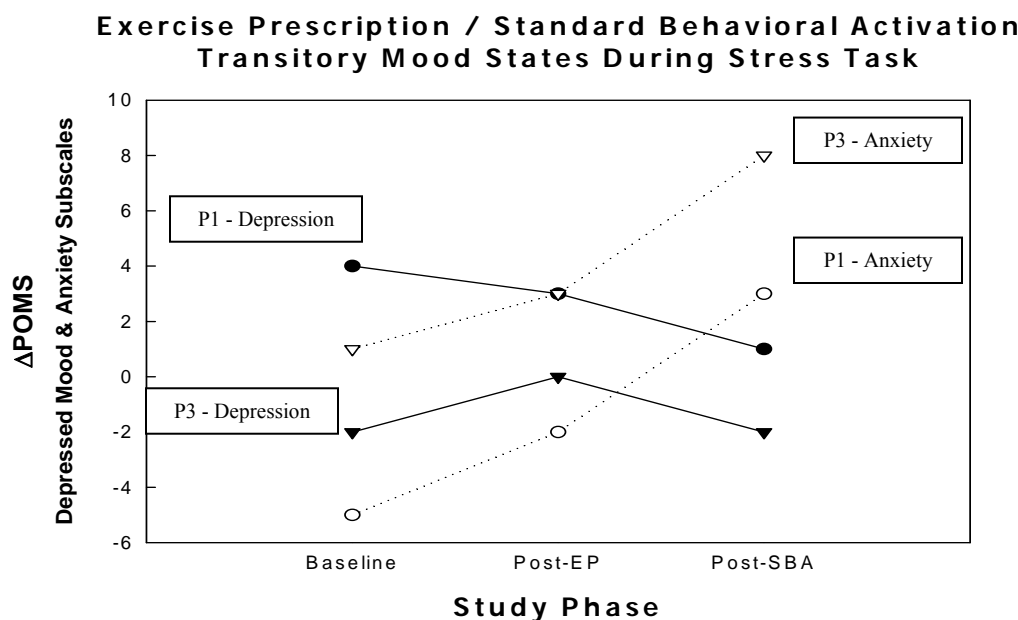


Figure 3. POMS difference score data for EP/SBA crossover group. Difference scores (post-task minus pre-task) for depressed mood (solid lines) and anxiety (dotted lines) data collected before and after laboratory stress tasks for P1 (circles) and P3 (triangles). Positive values represent rises in symptoms of depressed mood and anxiety.

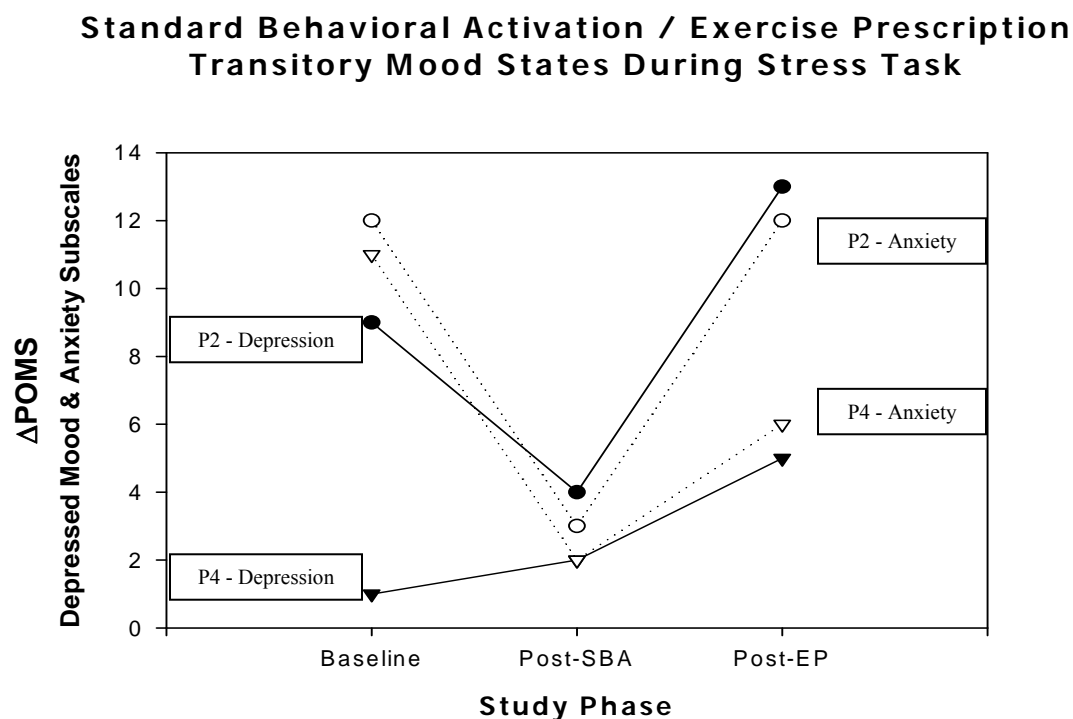


Figure 4. POMS difference score data for SBA/EP crossover group. Difference scores (post-task minus pre-task) for depressed mood (solid lines) and anxiety (dotted lines) data collected before and after laboratory stress tasks for P2 (circles) and P4 (triangles). Positive values represent rises in symptoms of depressed mood and anxiety.

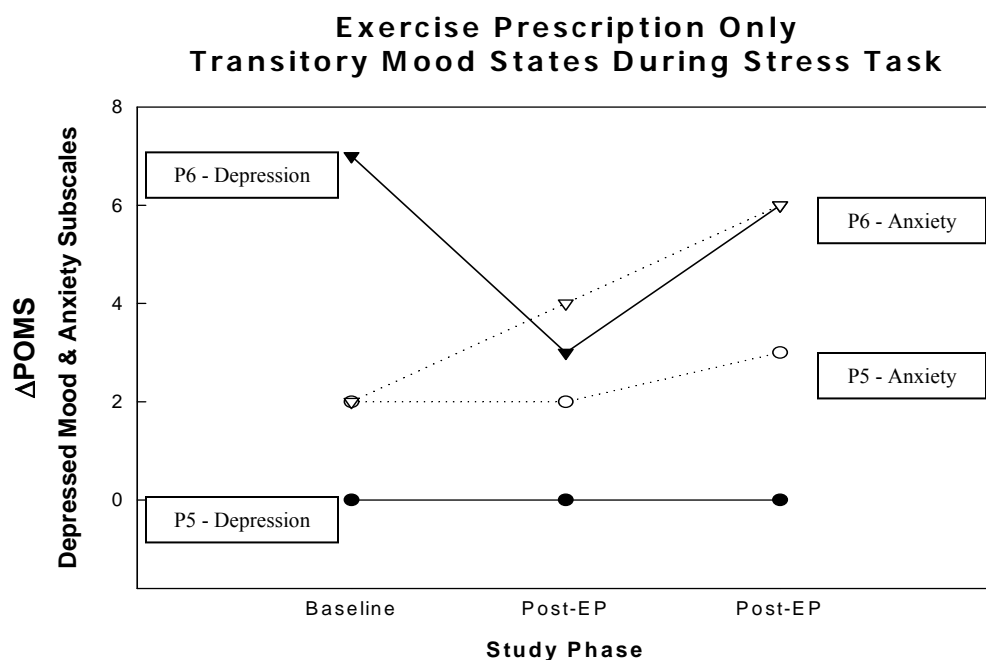


Figure 5. POMS difference score data for EP only. Difference scores (post-task minus pre-task) for depressed mood (solid lines) and anxiety (dotted lines) data collected before and after laboratory stress tasks for P5 (circles) and P6 (triangles). Positive values represent rises in symptoms of depressed mood and anxiety.

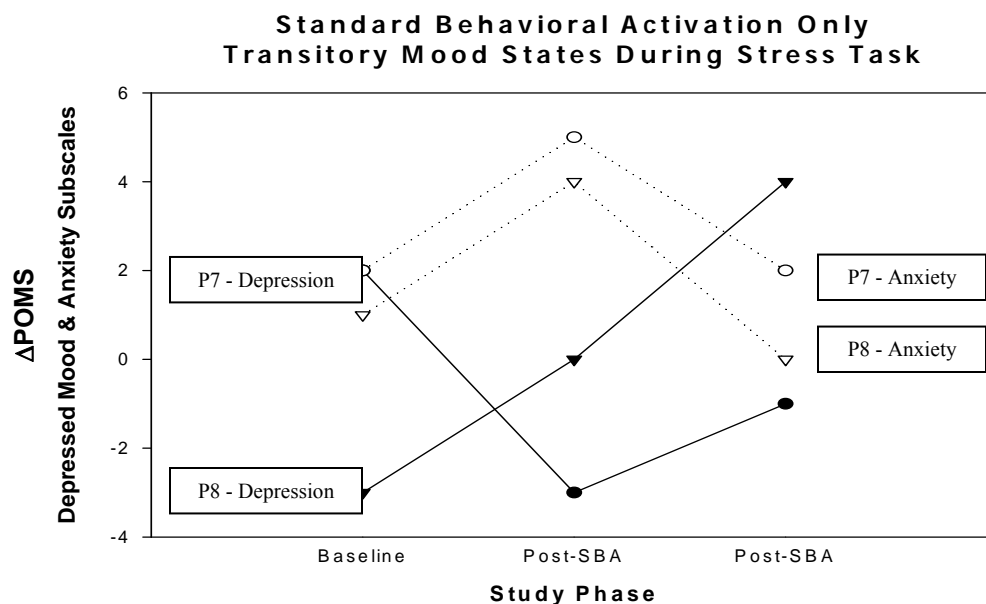


Figure 6. POMS difference score data for SBA only. Difference scores (post-task minus pre-task) for depressed mood (solid lines) and anxiety (dotted lines) data collected before and after laboratory stress tasks for P7 (circles) and P8 (triangles). Positive values represent rises in symptoms of depressed mood and anxiety.

Hypothesis 2: Impact of Exercise Treatment on Daily Stress and Mood

In addition to finding an impact on SCR frequency recordings, it was predicted that participants receiving the EP only protocol would report a lower degree of daily perceived stress during daily monitoring procedures than individuals participating in the SBA only protocol. No clear evidence for the prediction was found (see Figures 7, 8, 9, and 10). P8, who received SBA treatment for both phases, reported engaging in a program of exercise consistent with that prescribed in the EP protocol. This participant reported a decrease in daily stress ratings during the second phase of treatment and through the final baseline recording period following week 4 of treatment (SBA). For P8, it appears that exercise participation in the absence of a prescribed expectation may be a mediator of daily perceived stress (see Figure 10). However, P3, who received EP then SBA treatment, reported an increase in daily stress ratings during the SBA (second) treatment (see Figure 7).

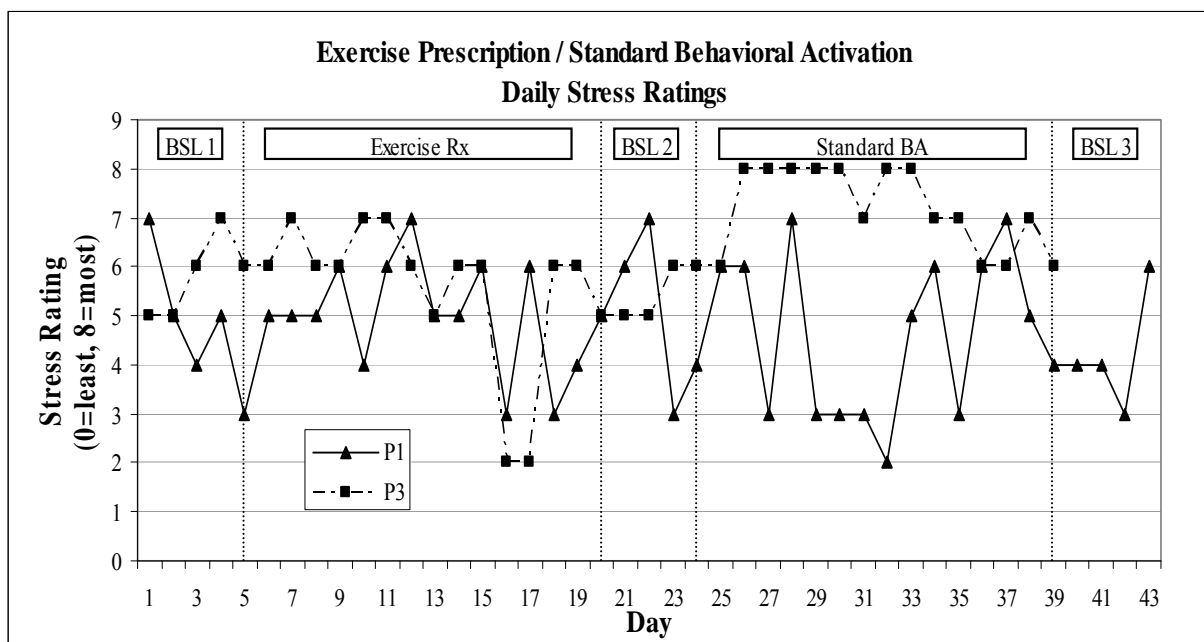


Figure 7. Perceived stress ratings for EP/SBA crossover. Daily report of perceived stress ratings for P1 (solid line) and P3 (dotted line).

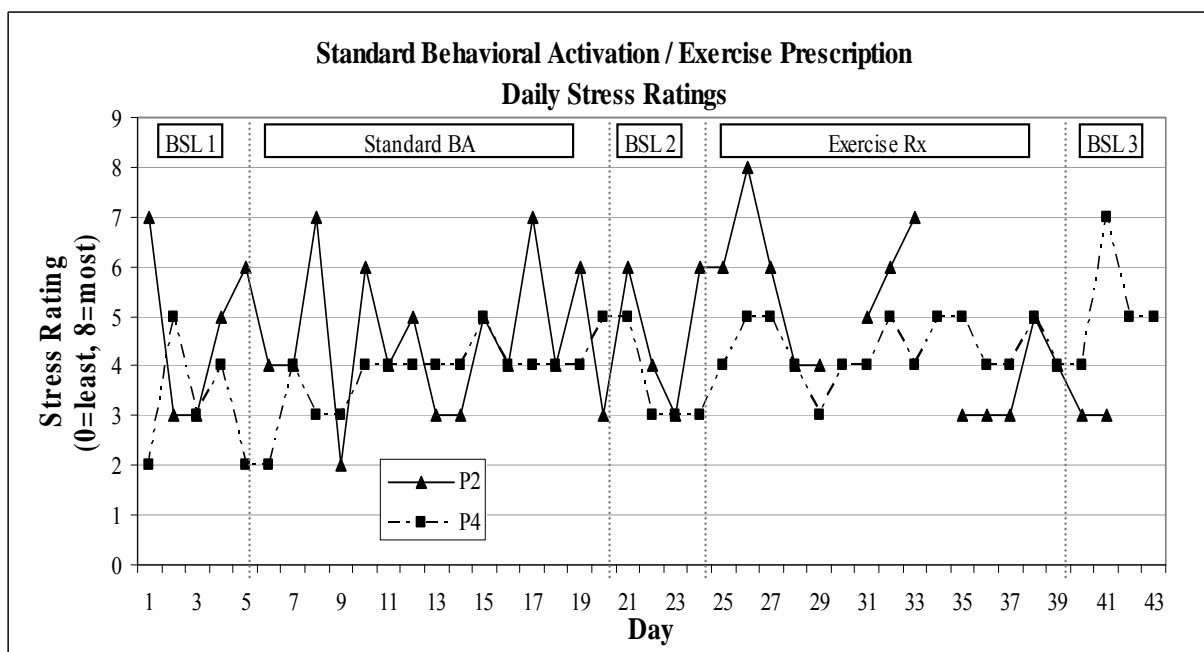


Figure 8. Perceived stress ratings for SBA/EP crossover. Daily report of perceived stress ratings for P2 (solid line) and P4 (dotted line).

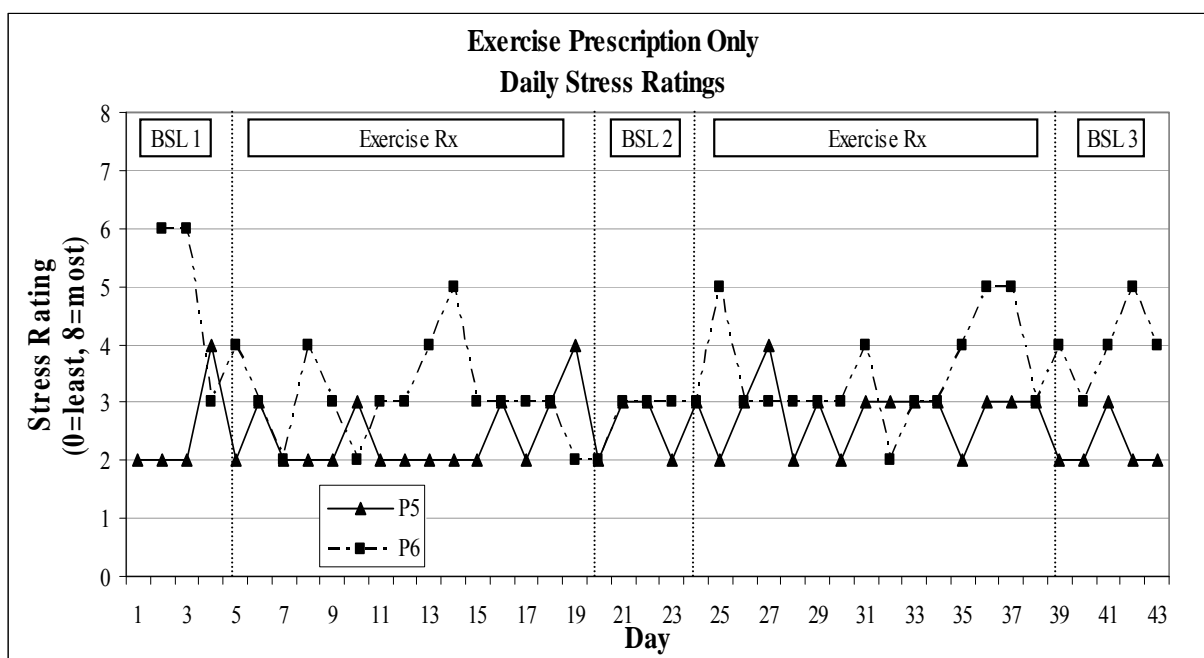


Figure 9. Perceived stress ratings for EP only. Daily report of perceived stress ratings for P5 (solid line) and P6 (dotted line).

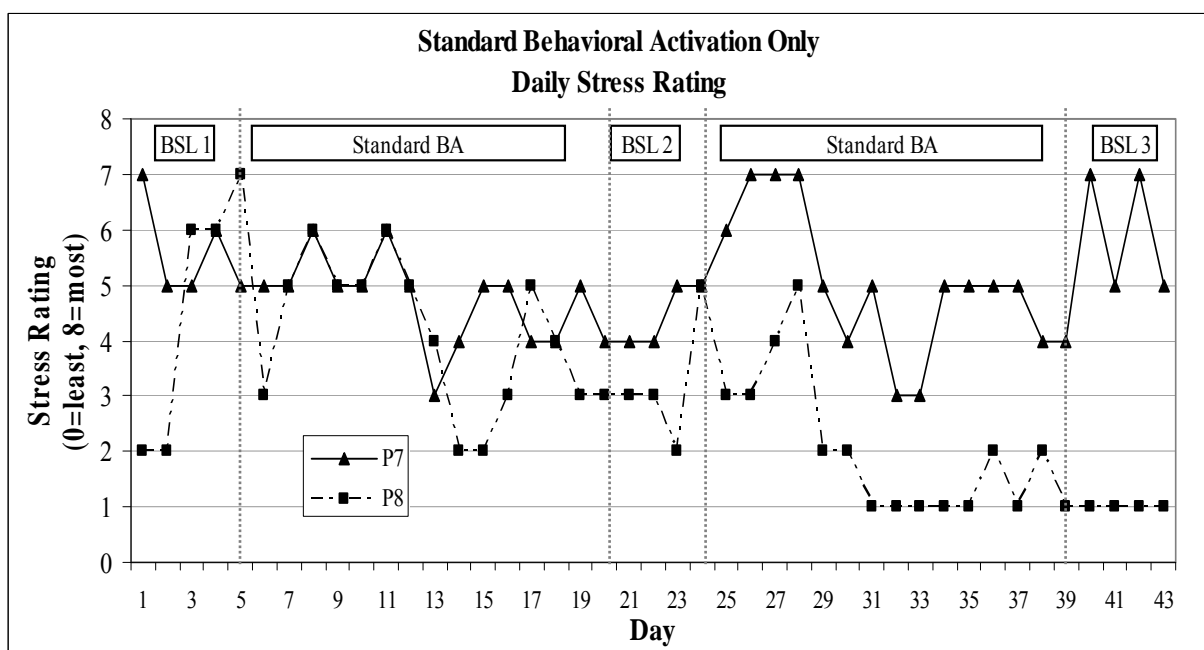


Figure 10. Perceived stress ratings for SBA only. Daily report of perceived stress ratings for P7 (solid line) and P8 (dotted line).

Hypothesis 3: Impact of Treatment on Depressive Symptom Reporting

It was predicted that individuals would report fewer depressive symptoms (i.e., BDI-II) after completion of the EP protocol, compared to individuals completing the SBA protocol. Depression symptom levels were obtained from participants on 8 occasions throughout the study (i.e., initial baseline, day 7 of each treatment, day 14 of each treatment, 1-, 3-, and 6-months follow-up). Individuals receiving 4 weeks of exercise treatment were expected to demonstrate the largest improvement, and this improvement was expected to be maintained over time (i.e., at follow-up assessments). Overall, it was expected that all participants would demonstrate the largest improvement in depressive symptoms at the end of the 4-week treatment program, regardless of treatment order, due to combined treatment effects.

EP Only

Participant 5 (P5) appears to experience no fluctuation of depressive symptoms throughout the study, with a maintained score in the “minimal” range (see Figure 13). This individual reported a score of zero after 4-weeks of study participation, although this is not a marked decrease from his previous endorsements (i.e., score of 1). However, this participant did report a gradual increase in his level of exercise participation (i.e., frequency and intensity) over time. P6 reported a marked decrease in depression symptoms after 2 weeks of exercise prescription. At day 7 of the second-order exercise treatment, this participant reported an increase in depression symptoms to a level consistent with initial baseline reporting. A return to lower levels of depression (i.e., consistent with that reported after 2 weeks of participation) was reported at the end of the fourth week of exercise participation. Interestingly, exercise participation declined

during the second baseline recording period, between exercise treatments. It is possible that this disruption in exercise participation impacted this participants experience of depression, as a return to lower levels of depression was reported with continued exercise participation at a consistent frequency and duration.

At each follow-up period, both participants (i.e., P5 and P6) reported continued participation in an exercise program consistent with EP protocol (see Figure 25). Depressive symptom reports were at a lowest level reported for both participants at the 1-month assessment period, suggesting that long-term participation in exercise may assist with mood maintenance/stability.

At 6-months follow-up, P5 continued to report a score of zero on the BDI-II (see Figure 13), and continued participation in a level of exercise consistent with, or in excess of, that prescribed during the study protocol (see Figure 25). However, P6 reported a return to his baseline level of depression (i.e., “moderate” range) at the 3-month follow-up assessment, even though this participant reported continued participation in a regular program of planned exercise (see Figure 25). In addition, P6 reported a continued worsening of depression symptoms. Interestingly, this participant reported a substantial decrease in exercise participation (see Figure 25), as well as a prolonged period of depression (i.e., 2 or more weeks), since the previous follow-up assessment.

SBA Only

Participant 7's (P7) report of depression symptoms did not stabilize over the course of the study. This participant endorsed a more chronic and debilitating form of psychopathology, had multiple comorbid illnesses, and reported more chaotic life circumstances (e.g., chronic substance dependence in current partial remission, current

methadone treatment, strained family relationships, etc.) compared to other participants in the study. This participant's depression levels varied considerably over the course of treatment (see Figure 14). However, it should be noted that this participant did report significant reductions in depression after the first and third weeks of treatment. This individual also reported engaging in exercise that was consistent with those in the EP only treatment order. Perhaps, with an expectation of participation in a standard program of exercise (such as that prescribed in the EP protocol), this participant may have demonstrated greater mood stability with the opportunity to observe the impact of a standardized program of exercise on daily functioning.

Participant 8's (P8) depression level also varied across the study (see Figure 14). After the first week of treatment, this individual's depression level worsened, and returned to baseline levels after the second week of treatment. Further reductions in depression severity were reported at the third and fourth week of treatment (see Figure 14). However, increases in depression severity were reported at the follow-up periods. It should be noted that her exercise participation gradually decreased in frequency over the course of the study (see Figure 26). In addition, this participant reported a less chronic and debilitating form of psychopathology (e.g., Bipolar II versus I Disorder, fewer comorbid psychiatric illnesses) compared to this participant's counterpart (P7) in the study (see Table 3).

At 3- and 6-months follow-up, data were only available for P8. This participant reported a slight increase in depressive symptom severity at 3-months follow-up, and a marked increase at 6-months. This participant reported continued participation in an exercise program; although the level of frequency and intensity decreased from that

reported during study participation and 1-month follow-up (see Figure 26). One additional possibility to account for this increase in depressive symptom severity is seasonal changes. At intake, this participant reported a seasonal pattern related to her experience of depression. It is important to note that this individual's 3- and 6-month follow-up assessments occurred in the fall and early-winter months.

EP/SBA Crossover

Participant 1 (P1) reported a decrease in depression severity after the first week of EP and then an increase after the second week of EP. However, after 2 weeks of SBA, depression symptom severity levels improved to a level below that reported at baseline (see Figure 11). There was a slight worsening of depression at the first month of follow-up and significant decreases in depression symptom severity were reported at the third and six month follow-up periods (see Figure 11). Interestingly, both participants (P1 and P3) reported exercising with greater frequency and intensity than requested with the prescribed regimen (e.g., aerobic workout videos plus walking; see Figure 23). Therefore, the initial increase in depression symptom severity may be related to a need for more time to habituate to a higher intensity exercise program. Consistent with this hypothesis, P1 reported a steady decrease over time in depression levels through the second-order treatment (SBA) with continued participation in an established program of exercise (see Figure 23). At follow-up, P1 reported continued participation in a regular exercise program (i.e., 3-4 days per week of aerobic exercise, and one day per week of anaerobic exercise) consistent with that prescribed during the study protocol (see Figure 23). This finding suggests that prolonged, elective participation in exercise may result in

sustained stability of depressive symptom severity after initial habituation occurs, consistent with the Salmon (2001) hypothesis.

P3 only completed the BDI through the Exercise treatment protocol. This individual's scores appeared to increase steadily through days 7 and 14 of the exercise intervention (see Figure 11). Although this participant verbally reported a decrease in depression symptom severity through the remainder of the study, there were no quantitative data to validate this report. Interestingly, this participant reported undergoing a remarkable level of stress (i.e., school- and work-related) during the time of the exercise intervention, when scores were increasing. Therefore, it is unclear what the impact of treatment was for this participant, as there were no data to interpret beyond the EP treatment phase of the study.

SBA/EP Crossover

Participant 2 (P2) reported significant reductions in depression symptom severity after the first week of SBA and then stabilized until the end of the EP treatment phase. At 1-month follow-up, this participant reported another significant decrease in depression. However, there are no follow-up scores for the second and third follow-up periods.

Participant 4 (P4) reported significant reductions in depression symptom severity after the end of the SBA treatment phase and maintained that level until the first follow-up. At the second and third follow-up periods, she reported increases in depression that exceeded her baseline score. This participant reported that she had experienced an extended period of depression during the interim between follow-up assessments, consistent with a more significant mood episode. This participant also related difficulty

adjusting to graduation from college (i.e., unemployed, social adjustment). Although this participant reported continued involvement in a program of regular exercise (see Figure 25), the level of reported intensity decreased and type of exercise changed (i.e., minimal walking only). This finding may be related to the issue of a dose-response relationship relative to exercise as a “treatment” for mood instability. For this participant, it is possible that the level of exercise reported at the second follow-up period was not adequate to moderate her experience of depressive symptoms.

At 6-month follow-up, P4 reported a decrease in depressive symptom severity (from a score of 21 to 18). In addition to continued participation in a regular program of planned exercise (i.e., walking; see Figure 25) generally consistent with that prescribed during the study protocol this participant reported initiating a regimen of mood-stabilizing medication (i.e., Geodon; see Table 4). Therefore, the decrease in depressive symptom severity may be due to a newly introduced treatment, and a secondary increase in exercise participation from the first follow-up period.

Following the SBA treatment, depression symptom severity appeared to stabilize for both participants. It is possible that inclusion of exercise in a weekly routine may act as a “mood stabilizing agent.” At 1-month follow-up, both participants reported participation in a level of exercise consistent with that prescribed during the EP study phase (see Figure 25). P2 showed a marked decrease (“moderate” to “minimal” range) in depressive symptoms, possibly explained by habituation effects. P4 reported maintenance of a “minimal” level of depression with continued exercise participation (see Figure 12).

Overall Impact of Exercise Participation on Depressed Mood

Given the lack of appreciable difference in exercise participation between treatment types (see Table 6), post-intervention BDI-II scores were compared to those reported at the initial baseline recording period (see Figure 15). P3 was excluded from this analysis, as she did not complete the second phase of reporting for this measure. Interestingly, this analysis revealed a similar effect for all participants. Specifically, a similar decrease in BDI-II scores were seen for all participants following 4 weeks of treatment, except for participants reporting BDI-II scores so low that no sizable decreases were possible due to floor effects (i.e., P5; see Figure 15).

Depressive Symptom Changes by Exercise Type

Following analysis of depressive symptom changes, difference scores of BDI-II reports (post-treatment minus initial baseline) were calculated and treated as a measure of intervention (i.e., exercise) effect. Given that no appreciable differences in exercise participation were found according to treatment order or type, participants were calculated according to three categories: a) all type of exercise, b) prescribed exercise only, and c) elective exercise only. Difference scores were correlated with each participant's total days of reported exercise and aggregated over the first baseline and four weeks of intervention to determine whether a dose-response relationship could be determined. Although not significant ($p > .05$), elective exercise was positively correlated with BDI-II score improvement, whereas prescribed exercise was not (see Table 6). The single variable that BDI-II score significantly correlated with was baseline BDI-II score ($r = .75, p = .05$). No clear dose-response relationship can be determined

from these findings, although results suggest that elective exercise participation may be an important contributor to improvement in depressive symptoms

Correlations with improvement in BDI-II score

Type of Exercise	<i>r</i>
Prescribed	- .43
Elective	+.18
All	- .11

Table 6. Correlations with improvement (decrease) in BDI-II score for all participants by treatment type. Reported days of exercise for each treatment condition were calculated and correlated with decreases in BDI-II scores.

Hypothesis 4: Impact of Treatment on Manic Symptom Expression

It was predicted that individuals endorsing manic symptoms at baseline (i.e., as measured by the Altman Self-Rating Mania [ASRM] scale) would report fewer symptoms after completing the full four weeks of treatment, due to combined treatment effects. Manic symptom scores were obtained from participants on 8 occasions throughout the study (i.e., initial baseline, day 7 of each treatment, day 14 of each treatment, 1-, 3-, and 6-months follow-up). This prediction was supported by findings for each treatment order. For the entire study, all participants reported manic symptom levels that were similar to a level at or below that at initial baseline (see Figures 11-14).

In addition, it was predicted that individuals receiving either exercise as a first-order, or sole, treatment approach would exhibit the greatest level of decline and overall stability related to manic symptoms at the end of the study as a result of habituation to a prolonged program of exercise participation. Most participants reported an initial increase in manic symptom severity with the introduction of a prescribed exercise program, or initiation of a self-initiated program of exercise consistent with that prescribed in the study protocol. Based on behavioral activation theories of stress

responding (e.g., Folkow, 1993), this finding was expected. All participants initiated and maintained participation in a program of planned exercise, regardless of treatment approach, which was an anticipated confound to the study. Therefore, the difference between these groups may be one of social accountability and treatment expectation. Overall, it was found that prolonged participation in a consistent program of mild to moderate-intensity exercise most days of the week resulted in an initial increase in manic symptom severity, and then a return to a level of manic symptom at or below that reported at initial baseline. For most participants, follow-up assessment reports revealed maintenance of a minimal to no manic symptoms below that reported at initial baseline.

EP Only

Four weeks of exercise participation appeared to maintain stability of self-reports of manic symptom severity over time for participants 6 and 7 (P6 and P7; see Figure 13). However, P6 did report an increase in manic symptom severity during the third week of exercise participation, which returned to baseline levels with continued participation (see Figure 13). It is not clear why this elevation in manic symptoms may have occurred. One possibility is that although this participant maintained compliance with treatment recommendations, she reported fewer days of walking during week 2 of EP, compared to this participant's level of participation during the first-order treatment. In addition, this individual reported engaging in no exercise during the second baseline period, with a return to EP during the second-order treatment phase. This varies from other EP participants, who demonstrated maintenance of EP exercise levels during the second baseline period. It is possible that the increase in manic symptom severity for P6 is similar to that seen for most participants receiving only 2 weeks of EP. More

specifically, this may support the hypothesis (Salmon, 2001), that one must ‘habituate’ to a *consistent* level of exercise in order for the greatest benefits to be experienced consistently (e.g., mood maintenance, or decrease of symptoms).

At 1- and 3-months follow-up, both participants reported continued participation in a level of exercise consistent with that prescribed during the EP protocol (see Figure 26). Interestingly, neither participant endorsed any manic symptoms at these follow-up assessments (see Figure 13). This finding suggests that consistent engagement in exercise may have a mood stabilizing effect in some individuals with BD. This pattern of responding was maintained at 6-months follow-up for both participants (i.e., score of zero on ASRM).

SBA Only

For both participants (i.e., P7 and P8) enrolled in this treatment order, self-reported manic symptom severity remained fairly consistent over time (see Figure 14). However, there is an interesting trend during the first 2 weeks of participation, when scores decrease, then rise again during the second week of participation (see Figure 14). It is important to note that both of these participants reported engaging in exercise levels consistent with the EP treatment protocol (see Figure 27), with an increase in frequency and/or duration over time (i.e., P8, in particular). Therefore, these participants were, essentially, engaging in two forms of behavioral activation experiences throughout the study (i.e., reading and walking).

At 1-month follow-up, P7 reported manic symptom severity consistent with baseline levels, and P8 reported somewhat lower levels of manic symptoms (see Figure 14). Both participants reported maintaining a consistent level of exercise participation

during the interim between the close of the study protocol and initial follow-up assessment. P8 reported maintenance of exercise of greater intensity than that expected during EP protocol completion. Therefore, it is important to note that she demonstrated a significant reduction in manic symptom severity as she habituated to these self-initiated levels of exercise. Interestingly, although this higher frequency may cause some fluctuation in mood symptoms initially, it appears that the habituation hypothesis may apply to this participant. However, it should be noted that this participant appeared to require a longer period of time to habituate to a more intense regimen of exercise. Intuitively, this need is understandable, given physiological demands that increased intensity of exercise places on the body.

Although data for P7 was lost for extended follow-ups, data were available for 3- and 6-month follow-up for P8 (see Figure 14). This individual reported continued involvement in a regular program of aerobic exercise (see Figure 27), consistent with that prescribed during the EP study protocol, and also reported decreases in manic symptom severity. This individual did report a significant increase in depression symptom severity during these final follow-up assessments, consistent with her intake report of experiencing seasonal fluctuations (i.e., fall and winter increases) in depression.

EP/SBA Crossover

Participant 1 (P1) reported an initial increase in manic symptom severity, followed by a large decrease in symptoms after 2-weeks of sustained exercise participation (see Figure 11). This individual endorsed no manic symptoms at the outset of the second-order treatment (SBA). However, she reported continued participation in exercise consistent with that recommended during the EP protocol (see Figure 24).

Through follow-up (1-, 3-, and 6-months), this participant endorsed continued participation in a program of exercise consistent with the EP, and demonstrated a stabilized level of manic symptom severity, accordingly. It appears, for this participant, that consistent involvement in exercise may play a key role in moderating manic symptom severity. However, this participant did report a five-day period of elevated mood during the interim between the 3- and 6-month follow-up assessments, which is not captured in the days reported on the ASRM (i.e., symptoms experienced during the past two weeks).

Participant 3 (P3) demonstrated a similar increase in manic symptom severity with the initiation of the EP protocol, albeit less dramatic than that reported by P1 (see Figure 11). ASRM data were not reported, and could not be collected retrospectively, for this participant during the second-order treatment. Similarly, data for this participant were lost at follow-up. Given the lack of data for this participant, it is difficult to interpret the impact of treatment, or treatment order, on manic symptom severity for this individual.

SBA/EP Crossover

Participant 2 (P2) demonstrated a mild increase in manic symptom severity with initial behavioral activation (See Figure 12). During the second week of treatment, this participant reported a level of manic symptoms that was similar to her baseline level. For P4, manic symptom reports increased after 2 weeks of participation in the first-order treatment (SBA), with the largest increase, compared to baseline, occurring after exercise participation (see Figure 12). During the second phase of treatment, when exercise was introduced, there is a trend similar to that demonstrated by individuals completing the

aforementioned crossover treatment protocol (i.e., EP to SBA; see Figures 11 and 12). Specifically, 2-weeks of exercise prescription appeared to elevate self-reports of manic symptom severity for both participants in this treatment group.

At 1-month follow-up, P2 reported a generally consistent level of manic symptom severity and P4 reported manic symptoms consistent with her baseline level. It is important to note that both participants endorsed maintenance of a regular exercise program similar to that prescribed during EP protocol.

At the 3- and 6-month follow-up assessments, data were only available for P4. This participant reported continued participation in a regular program of exercise, generally consistent with that expected during the EP protocol (see Figure 25). She reported a gradual decrease in manic symptom severity (to a score of zero at final follow-up). It is important to note that this participant did report experiencing a prolonged period of depression, which she attributed to a significant elevation in life stress associated with unemployment and adjustment to new social roles. Therefore, it is difficult to conclude that maintenance of an exercise program was responsible for the slight decrease in manic symptom endorsement between the second and final follow-up assessments.

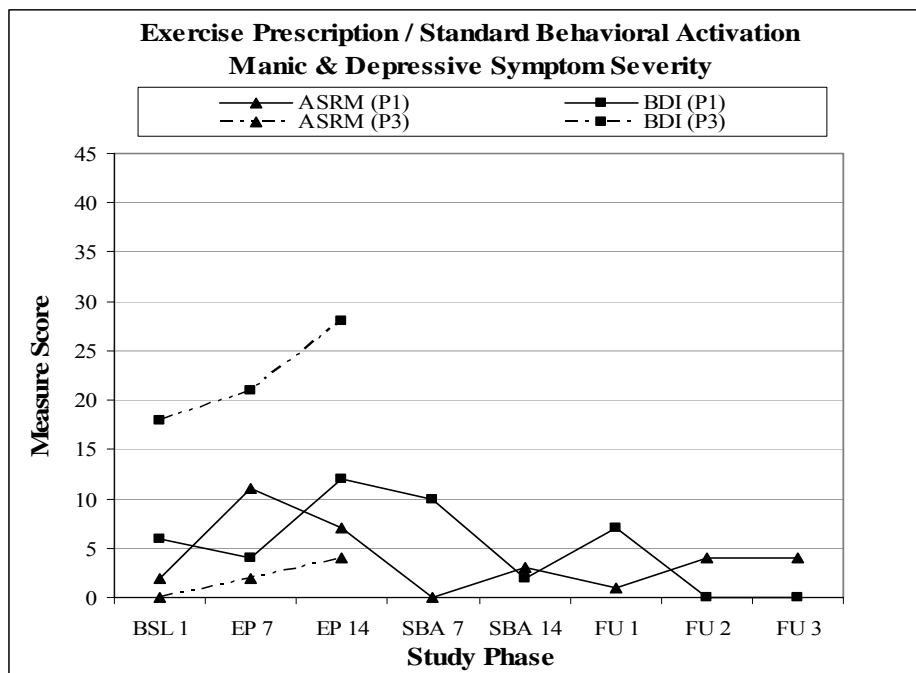


Figure 11. ASRM and BDI-II scores for EP/SBA crossover. Manic (triangles) and depressive symptom (squares) endorsement for P1 (solid line) and P3 (dotted line), who completed the EP to SBA crossover treatment protocol.

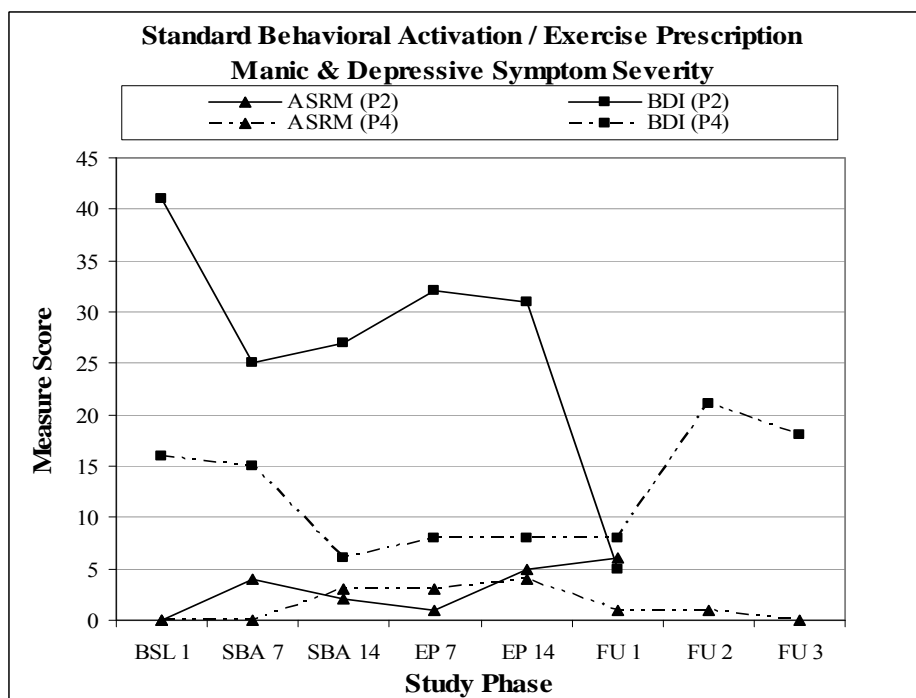


Figure 12. ASRM and BDI-II scores for SBA/EP crossover. Manic (triangles) and depressive (squares) symptom endorsement for P2 (solid line) and P4 (dotted line), who completed the SBA to EP crossover treatment protocol.

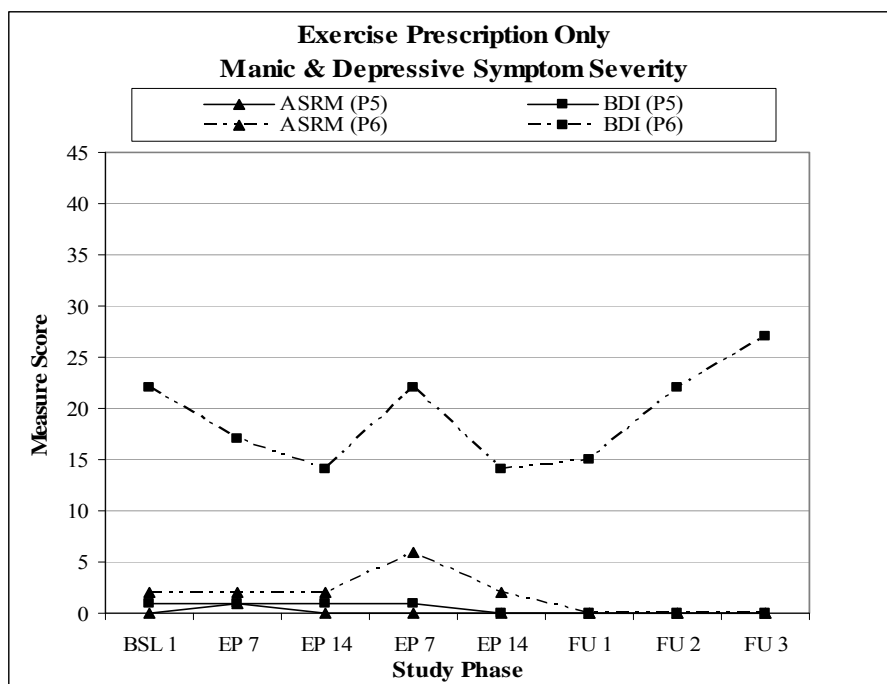


Figure 13. ASRM and BDI-II scores for EP only. Manic (triangles) and depressive (squares) symptom endorsement for P5 (solid line) and P6 (dotted line) who completed the EP only treatment protocol.

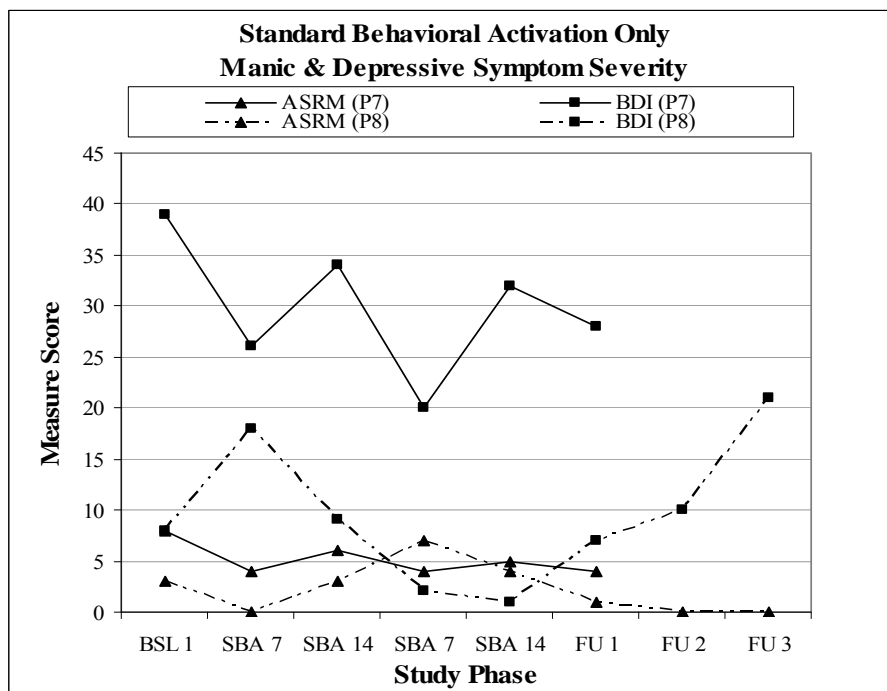


Figure 14. ASRM and BDI-II scores for SBA only. Manic (triangles) and depressive (squares) symptom endorsement for P7 (solid line) and P8 (dotted line) who completed the SBA only treatment protocol.

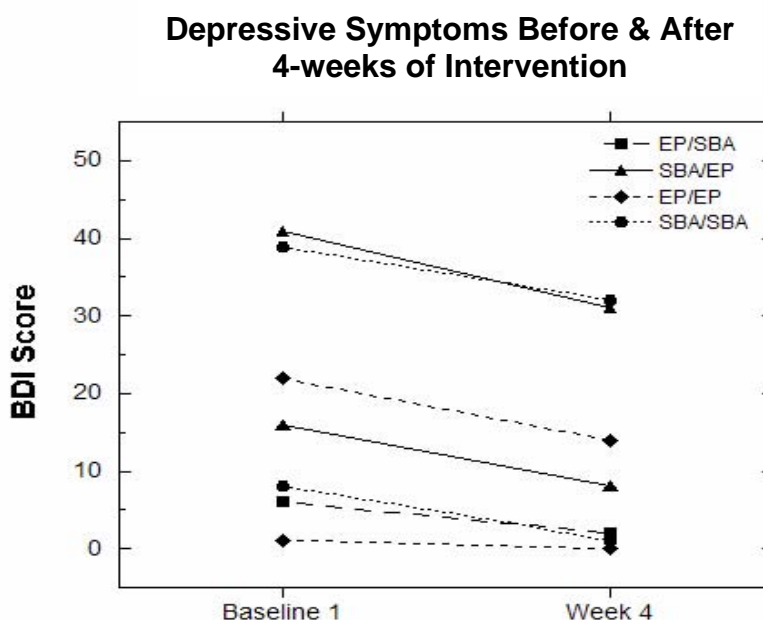


Figure 15. BDI-II score changes following 4 weeks of treatment. Beck Depression Inventory – II (BDI-II) score changes for all participants completing both phases of reporting for the study (i.e., excluding P3 who did not report BDI-II data during the second phase of the study).

Hypothesis 5: Impact of Treatment on Coping Strategy Use

It was predicted that individuals participating in the EP intervention would report decreases in the use of maladaptive coping strategies (i.e., substance use, denial, behavioral disengagement, and self-blame), and related increases in the use of adaptive forms of coping (i.e., active coping, planning, positive reframing, acceptance, humor, religion, using emotional support, using instrumental support, self-distraction, and venting), as measured by the Brief COPE inventory. It was expected that individuals who maintained participation in exercise, at a level consistent with exercise prescription, would stabilize regarding the use of adaptive and problematic coping strategies. Similarly, decreases in exercise participation were expected to result in an increase in the use of maladaptive coping, and also a decrease in the use of adaptive, coping strategies.

In addition, it was predicted that individuals who completed the EP only intervention protocol would demonstrate the greatest reduction in the use of maladaptive coping strategies, as more time would be available to habituate to a new program of exercise-specific behavioral activation.

EP Only

Participant 5 (P5) reported a stable level of adaptive coping strategy use from baseline to post-treatment (see Figure 18). At 1-month follow-up, this participant reported a remarkable increase in use of adaptive coping strategies; however, at follow-ups 2 and 3, this participant reported a decline near baseline levels of use. It should be noted that this participant indicated significant life changes (i.e., retirement) had occurred between the first and second follow-up assessments. In addition, P5 reported continued involvement in exercise at a level at or exceeding that prescribed during the EP intervention at 6-month follow-up.

Participant 6 (P6) reported a decline in the use of adaptive coping strategies from baseline to post-treatment (see Figure 18). This participant also indicated significant life stressors (e.g., marital distress, health problems) during this time. However, this participant reported an increase in adaptive strategy use at the 1-month follow-up which appeared to stabilize across remaining follow-ups. Interestingly, P6, who reported a decrease in daily exercise participation days (i.e., 4 days per week, compared to 6 days per week in previous follow-up assessments; see Figure 25), reported a continued decrease in adaptive and subsequent increase in problematic coping strategy use at 6-months follow-up (see Figure 18).

SBA Only

Individuals in the SBA only treatment protocol (i.e., P7 and P8) reported stable use of maladaptive coping strategies (see Figure 19). P7 reported a gradual decline in the use of adaptive coping strategies from baseline to post-treatment; however this participant reported a considerable use of adaptive strategies at the first follow-up (no data were available for 3- and 6-month follow-ups). P8 reported a slight decline in the use of adaptive coping strategies from baseline to the end of the first 2 weeks of treatment, and a more considerable decline from 2 weeks to post-treatment (see Figure 19). This participant reported a slight decline in adaptive coping strategy use from post-treatment to the 3-month follow-up, with a significant increase of adaptive strategy use at 6-months follow-up. However, the level reported at 6-months follow-up remained below use of adaptive strategies reported at baseline. This finding may be related to this participant's reported seasonal pattern of depression given that follow-ups occurred in the fall/winter months.

Interestingly, both individuals receiving the SBA only intervention reported participating in a program of exercise consistent with that prescribed in the EP protocol, without the expectation of exercise participation (see Figure 26). Exercise participation may be important regarding enhancement of perceived availability and use of adaptive coping strategies. This finding also suggests some utility of exercise “prescription” being monitored by a professional.

EP/SBA Crossover

Both participants in this treatment order (i.e., P1 and P3) reported relatively consistent use of both adaptive and maladaptive coping strategies over time (see Figure

16). P3 declined to participate in all follow-up assessment periods. However, P1 reported data for the completion of the study protocol. For P1, there is a general positive relationship between the use of adaptive and maladaptive coping strategies (i.e., a slight increase in one style was mirrored by a slight increase in the alternate style of coping). Although slight variations in the use of maladaptive coping strategies are seen over time for this participant, she remained generally stable in reported use. However, this participant's use of adaptive coping strategies increased (beginning at 1-month follow-up), and was maintained until the final (6-month) follow-up assessment (see Figure 16).

SBA/EP Crossover

Both participants (i.e., P2 and P4; see Figure 17) reported using similar levels of maladaptive coping strategies throughout the study. P2 reported using more adaptive coping strategies compared to baseline, especially after completion of the EP phase. This pattern of increased use of adaptive coping strategies remained through the 1-month follow-up (no data for follow-ups 2 and 3). P2 reported a steady decline in the use of maladaptive coping following the EP (second) treatment, though 1-month follow-up. Her use of adaptive strategies remained consistent through these assessment periods (see Figure 17).

Participant 4 (P4) reported a gradual decline in adaptive coping use from baseline to the end of the SBA treatment phase, which continued until the 3-month follow-up (see Figure 17). This individual indicated that she experienced a prolonged period of depression between 1- and 3-month follow-up periods, and attributed this to significant stressful life circumstances (i.e., graduation from college, prolonged unemployment). However, this participant reported an increase in adaptive coping strategy use at the 6-

month follow-up compared to baseline. Both participants reported continued participation in an exercise program consistent with that prescribed during the EP protocol (see Figure 25).

After two weeks of treatment, only P1 (EP to SBA) and P2 (SBA to EP) reported greater use of adaptive coping strategies over baseline. Only P3 (EP to SBA) reported a slight decrease in maladaptive coping use. After 4 weeks of treatment, only P2 reported an increase in adaptive coping strategy use over baseline. Only P1 (EP to SBA) and P2 (SBA to EP) reported decreases in maladaptive coping strategy use. Although not hypothesized, it appears that the crossover treatments resulted in the greatest impact on the perception of adaptive and maladaptive coping use.

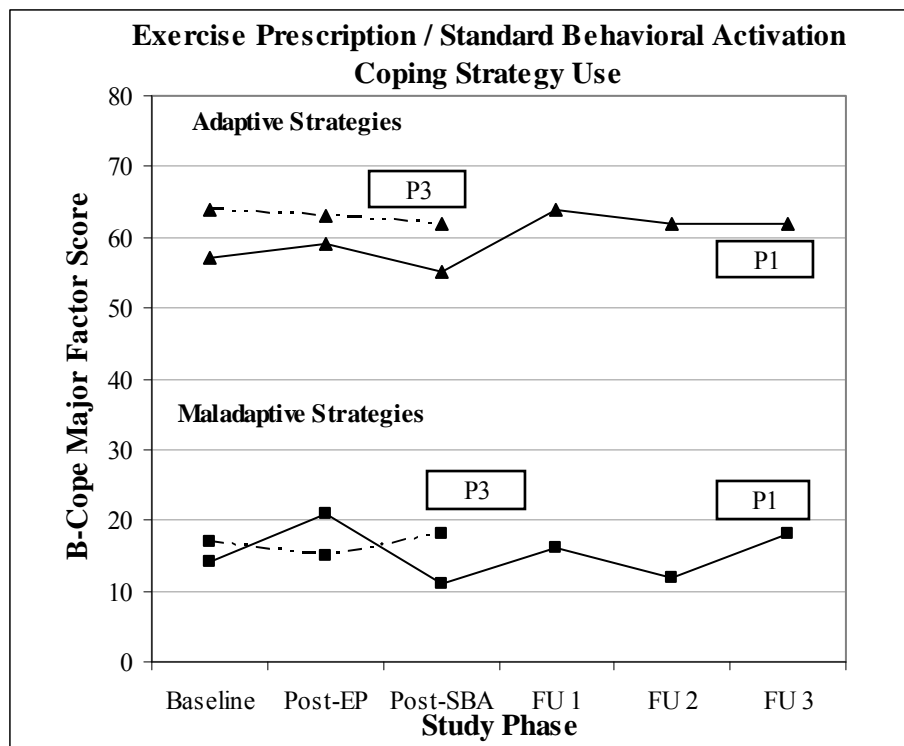


Figure 16. Coping strategy use for EP/SBA crossover. Coping strategy use for P1 (solid line) and P3 (dotted line).

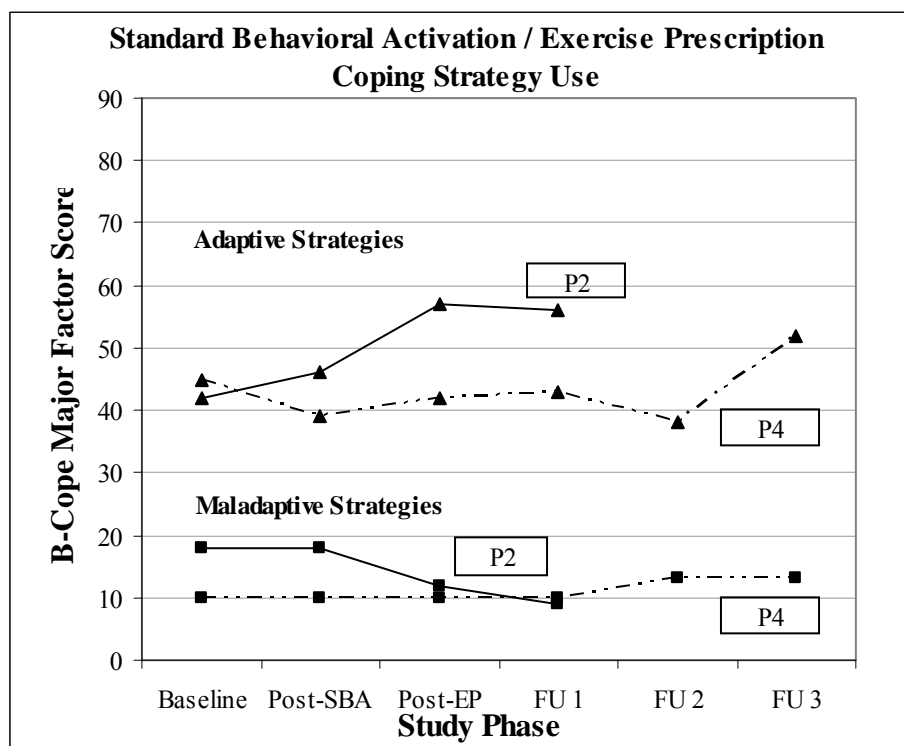


Figure 17. Coping strategy use for SBA/EP crossover. Coping strategy use for P2 (solid line) and P4 (dotted line).

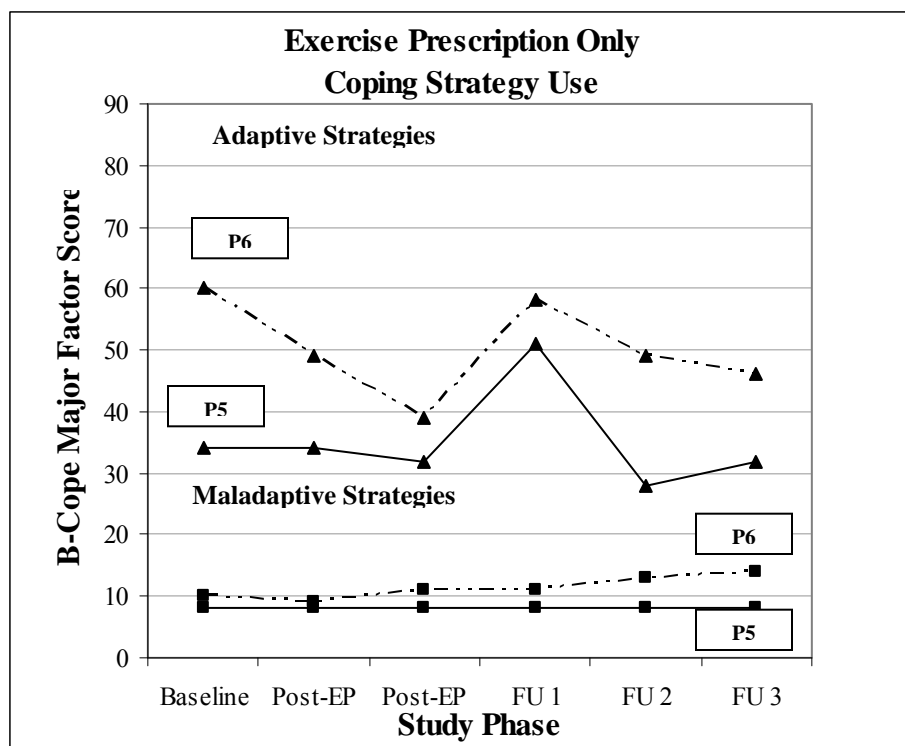


Figure 18. Coping strategy use for EP only. Coping strategy use for P5 (solid line) and P6 (dotted line).

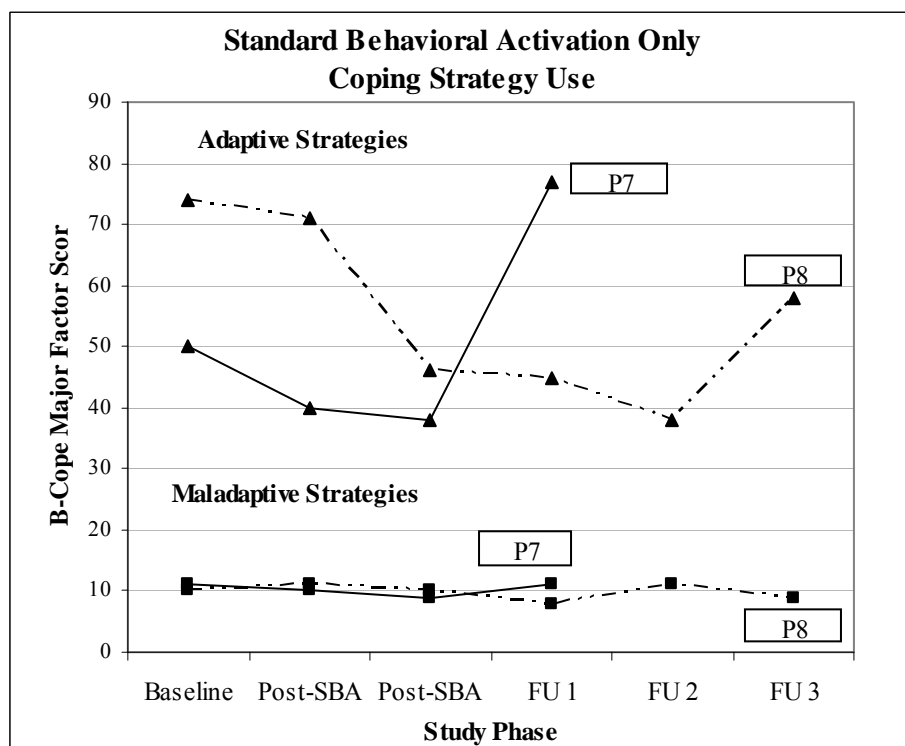


Figure 19. Coping strategy use for SBA only. Coping strategy use for P7 (solid line) and P8 (dotted line).

Hypothesis 6: Impact of Exercise on Perception of Experienced Daily Hassles

It was predicted that individuals participating in the EP only intervention would demonstrate the greatest improvement in perceived experiences of daily hassles (i.e., as measured by the Survey of Recent Life Events [SRLE]). In addition, it was expected that individuals participating in the 4-week exercise intervention would maintain exercise participation, and, therefore maintain their report of less stressful experiences, compared to individuals in alternative treatment orders. Similarly, it was predicted that individuals not receiving a prescription for exercise participation would report less improvement on the SRLE measure over the course of the study.

EP Only

Individuals in this treatment order (i.e., P5 and P6) reported the most consistent pattern of daily hassles over the course of the study (see Figure 22). In addition, these individuals reported the lowest levels of daily hassles compared to participants in other treatment orders. However, P5 reported a level of stressful daily hassles substantially lower than other participants enrolled in the study. P6 reported a level of daily hassles consistent with individuals receiving other treatments in the study (see Figures 20-23). Both individuals receiving the EP only treatment reported a minor increase in the report of stressful daily hassles at 1-month follow-up, following one month of adjusting to the removal of an exercise prescription. P6 continued to report an increase in daily hassles at 3- and 6-months follow-up (see Figure 22). P5 continued to report a fairly consistent level of daily hassles throughout the study (see Figure 22), with continued participation in a consistent level of exercise (see Figure 26) in the absence of prescription. This finding

lends further support for the hypothesis that habituation to an exercise regimen may positively impact perception of stressful events.

SBA Only

Individuals in the SBA only intervention (P7 and P8) reported the most inconsistent or erratic experience of stressful daily hassles during the treatment component of the study (see Figure 23). P7 reported a dramatic decrease from baseline to the end of the first 2-weeks of treatment, and then reported a slight increase after 4-weeks of treatment. A further slight decrease was observed for P7 at the 1-month follow-up (no follow-up data for 2 and 3). P8 reported a significant increase in daily hassles from baseline to after 2-weeks of SBA treatment. However, this individual reported a significant decrease after 4 weeks of treatment. At follow-up assessments, P8 reported steady increases in the experience of daily hassles (see Figure 23). Although these individuals did not receive a prescription of exercise, each reported regular involvement in a self-initiated exercise program (see Figure 27). This suggests support for the prediction that participating in a prescribed and monitored program of exercise may be most useful regarding the impact on perceived experiences of daily life hassles.

EP/SBA Crossover

Participant 3 (P3) reported a substantial decrease in daily hassles following the EP intervention (see Figure 20). This level was maintained through the end of the second-order treatment (SBA of reading; no follow-up data were available for this participant). Similarly, participant 1 (P1) reported a substantial decrease in daily hassles following the end of the second-order (SBA) treatment (see Figure 20). However, this participant's report of daily hassles increased to a level exceeding that reported at baseline through all

follow-up periods. It is possible that the decreased frequency and intensity of monitoring activity level may result in less structure related to exercise behavior, and an increase in perceived stressful daily hassles, as a result.

SBA/EP Crossover

Individuals enrolled in this treatment order (P2 and P4) reported an interesting pattern of gradual decreases in daily hassles, with the lowest report occurring at the end of the EP protocol (see Figure 21). However, when the prescription of exercise was removed, these individuals reported a gradual increase in experience of daily hassles. Interestingly, individuals in this treatment order reported participating in a program of exercise consistent with that prescribed during the EP protocol beginning at the outset of study participation, when no prescription was in place. This finding, that reported stressful daily hassles was lowest following the EP intervention, lends support for the hypothesis that habituation to exercise may result in decreases in perceived stress. In addition, this finding provides support for the hypothesis that a prescription of mild to moderate exercise levels may decrease an individual's perception of daily life hassles.

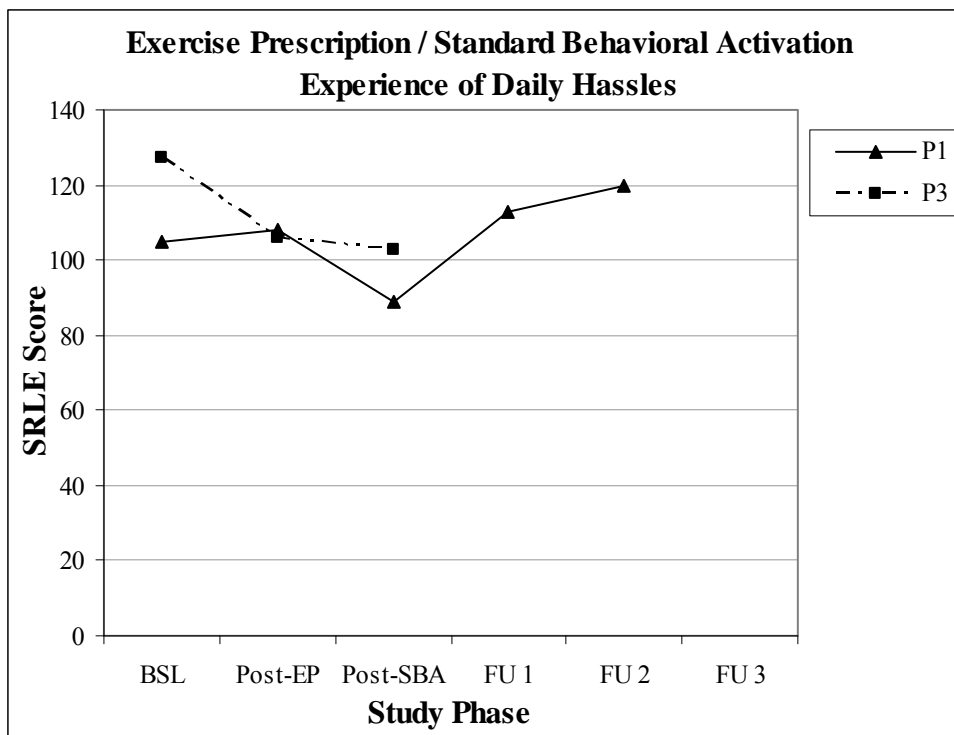


Figure 20. SRLE scores for EP/SBA crossover. Experience of daily hassles for P1 (solid line) and P3 (dotted line), who received the EP to SBA crossover treatment protocol.

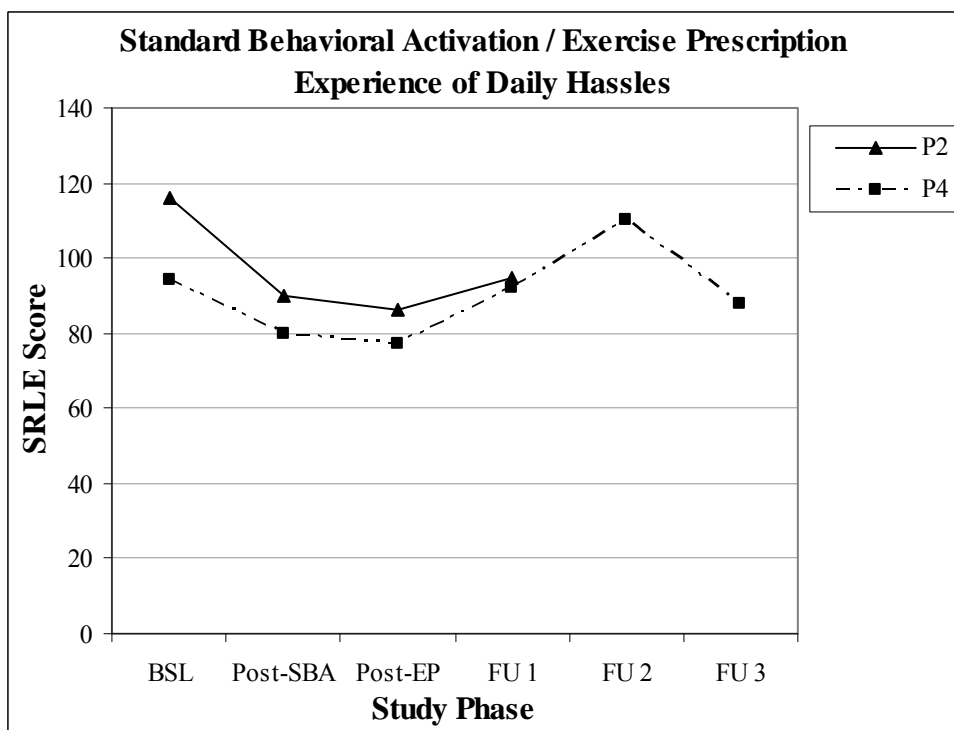


Figure 21. SRLE scores for SBA/EP crossover. Experience of daily hassles for P2 (solid line) and P4 (dotted line), who received the SBA to EP crossover treatment protocol.

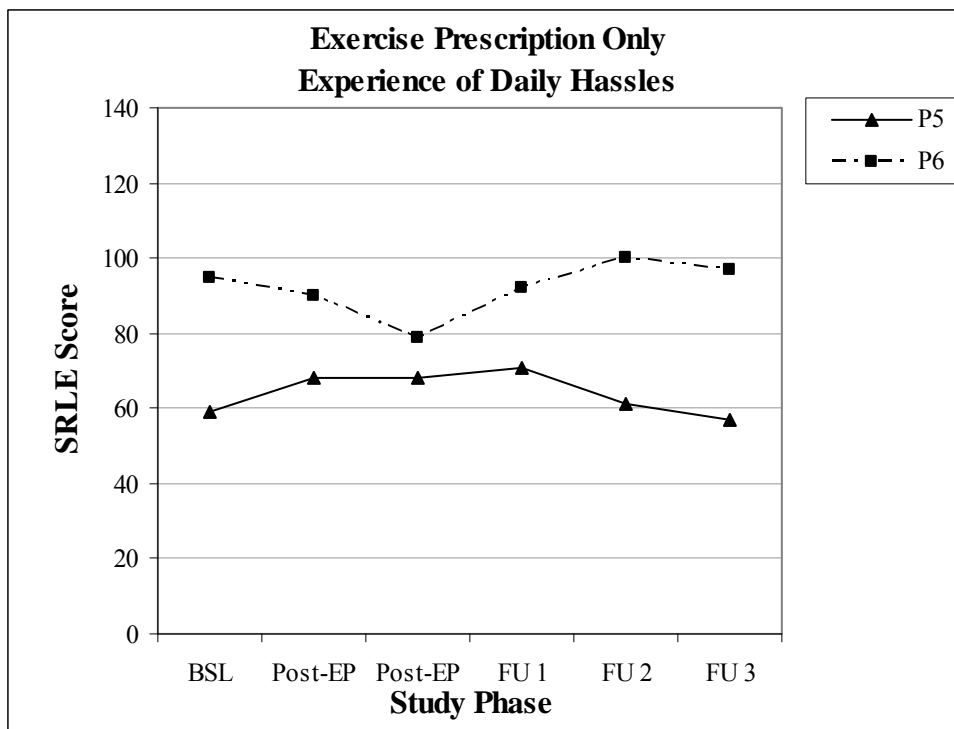


Figure 22. SRLE scores for EP only. Experience of daily hassles for P5 (solid line) and P6 (dotted line), who received the EP only treatment protocol.

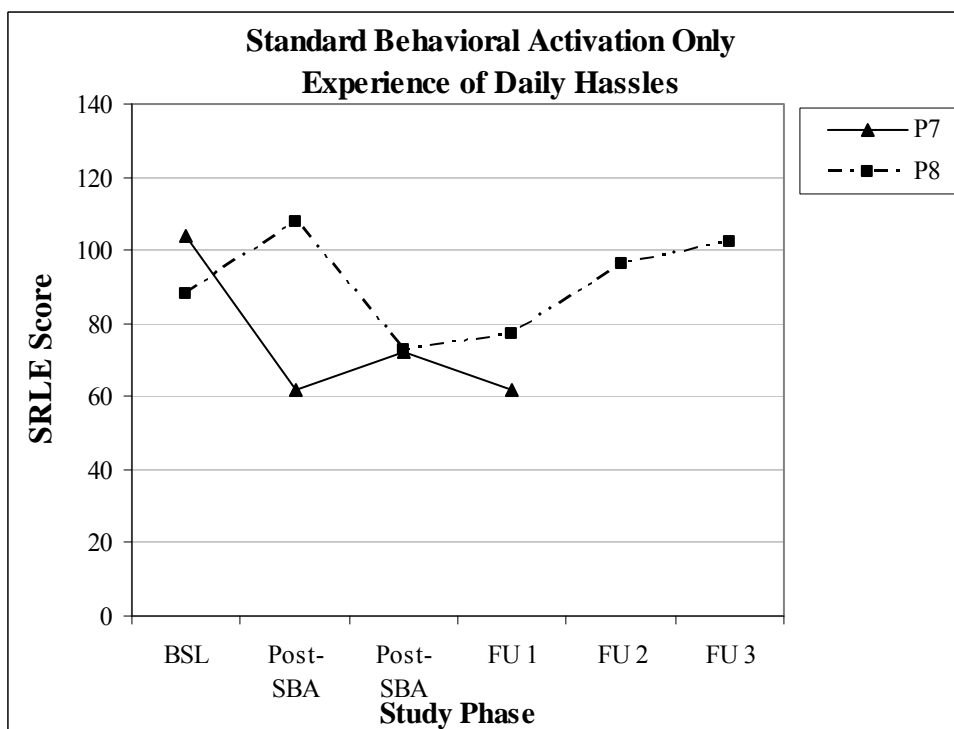


Figure 23. SRLE scores for SBA only. Experience of daily hassles for P7 (solid line) and P8 (dotted line), who received the SBA only treatment protocol.

Additional Findings

Exercise Participation

Individuals who completed the study were screened for level of exercise participation prior to study enrollment. All participants enrolled reported engaging in minimal weekly exercise (i.e., less than 3 days per week). Interestingly, all participants, regardless of treatment order, reported initiation and maintenance of a regular program of exercise (see Figures 24-27). In fact, frequency of participation (i.e., days per week) did not differ substantially between groups (see Table 7). Although participants were instructed to engage only in activities that they would have prior to study entry, exercise participation during baseline recording periods did not return to pre-study (i.e., sedentary) levels. A single exception to this finding was found for participants receiving the EP only intervention, who engaged in minimal levels of exercise during the second baseline recording period (i.e., between treatment conditions).

Although not included as a hypothesis, an increased level of exercise in all participants was an expected confound. This expectation was a primary reason for including the two interaction group treatment protocols in the study; so that the effect of prolonged prescribed exercise could be more clearly differentiated from self-initiated and self-maintained exercise participation. Findings from non-exercise interventions were interpreted from the perspective that each individual was participating in two forms of behavioral activation, and suggest that for some variables (e.g., physiological reactivity) may be most useful in decreasing physiological response to an acute stressor.

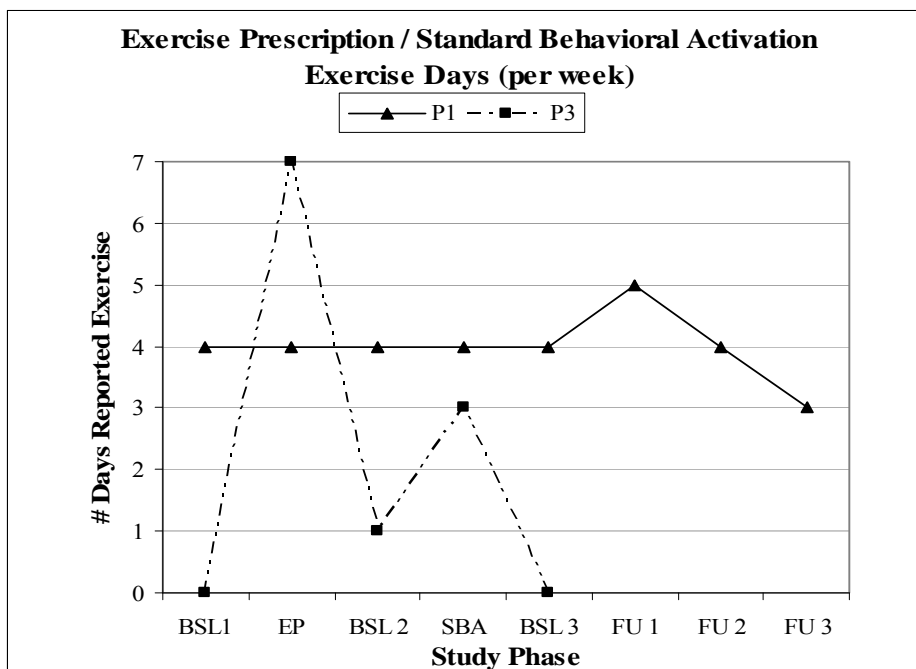


Figure 24. Exercise participation for EP/SBA crossover. Reported days of exercise for P1 (solid line) and P3 (dotted line), who completed the EP to SBA crossover treatment protocol. A total of 5 days of exercise were possible for baseline (BSL) recording periods, compared to 7 days possible for all other recording periods.

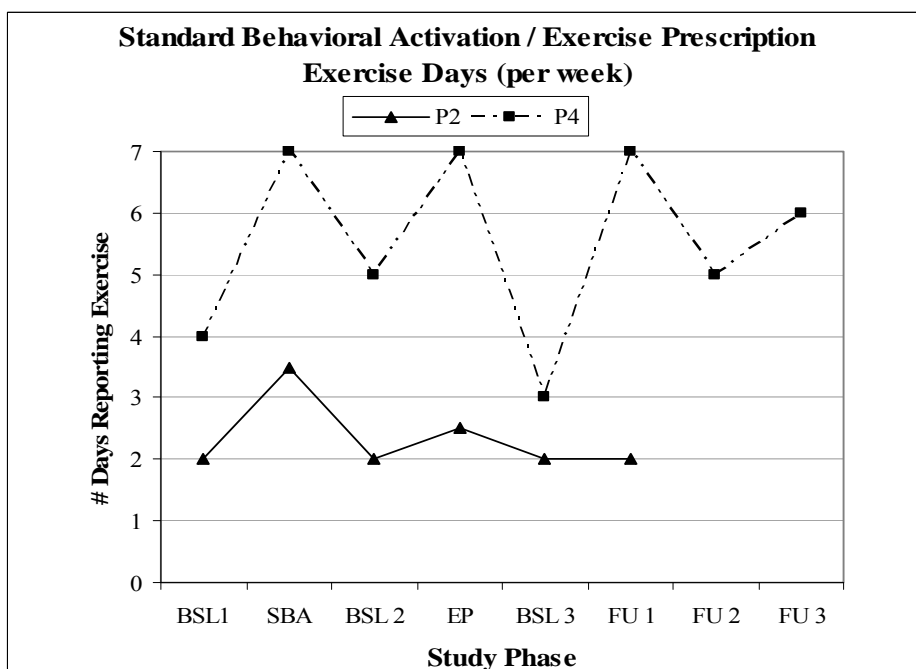


Figure 25. Exercise participation for SBA/EP crossover. Reported days of exercise for P2 (solid line) and P4 (dotted line), who completed the SBA to EP crossover treatment protocol. A total of 5 days of exercise were possible for baseline (BSL) recording periods, compared to 7 days possible for all other recording periods.

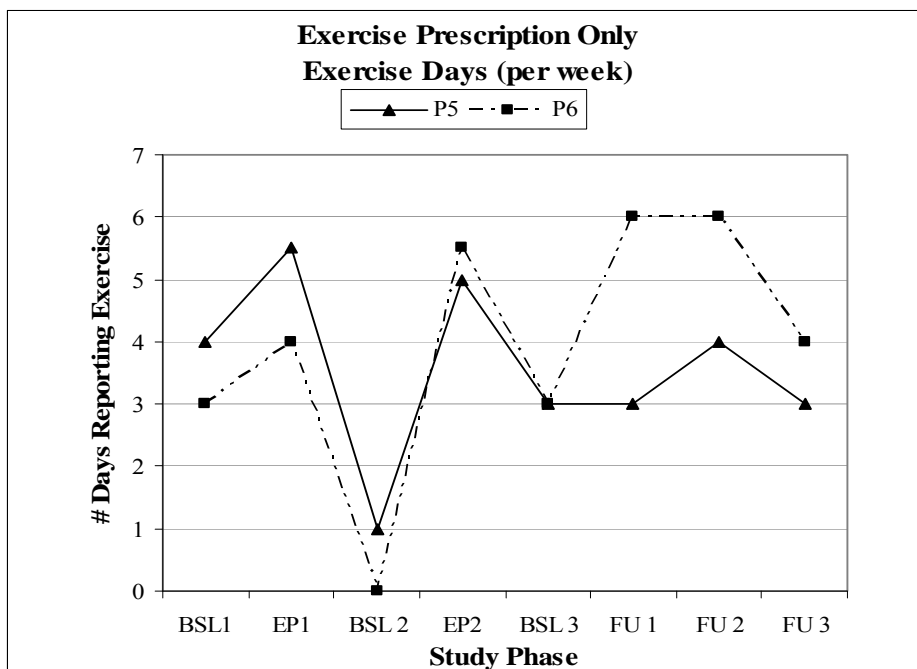


Figure 26. Exercise participation for EP only. Reported days of exercise for P5 (solid line) and P6 (dotted line), who completed the EP only treatment protocol. A total of 5 days of exercise were possible for baseline (BSL) recording periods, compared to 7 days possible for all other recording periods.

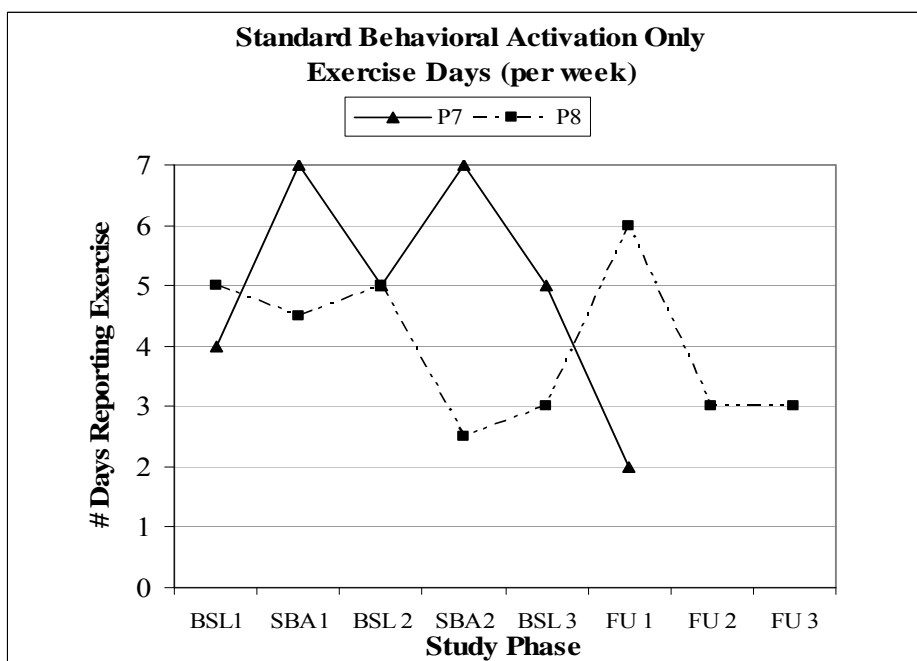


Figure 27. Exercise participation for SBA only. Reported days of exercise for P7 (solid line) and P8 (dotted line), who completed the SBA only treatment protocol. A total of 5 days of exercise were possible for baseline (BSL) recording periods, compared to 7 days possible for all other recording periods.

Exercise Participation by Treatment Type

	Days of Reported Exercise	# of Available Days	% of Exercise Participation
Baseline 1	26	40	65%
Exercise Prescription	81	112	72%
Standard Behavioral Activation	76	112	68%

Table 7. Percentage of total days of exercise by treatment type for all participants.

Body Mass Index

Body Mass Index (BMI) was used as a general measure of body composition in the current study. BMI calculations allow for broad classification of individuals into weight groups (see Table 8). All participants fell into overweight or obese categories. Four participants were in the “overweight” category (P2, P3, P4, and P6), three participants were classified in the “Class I Obesity” category (P5, P7, P8), and one participant fell into the “Class II Obesity” category (P1). P1 (EP to SBA crossover group) demonstrated the largest percentage decrease in BMI (i.e., 45 at baseline to 37.2 at 6-months follow-up; see Figure 28). All other participants’ BMI remained relatively constant through study participation. No significant changes in BMI were expected, given that a mild form of exercise was utilized for the EP protocol for a period of time likely insufficient to produce significant changes relative to overall body composition. Interestingly, P1 who demonstrated a classification change related to BMI reported engaging in a more moderate program of exercise participation (i.e., aerobic training in addition to prescribed exercise) which was maintained throughout study participation.

Weight Classification	BMI calculation (g/km²)
Underweight	< 18.5
Normal Weight	18.5 – 24.9
Overweight	25 – 29.9
Obesity (Class 1)	30 – 34.9
Obesity (Class 2)	35 – 39.9
Morbid Obesity (Class 3)	≥ 40

Table 8. Body Mass Index (BMI) classification table.

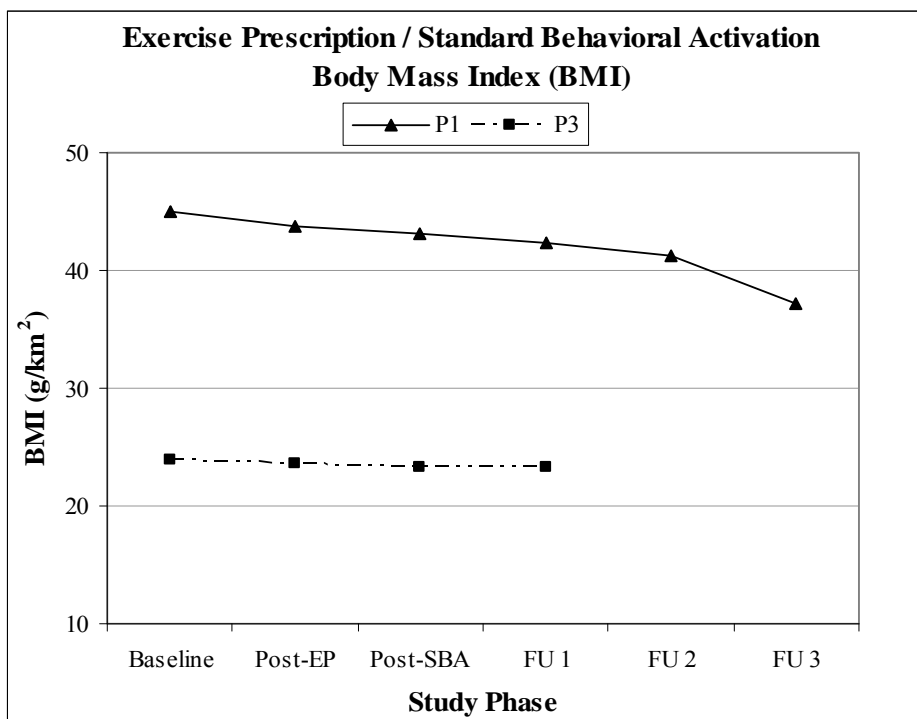


Figure 28. Body Mass Index (BMI) changes for EP/SBA crossover. BMI data for P1 (solid line) and P3 (dotted line).

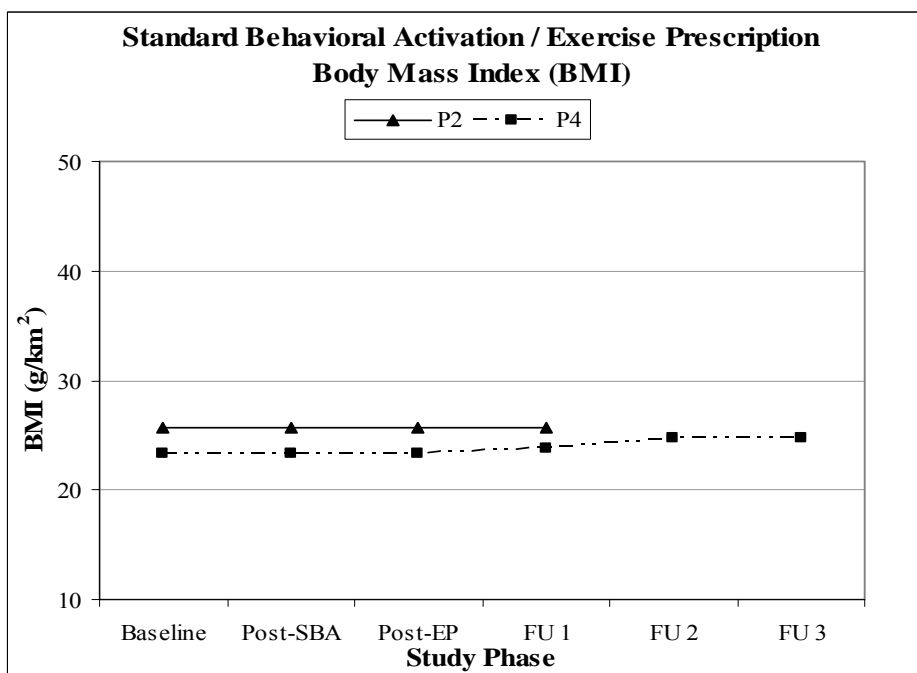


Figure 29. Body Mass Index (BMI) changes for SBA/EP crossover. BMI data for P2 (solid line) and P4 (dotted line).

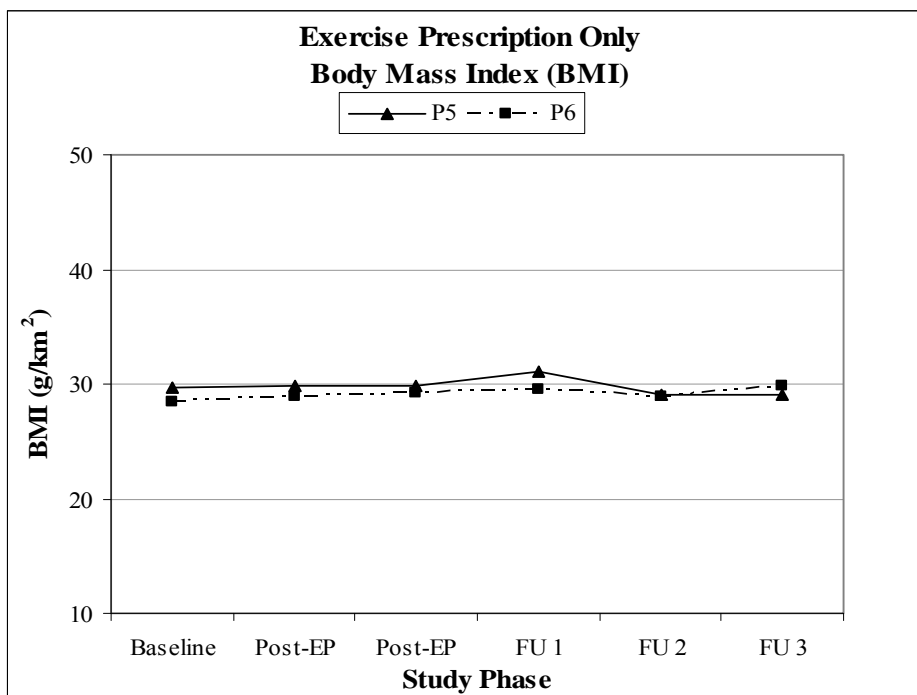


Figure 30. Body Mass Index (BMI) changes for EP only. BMI data for P5 (solid line) and P6 (dotted line).

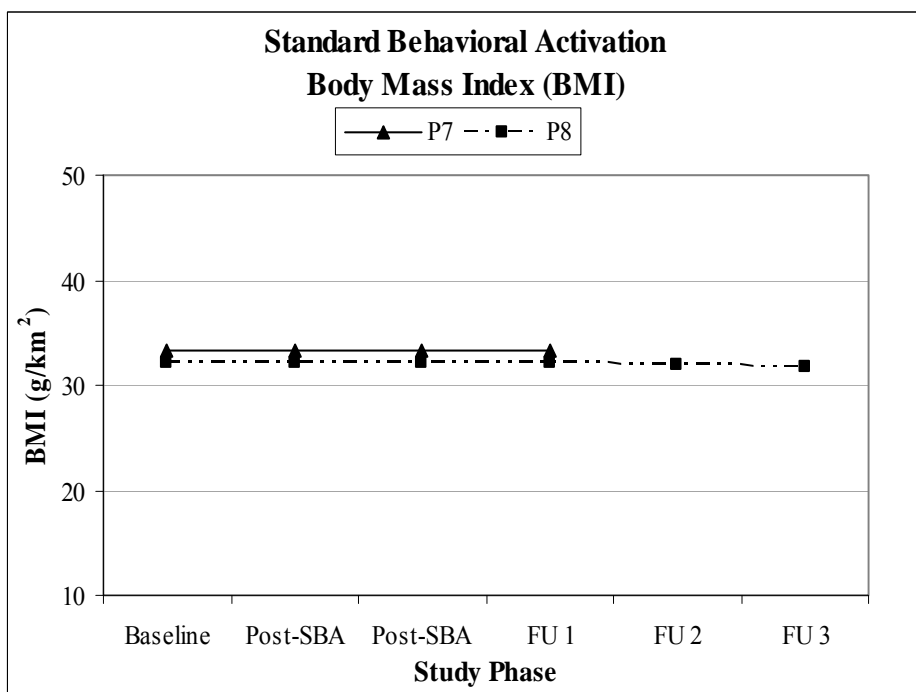


Figure 31. Body Mass Index (BMI) changes for SBA only. BMI data for P7 (solid line) and P8 (dotted line).

CHAPTER 4

DISCUSSION

The current investigation utilized a combined series, crossover (i.e., A/B/A/C/A, A/C/A/B/A) and interaction (i.e., A/B/A/B/A or A/C/A/C/A) single-participant methodology to investigate the effects of exercise on stress reactivity and mood changes in individuals with Bipolar Disorder (BD). Four possible treatment orders were included in the study: a) crossover from standard behavioral activation (SBA; e.g., reading) and exercise prescription (EP); b) crossover from EP to SBA; c) EP only; and d) SBA only. Eight individuals were enrolled in the study and two individuals were assigned to each of the four possible treatment conditions. All individuals completed three separate 5-day baseline recording periods (i.e., prior to first-order treatment, between treatment phases, following second-order treatment). Individuals were contacted for a 1-, 3-, and 6-month follow-up assessment of mood, perception of daily hassles and stress, and weekly exercise participation.

An interesting finding of the current study relates to rates of compliance. Although attrition rates in studies of individuals with BD are often reported as high as 50% or higher, individuals participating in the current study demonstrated a much more consistent level of participation. For the 43-day treatment and baseline recording protocol, 100% of participants completed study requirements. In addition, a high level of participation was maintained throughout follow-up periods, as well (i.e., 88% at 1-month follow-up and 63% compliance at 3- and 6-months follow-up). Although there is a sample selection bias inherent in all research designs, individuals in the current study verbally reported a high level of interest in learning more about the impact of exercise on

mood regulation. The high level of interest expressed by individuals screened for the current study in learning more about complimentary, non-pharmacological forms of assistance is notable. Attending to and building upon such interest in the clinical arena may improve compliance rates in the overall treatment of individuals with BD.

Another interesting finding from the current study was regarding the phenomenon of *hypercompliance* demonstrated by all participants. More specifically, individuals receiving a prescription for exercise participation reported engaging in greater than the expected amount of weekly exercise. In addition, those who did not receive the exercise prescription also initiated exercise participation at rates similar to those who did receive the prescription for exercise. This effect was seen immediately following enrollment into the study (i.e., at first baseline recording period), prior to initiation of any formal treatment included in the study (i.e., EP or SBA). This finding represents an exercise treatment effect at baseline, as this is the only period during the study during which exercise was newly initiated by any participant. Given that all participants reported similar levels of exercise throughout the remainder of the study, regardless of treatment condition, comparisons between EP and SBA during later phases of the study were difficult to make. It is possible that this hypercompliance phenomenon may be related to initiation of daily recording procedures, which were monitored closely by the experimenter, as all individuals (regardless of treatment condition) were asked to report on exercise participation (e.g., reporting whether exercise was a part of each day, reasons for exercise/no exercise, intensity and duration of exercise, type of exercise). Therefore, this finding may represent the utility of including a simple, accessible, and inexpensive process of daily monitoring exercise participation when working with individuals with

BD. This simple process may result in elective exercise participation, which in the current study was most correlated with depressed mood improvements and decreased reactivity to acute stressors.

Exercise is widely accepted as an accessible treatment, with fewer negative side-effects than some psychopharmacological interventions (particularly mood stabilizers and antidepressants). Given the historical focus on biological mechanisms and pharmacological treatments for Bipolar Disorder, it is possible that individuals who have been exposed to the traditional course of treatment for this psychiatric illness, and associated side-effects (e.g., weight gain), may view adjunct treatments such as exercise as appealing. Given the mild rise in reported manic symptoms following initial onset of exercise participation for individuals in the current study, and the lack of understanding of how mood stabilizing medication (e.g., lithium) is affected by sweat rates during exercise, it is possible that exercise participation may induce unwanted shifts (i.e., elevations) of mood. Therefore, it is important that professionals working with individuals with BD closely monitor exercise participation, whether prescribed or elective. In addition, more research is needed to address dose-response relationship between exercise and mood stability, as well as the impact of exercise on neurochemical functioning. Although findings from the current study show an interesting trend of improved depression ratings (i.e., BDI-II scores) for individuals engaging in elective exercise, no clear dose-response relationship was found.

Regarding stress reactivity, results from the current study suggest that prolonged participation in a self-initiated program of exercise (i.e., in absence of a formal prescription of exercise) may be most important for decreasing an individual's perception

of reactivity to an acute laboratory stress task. Whereas individuals participating in the EP only treatment condition exhibited a slight increase in depressed mood to the stress task, those in the SBA only condition demonstrated the largest decrease in depressed mood. These findings are consistent with previous research, which suggests that participation in a program of exercise that involves a chosen regimen of exercise (i.e., preferred versus prescribed) may be most effective regarding improvement of both physiological and psychological outcomes (Parfitt, Rose, & Markland, 2000).

Regarding objectively monitored reactivity to the same stressful task (i.e., frequency of significant skin conductance responses; SCR) collected during the stress task), it appears that receiving a prescription for exercise participation is important. Although individuals receiving the EP only demonstrated an initial increase in SCR in response to the stress task following 2 weeks of EP, these individuals' SCR following the stress task after 4 weeks of EP returned to levels at or below baseline responses. Conversely, individuals receiving the SBA only intervention showed an initial decrease in SCR responses, with an increase to levels similar to those seen at baseline. Although inconclusive, these findings may suggest that individuals engaging in a prescribed and monitored program of exercise may be protected from the acute effects of stress over time. Given that individuals completed the SBA only intervention engaged in a similar level of exercise, although electively initiated, findings suggests that participation in multiple forms of behavioral activation, including a mild exercise, may also be useful in decreasing physiological response to an acute stressor.

In general, it appears that receiving a prescription to initiate a program of exercise, which is monitored by a professional, may be effective in reducing an

individual's objective physiological reactivity to an acute stressor following a period of exercise habituation. More specifically, this process may play an important role in regulating stress reactivity and mood stability in a population of individuals with BD. However, it is unclear to what extent habituation to the stressful task itself may have played in this pattern of responding, as the same stressful task was used at each of the three laboratory sessions. However, this explanation seems unlikely, given the amount of time (i.e., 14-20 days) between each session.

When considering more subjective experiences (i.e., depressed mood and anxiety) of stress reactivity, it appears that receiving a prescription for mild to moderate exercise may play a less important role. Individuals receiving 4-weeks of EP did, however, demonstrate a fairly stable pattern of responding regarding transitory mood states in response to the stressful laboratory task. Individuals engaging in an elective program of exercise, and a prescribed regimen of non-exercise behavioral activation (e.g., reading), demonstrated a worsening of symptoms of transitory depressed mood and anxiety following 2 weeks of treatment. Interestingly, with continued elective exercise participation, paired with SBA, both of these participants (P7 and P8) demonstrated a return to low-level symptoms of depressed mood and anxiety following the stress task. These findings are inconclusive, and suggests that while some individuals may require a prescribed and monitored program of exercise to see an impact on functioning, other individuals with BD may benefit most from self-initiation (and maintenance) of a program of exercise in the absence of a monitored prescription. More research is needed to determine what features predict response to these differing treatment approaches.

Overall, it appears that prolonged participation in a program of exercise is correlated with a reduction in both physiological responding to an acute stressor, as well as with perception of daily hassles. This finding was expected, and may be particularly important, given the established relationship between perception of life stress and mood episode relapse in individuals with BD (e.g., Ehlers, Kupfer, Frank, & Monk, 1993; Miklowitz & Goldstein, 1997).

Regarding the impact of exercise on the report of depression symptom severity, interestingly, all participants showed a similar (i.e., slope of lines) decrease in BDI-II following 4-weeks of treatment, regardless of treatment order (see Figure 15). Of note, some participants (e.g., P5) reported an initial baseline BDI-II score that did not allow for significant change due to floor effects. Given that all participants reported participating in similar levels of exercise, regardless of treatment type received, this finding suggests that exercise (in general) may be effective in improving symptoms of depressed mood for individuals with BD reporting more severe symptom experiences consistent with behavioral theories regarding treatment of unipolar depression (e.g., Lewinsohn & Gotlib, 1995; Lewinsohn et al., 1985; Martell et al., 2001). It is also possible, however, that these findings may represent a simple regression to the mean of BDI-II scores, given historical evidence that symptoms of depression remit over time, with or without treatment. Given that difference scores in BDI-II responses between baseline and post-intervention were most significantly correlated with elective exercise participation, it appears that previous findings that exercise may help to reduce symptoms of depressed mood in both clinical and sub-clinical forms of unipolar depression may also apply to individuals experiencing depressive symptoms in the context of bipolar illness. More

research is needed to determine a dose-response relationship between exercise and depressed mood in BD samples, as well as to determine the impact of sweat-rate on medication levels, and potential for manic-symptom induction, in these individuals.

In general, it appears that prolonged, elective participation in a program of mild to moderate exercise results in the greatest stability of low-level depressive symptoms in this sample of individuals with BD. Individuals receiving a prescription of exercise during the first 2-weeks of treatment, who independently maintained participation over time, reported the largest decrease in the experience of depression symptom severity following completion of the second-order treatment (SBA). This finding is consistent with previous research, which suggests that participation in preferred forms of exercise (e.g., preferred intensity) may have the most impact on both psychological and physiological benefits (e.g., Parfitt et al., 2000). However, these individuals did report a worsening of depression after 2-weeks of participation in a level of exercise that exceeded (i.e., greater intensity, longer bouts of exercise) that prescribed in the protocol. According to the Salmon (2001) stress-adaptation hypothesis, these findings are expected, as it would take a greater period of time (i.e., 4 versus 2 weeks) to habituate to a higher level of exercise participation. The stress-adaptation hypothesis was generally supported for even the most chronically ill individuals (i.e., those with the most chronic BD presentation, and the greatest number of comorbid illnesses) enrolled in the study. Given the aforementioned correlation between elective exercise participation and decreased depression scores, it is possible that exercise may be most useful for individuals experiencing more severe symptoms of depressed mood.

These findings are further supported with long-term follow-up data, which suggests that a change (decrease) in frequency of exercise participation resulted in a greater level of depression symptom severity. This finding was particularly true for individuals receiving the greatest amount of prescribed exercise (i.e., 4-weeks in the EP only intervention) during the treatment phase of the study. However, in the face of a significantly increased level of life stress (i.e., P4, who reported sustained unemployment and financial distress), exercise levels decreased, and simultaneous worsening of depression was reported at 6-months follow-up. Overall, these finding lends greater support for the impact of habituation to a particular level of exercise on improvements in level of depression symptom experience. However, more research is needed to determine an adequate dose-response relationship with varying levels of life stress.

In conjunction with established evidence of an antidepressant effect of exercise in individuals with unipolar depression (e.g., Babyak et al., 2000; Blumenthal et al., 1999; Dunn et al., 2002; Dunn et al., 2005), these findings appear to support the hypothesis that exercise may also have mood stabilizing properties when used as an adjunct treatment for individuals with BD; especially considering the frequency with which these individuals experience depressive versus manic mood shifts. More research, however, is needed to support this hypothesis.

Regarding symptoms of mania, all participants reported maintenance of a low-level of manic symptom severity through completion of the study, including follow-up. The lowest level of manic symptom expression was observed following 4-weeks of treatment completion for all participants, regardless of treatment condition. For individuals receiving EP as a first-order, or sole treatment condition, a mild increase in

manic symptom endorsement was observed after 2-weeks of exercise participation. This finding supports the defense-defeat theory of response to psychosocial stressors (Folkow, 1993), which holds that there is a “defense” reaction (i.e., behavioral activation) in response to psychosocial stressors. Interestingly, prolonged participation in a program of exercise (prescribed or self-maintained) resulted in a decrease in manic symptom endorsement to a level at or below that reported at initial baseline; supporting a habituation to the “stressor” (i.e., exercise), consistent with Salmon’s (2001) stress-adaptation theory proposed to explain the antidepressant effect of exercise. No episodes of manic episode relapse were reported through 6-months follow-up. Although additional research is needed, these preliminary findings suggest that the impact of exercise habituation on stress reactivity may be important for maintaining mood stability for prolonged periods of time, particularly as it relates to the manic phases of BD.

In addition to maintenance of more stable mood, it appears that exercise may play an important role related to how individuals with BD cope with life circumstances. The greatest improvement in use of adaptive coping strategies was found in those individuals participating in the SBA to EP crossover treatment. These individuals self-initiated a program of exercise during the initial treatment phase of the study, and later received a prescription for exercise participation. All individuals receiving the crossover treatment protocols, regardless of order of presentation, reported a related decrease in maladaptive coping strategy use as adaptive strategy employment increased. Individuals receiving 4-weeks of a treatment (i.e., EP or SBA only) reported a decrease in the use of adaptive coping strategies during the 4-week treatment protocol, with stable use of maladaptive strategies. It appears that participating in a prescribed program of exercise for 4-weeks,

and subsequent independent maintenance of this program, may result in a longer-term stabilization in the use of adaptive coping strategies. In addition, altering the program of exercise to which these individuals had habituated resulted in a related decrease in the use of adaptive and increased use of maladaptive coping strategies at 6-months follow-up. This finding suggests that individuals may need more frequent monitoring of their participation in a voluntary program of exercise to experience the greatest sustained improvement on the use of adaptive versus maladaptive coping strategies. In addition, these findings suggest that participation in multiple forms of behavioral activation, including a prescribed regimen of exercise, may be most useful in enhancing long-term adaptive coping strategy use.

Regarding the perception of daily hassles, predictions regarding exercise participation and reported experiences of stressful daily hassles were not fully supported in the current study. However, it is interesting to note that individuals receiving the EP only intervention reported the lowest levels of daily hassles throughout participation in the study, and the most consistent pattern of responding. In contrast, individuals in the SBA only intervention reported the most inconsistent or erratic experience of daily hassles during the treatment component of the study. At follow-up assessments, the pattern of reporting daily hassles for individuals in the SBA group became more consistent, although the trend was to report more experiences of stressful daily hassles. Although these individuals did not receive a prescription of exercise, each reported regular involvement in a self-initiated exercise program.

These findings support the prediction that participating in a prescribed and monitored program of exercise may be most important regarding stabilizing an

individual's perception of daily life hassles over time. However, it should be noted that this treatment program appears to be less important in changing an individual's perception of general life stress (i.e., daily stress ratings) or of more acute daily stressors (i.e., acute laboratory stress task) over time. Furthermore, individuals who did not receive a prescription for exercise appeared to demonstrate an increase in perceived daily hassles at follow-up compared to their study counterparts. This finding suggests that participation in a prescribed and monitored program of exercise that is consistent with the recommended guidelines for exercise participation (i.e., ACSM, 1995; Pate et al., 1995) may be most beneficial in effecting long-term benefits relative to perception of daily hassles.

These findings are also consistent with previous research that suggests that mood episode relapse in BD is triggered by disruption in daily routine (e.g., Ehlers et al., 1988). In addition, these findings lend support for the hypothesis that exercise may enhance routine regulation; consistent with current conceptualizations of behavioral activation (BA) interventions that focus on assisting individuals with the establishment and maintenance of a regular routine. Furthermore, repeated exposure to uncontrollable (unexpected) stressors is thought to result in the manifestation of a resistance to stress (i.e., habituation to the stressor; Salmon, 2001), and achievement of adaptation more quickly with regular exposure to a controllable stressor (Maier & Seligman, 1976; Weiss & Glazer, 1975). More efficient habituation, and associated stress adaptation, may result in a larger long-term benefit regarding decreasing reactivity to stressful daily hassles.

Findings from the current study are consistent with the established literature, which suggests that regular exercise may provide a more structured life system for

individuals, thereby reducing vulnerability to stress (Cramer, Nieman, & Lee, 1991; Crews & Landers, 1987; Salmon, 2001) and risk of relapse. Regular exercise has been consistently shown to reduce anxiety, depression, and stress for individuals of all ages and both genders, and is also thought to increase life structure and decrease stress reactivity in individuals with mood regulation difficulties. These findings have contributed to the current conceptualization regarding “gold standard” treatment approaches for individuals with BD; which currently include a combination of psychopharmacological treatments with a validated psychotherapeutic approach (e.g., FFT, IPSRT, or CBT).

Recent findings from the STEP-BD research program have suggested that mood episode relapse in Type I Bipolar disorder is predicted by the presence of interepisode residual mood symptom (Perlis, Ostacher, Patel, Marangel, Zhang, Wisniewski, et al., 2006). Additional findings from the STEP-BD projects suggest that inclusion of psychotherapy may help to improve treatment outcome (e.g., improve psychosocial functioning) in individuals with moderate to severe presentations of BD (Miklowitz, Otto, Wisniewski, Agra, Frank, Reilly-Harrington, Lembke, et al., 2006). Given findings that exercise may be most effective in treating mild to moderate symptoms of affective distress (e.g., Dunn, 2002; Dunn et al., 2005; Wyshak, 2001), it is possible that incorporating exercise into an integrated treatment approach for BD may target reduction or omission of residual symptom presence, thereby reducing an individual’s risk of relapse. Further, regular exercise has also been associated with lower perceived stress in daily living (Stephoe, Lipsey, & Wardle, 1998). Therefore, it seems likely that including

prescribed exercise as an adjunct to gold standard treatments for BD may further reduce the risk of relapse in this population.

Given the current findings related to compliance in this sample of individuals with BD, including exercise may also assist with improving rates of compliance with concurrent therapies, which have consistently been shown to be an area of concern in this population (e.g., Miklowitz, 2001). An integrated treatment approach, which combines gold standard treatments with regular exercise, may represent a more comprehensive and cost-effective intervention. Overall, this type of treatment approach for BD may enhance relapse prevention efforts by assisting individuals to develop more structured daily routines, thereby improving compliance with concurrent therapies, and decreasing stress reactivity and ultimately, vulnerability for relapse.

Limitations of the Study

Although single-participant research designs have an extensive history in the behavioral sciences, and it has been argued that preliminary research of treatment effectiveness should utilize these designs prior to large group designs, there are inherent limitations associated with these methodologies. Generalizability of findings from small *n* research designs is difficult due to factors such as heterogeneity of participants. Whereas large-scale studies allow for greater power to minimize the impact of general heterogeneity of participants, these differences cannot be easily ruled out as contributors to overall outcome in single-participant research. However, replication of single-participant research, which can be reviewed and compiled into a larger body of data, may also be a useful future direction.

Order effects, resulting in methodological limitations, are inherently present in crossover treatment designs. More specifically, it is unclear whether effects demonstrated at the end of the second-order treatment are a result of that unique treatment, or whether changes are a result of combined treatment effects. Therefore, conclusions for crossover treatment components of this study can only be drawn regarding the effect of the first-order treatment and the effect of the two treatments combined. As a consequence, interpretations regarding the effect of standard behavioral activation (for P1 and P3) and exercise prescription (P2 and P4) cannot be made. However, given the substantial evidence that supports standard forms of behavioral activation as effective methods for alleviating symptoms of depressed mood, the crossover treatment design is ideal for comparing the effect of exercise on stress reactivity and mood changes compared to an already validated approach in individuals with mood disorders. Given the aforementioned limitations of crossover treatment designs, however, the current study included two interaction treatment orders. Individuals received either 4-weeks of EP or SBA, only, so that the effect of exercise alone could be determined for a subset of individuals with BD.

An additional limitation of the study is related to standardization of the exercise prescription protocol. Although individuals were asked to engage in a consistent amount of exercise (i.e., 4 separate 30-minute sessions of walking per week of exercise treatment), no objective standardization of intensity, duration, or frequency was possible. Therefore, the results are reliant upon individuals' report of exercise participation. Although all participants reported engaging in the minimum prescribed amount of weekly exercise, some reported exceeding the "dose" of exercise during the treatment protocol.

Although pedometers were used as an objective source of monitoring increased exercise participation, daily step count recordings were made by participants. Unfortunately, there is no way to validate participants' reports of exercise participation as individuals were expected to exercise outside of a controlled laboratory setting. Due to practical considerations (e.g., time, participant burden, equipment availability), a more standardized system of weekly exercise participation was not utilized. Future research, utilizing standardized (i.e., laboratory treadmill exercise utilizing VO₂ maximal testing to determine intensity of exercise for participants), is needed.

Additional limitations of the study are related to differing levels of life stress and psychiatric comorbidity seen between participants. Given the relationship between life stress and illness in Bipolar Disorder, this variable was not controlled for during study recruitment and enrollment. Further, given the high rates of Axis I psychiatric comorbidity in this population, diagnostic profile was not restricted in the current study, with the exception of excluding individuals meeting criteria for thought disorders, psychosis outside of the context of mood episodes, and current alcohol or substance dependence. Although having a more inclusive approach to study inclusion allowed for the study of a more 'natural' sample of individuals with BD, this makes it difficult to rule out extraneous variables that may impact study outcome. Future research investigating exercise as an adjunct treatment in a more 'pure' sample of individuals with BD is needed to enhance generalizability of findings.

In addition to the aforementioned limitations, the current study could have benefited from utilizing more objective strategies to monitor adherence to treatment expectation. The current protocol relied on participants written self-report of daily

exercise, or other behavioral activation, participation. Validation of the accuracy of reporting was not possible. Research utilizing laboratory controlled treatment protocols, and more standardized methods of monitoring of treatment adherence is needed. More standardized, well-controlled methods of monitoring would allow for greater certainty regarding the impact of treatment on outcome variables (e.g., mood, stress reactivity).

Conclusions

In general, findings from the current study suggest that elective exercise participation may be most important regarding improvements in mood, or maintenance of stabilized mood over time, for individuals with BD. Given the observed *hypercompliance* phenomenon seen in the current study, it appears that simple monitoring of exercise participation may be strongly linked to self-initiation of exercise. All individuals enrolled in the current study were sedentary prior to initial baseline, and subsequently reported onset of elective exercise participation following initiation of daily monitoring procedures. Therefore, it appears that the baseline monitoring period served as a treatment in the current study. This finding suggests that greater monitoring of exercise participation may result in elective exercise performance, and further calls for involvement of behavioral treatment providers (e.g., psychologists) in the treatment of individuals with BD.

Overall, it appears that prolonged, elective participation in mild to moderate intensity exercise is most important in reducing an individual's perception of stress reactivity to an acute stressor. Participation in multiple forms of behavioral activation (e.g., pleasant activity scheduling in addition to exercise) may be most effective in decreasing an individual's perception of stress reactivity. However, it appears that

participation in a prescription program of exercise is most effective in reducing underlying physiological response to more acute stressors. In addition, engagement in multiple forms of behavioral activation, including prescribed and monitored exercise, may result in a positive change in individuals' use of adaptive versus maladaptive coping strategies. Participation in a program of exercise for a longer (i.e., 4 weeks) period of time appeared to provide the most benefit, consistent with exercise habituation theories.

Overall, findings from the current investigation suggest that it may be most helpful to include exercise-specific forms of behavioral activation (i.e., a monitored prescription of mild to moderate intensity exercise 3-4 days per week) when working with individuals with BD. In addition, rates of compliance that were higher than expected (i.e., 100% through treatment, 88% at 1-month follow-up and 63% compliance at 3- and 6-months follow-up) suggest that including exercise as an adjunct treatment may provide some benefit regarding overall treatment compliance. This issue is frequently cited as a significant area of concern when working with individuals with BD. Including these components in the conceptualization of a treatment approach for individuals with BD may be the most effective method of decreasing stress perception and responding, improving availability of and perceived use of adaptive versus problematic coping strategies. In general, current findings provide preliminary support for the prophylactic benefits of including regular monitoring of exercise participation, as well as exercise prescription, as an adjunct treatment for individuals with BD.

Future Directions

Although current findings suggest that exercise may be an important component to treatment for individuals with BD, the dose-response relationship relative to exercise,

stress reactivity, and mood stability remains unclear. In addition, the current study was unable to control for level of intensity chosen by participants during bouts of exercise. Larger, randomized controlled studies of exercise (e.g., standardized intensity, frequency, and duration in controlled setting) as an adjunct treatment for BD is needed so other mechanisms of change regarding mood stabilization and stress reactivity may be ruled out.

Research is needed to further explore the role of exercise participation in impacting physiological stress responses in individuals with BD. For example, larger scale studies investigating changes in blood pressure, respiratory rate, skin conductance, heart rate, and cortisol may assist in furthering our understanding of the impact of exercise participation on basic physiological responses to acutely stressful situations. In addition, this line of research may further our understanding of individual responses to psychosocial stressors. Such research may help to clarify the impact of exercise on behavioral activation versus habituation in individuals with BD. These issues are important to consider regarding how to incorporate exercise as an adjunct treatment for BD (e.g., intensity and frequency of exercise).

Similarly, research utilizing rigorous methodology investigating the impact of varying intensities of exercise participation (e.g., mild, moderate) on medications levels in individuals with BD is needed. Given that one of the most common psychopharmacological treatments for BD is lithium carbonate (i.e., which is effectively a salt and impacts the flow of sodium through the body), vigorous forms of exercise that result in high sweat rates (and thereby loss of sodium in the body) may require lithium

supplementation. This dose-response relationship is important to understand so that a safe and more effective prescription of exercise can be determined.

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APPENDIX A

Brief Screening Questionnaire

Age: _____ Gender: _____ Years of Education: _____

1. 1. Do you feel that you currently suffer from depression? YES NO

If you answered YES, in the past month...

- a) Have you felt depressed or down for most of the day nearly every day, for at least 2 weeks?

YES NO

- b) Have you lost interest or pleasure in things you usually enjoy, for at least 2 weeks?

YES NO

2. Have you ever had a period of 2 weeks or more that you:
a. Felt depressed or down for most of the day nearly every day, for at least 2 weeks?

YES NO

Lost interest or pleasure in things you usually enjoy, for at least 2 weeks?

YES NO

- b. Lost interest or pleasure in things you usually enjoy, for at least 2 weeks?

YES NO

3. In the **last month**, has there been a period of time when you felt so “high”, excited, or hyper that others thought you were not your normal self, or you got into trouble?

YES NO

If YES in the past month...

- a) How long did that period of time last? _____

4. Have you ever had a period of time when you felt so “high,” excited, or hyper that others thought that you were not your normal self, or you got into trouble?

YES NO

If YES. . .

- a) How long did that period of time last? _____

5. Are you currently taking any medications prescribed for a psychiatric disorder? YES NO

Optional – If YES, which one(s)?

6. Has there ever been a time when you've had five or more drinks on one occasion?

YES NO

7. Have you ever used recreational drugs? YES NO

8. Have you ever been "hooked" on a prescribed medicine or taken a lot more than you were supposed to?

YES NO

9. Has it ever seemed like people were talking about you or taking special notice of you?

YES NO

10. Did you ever hear things that other people couldn't hear (e.g., noises, voices of people whispering or talking)?

YES NO

11. On average, how many days per week do you exercise for 30 minutes or more? _____

12. On average, how many days per week do you walk for 30 minutes or more? _____

13. Do you lift weights? YES NO

If yes...

How many days per week, on average? _____

APPENDIX B

The Role of Exercise and Stress in Bipolar Disorder **Informed Consent Form**

Because you are at least 18 years old, and responded to recruitment efforts for an “Exercise, Stress, and Mood Project,” you are being asked to participate in this study. The principal investigator for this study is Teresa Edenfield, a doctoral student in Psychology. Sandra Sigmon, Ph.D., a professor in the Psychology Department, is supervising this project. The purpose of this study is to gain more information about the relation between exercise and mood disorders (i.e., Bipolar Disorder). Your participation in this study will help further the understanding of different ways to help individuals cope with particular mood disorders (i.e., Bipolar Disorder). Also, by participating in this study, you will help us learn more about ways that may help decrease the risk of relapse in mood disorders such as Bipolar Disorder.

What will you be asked to do?

1. First lab visit (355 North Stevens Hall):

- You will be asked to answer questions to see whether or not you will be able to participate in the study. You will be asked to answer questions about:
 - Your mood (e.g., *“In the past month, has there been a period of time that you felt so good, ‘high,’ excited, or hyper that others thought you were not your normal self?”*)
 - Alcohol and substance use (e.g., *“Has there been a time in your life when you had five or more drinks on one occasion?”*; *“Have you ever used street drugs?”*)
 - Bizarre experiences that people sometimes have (e.g., *“Did you ever hear things that other people couldn’t hear?”*)
 - Anxiety (e.g., *“In the past six months, have you felt particularly nervous or anxious?”*)
 - This interview will take about one hour to finish.
- If you qualify for the study, you will be asked to count backwards while we measure your skin conductance (how much your palm sweats). This will take about 10 minutes.
- After the counting task, you will be asked some questions about:
 - Some stressful life events (e.g., *“How much has disliking your daily activities been part of your daily life in the past month?”*) and how you handle them (e.g., *“I’ve been giving up trying to deal with it.”*)
 - Exercise (e.g., *“Did you exercise today?”*) and mood
- **Daily Recording:**
 - During the rest of the study, you will be asked to record information about activity, mood, stress, sleep, and medications you take for a total of 43 days. The experimenter will explain how to do this.
 - The first lab visit will take about 2 hours to complete.

2. Treatment phases of the study

- During two different 2-week periods, you will be asked to either:
 - Walk for 30 minutes a day on 4 separate days a week for 2 weeks, or
 - Do another kind of activity that is *not* exercise (like read or write in a journal) for the same amount of time

3. Second and Third lab visits (355 North Stevens Hall)

- The second and third lab visits will last approximately 45 minutes each. They will be the same as the first visit (e.g., questionnaires, counting task), except the interview will not need to be completed again.
4. Follow-up contacts:
- You will be contacted 3 times (1-, 3-, and 6-months) after the last visit to the lab to answer questions about your mood and activity level over the past month. You will mail the questionnaires back to the experimenter in the pre-postage-paid envelope she gives you. Answering these questions will take about 7 minutes each time.
5. *A table of study guidelines and time commitment is provided on the last page of this form.*

Benefits

Your responses will tell us more about the relation between exercise and mood disorders (e.g., Bipolar Disorder). Learning more about how regular exercise affects the course of Bipolar Disorder (e.g., may decrease risk of relapse) could lead to better treatments for people with this type of mood disorder. You will be paid for participation in the study: \$10 for first lab visit; \$1 per day of daily monitoring completed (total of 43 days, and possible \$43 for daily monitoring); \$5 for second lab visit; \$5 for third lab visit. All participants will be paid for completing the follow-up contacts: \$5 for each follow-up contact (total of \$15 possible). It is possible to earn a total of \$78, if each step of the project is finished.

Risks

The risks associated with the project are minimal. However, if you experience any distress associated with completing questionnaires, the experimental tasks, or the treatment phases, please contact the experimenter. You do not have to answer any questions that make you feel uncomfortable and you may withdraw from the study at any point. You will be compensated for your participation up to that point. Teresa Edenfield (207-581-2063) and Dr. Sandra Sigmon (207-581-2049), a licensed psychologist, may also be contacted at any time during the study at this address: 301-B Little Hall, Orono, ME 04469. Also, referral information for counseling services is provided below. If you have any questions about your rights as a research participant, please contact Gayle Anderson at Research and Sponsored Programs: (207) 581-1498, 415 Corbett Hall, Orono, ME 04469.

Counseling Referral Information:

Peter Ippoliti, Ph.D. (207) 942-8200
 Dr. Lucy Quimby, Ph.D. (207) 945-3675
 Psychological Services Center (207) 581-2034

These are choices and in no way do they reflect an endorsement by the University of Maine. The Psychological Services Center operates on a sliding fee scale that is based on your income, and the two psychologists listed charge by the hour.

Confidentiality

Identifying information will not be shared with anyone. Your responses will be stored in the principal investigator's locked laboratory. A code number will be used to protect your identity. The key linking your name to the data will be destroyed after the data have been entered on the computer. If the study is published or presented, only information based upon the entire group of participants will be used. Your data, without identifying information, will be stored indefinitely in the principal investigator's locked lab.

Your signature below indicates that you have read and understand the information stated above.
You will receive a copy of this form.

Signature: _____

Print name here: _____

Date: _____

Phone Number: _____

E-mail address: _____

Please write an address that you can be contacted at for follow-up parts of the study:

Would you like to see a summary of results of this study? _____ **YES** _____ **NO**

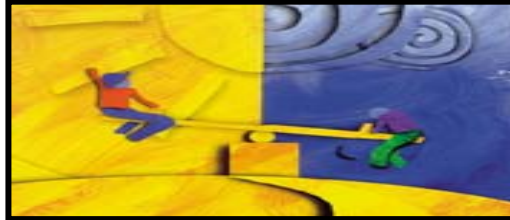
Study time-line:

Activity:	What will I do?	How long will it take?	Benefit (\$78 possible):
Lab visit #1 355 N. Stevens Hall	Interview Mental counting task Answer some written questions Learn about the first treatment of the study and daily monitoring Activities	Total time ~ 2 hours	\$10 for completion
Baseline Recording #1	Answer written questions	About 2 min. for 5 days	\$5 for completion
Treatment phase #1	Walking or another non-exercise activity	Total = 2 weeks 4 days (8 times) of walking or other Activity 30 minutes per time on each day Daily forms for 14 days	\$14 for completion
Lab visit #2 355 N. Stevens Hall	Mental counting task Answer some written questions Learn about the second treatment of the study and daily monitoring Activities	Total time ~ 45 minutes	\$5 for completion
Baseline Recording #2	Answer written questions	About 2 min. for 5 days	\$5 for completion
Treatment phase #2	Walking or another non-exercise activity	Total = 2 weeks 4 days (8 times) of walking or other Activity 30 minutes per time on each day Daily forms for 14 days	\$14 for completion
Lab visit #3 355 N. Stevens Hall	Mental counting task Answer some written questions Learn about the first treatment of the study and daily monitoring Activities	Total time ~ 45 minutes	\$5 for completion
Baseline Recording #3	Answer written questions	About 2 min. for 5 days	\$5 for completion
Follow-up #1 (1-month after end of baseline #3)	Respond to written questions Mail the forms back to experimenter	5 minutes	\$5 for completion
Follow-up #2 (3-months after end of second visit)	Respond to written questions Mail the forms back to experimenter	5 minutes	\$5 for completion
Follow-up #3 (6-months after end of second visit)	Respond to written questions Mail the forms back to experimenter	5 minutes	\$5 for completion

APPENDIX C

Recruitment Flyer

Exercise, Stress, & Mood



Have you ever felt so good, excited, or hyper (for 1 week or more) that..

- Friends or family might have told you that you were not yourself
- You might have gotten into trouble, made impulsive decisions, or engaged in risky behaviors?
- You were diagnosed with Bipolar Disorder?

If you answered **YES** to these questions, you may qualify to participate in a study investigating activity level and mood changes. The study is being conducted by Teresa Edenfield, a Psychology graduate student at the University of Maine. Participants can earn up to \$78 for completing the study.

If you are 18-years-old or older and are interested in participating, call **Teresa Edenfield** at the University of Maine (207-581-2063), or e-mail her at Teresa.Edenfield@umit.maine.edu.

APPENDIX D

The MDQ

1. Has there <u>ever</u> been a period of time when you were not your usual self and...	YES	NO
...you felt so good or hyper that you were not your normal self or you were so hyper that you got into trouble?		
...you were so irritable that you shouted at people or started fights or arguments?		
...you felt much more self-confident than usual?		
...you got much less sleep than usual and found you didn't really miss it?		
...you were much more talkative or spoke faster than usual?		
...thoughts raced through your head or you couldn't slow your mind down?		
...you were so easily distracted by things around you that you had trouble concentrating or staying on track?		
...you had much more energy than usual?		
...you were much more active or did many more things than usual?		
...you were much more social or outgoing than usual, for example, you telephoned friends in the middle of the night?		
...you were much more interested in sex than usual?		
...you did things that were unusual for you or that other people might have thought were excessive, foolish, or risky?		
...spending money got you or your family in trouble?		
<p>2. If you checked YES to more than one of the above, have several of these happened during the same period of time?</p> <p><i>Please circle ONE response only.</i></p> <div style="display: flex; justify-content: space-around; margin-top: 10px;"> YES NO </div>		
<p>3. How much of a problem did any of these cause you -- like being unable to work, having family, money or legal troubles; getting into arguments or fights?</p> <p><i>Please circle ONE response only.</i></p> <div style="display: flex; justify-content: space-around; margin-top: 10px;"> No Problem Minor Problem Moderate Problem Serious Problem </div>		

APPENDIX E

PAR-Q

Please read the following questions carefully and answer each one honestly. Check YES or NO for each question.

YES

NO

☐
☐

1.) Has your doctor ever said that you have a heart condition AND that you should only do physical activity recommended by a doctor?

☐
☐

2.) Do you feel pain in your chest when you do physical activity?

☐
☐

3.) In the past month, have you had chest pain when you were not doing physical activity?

☐
☐

4.) Do you lose your balance because of dizziness or do you ever lose consciousness?

☐
☐

5.) Do you have a bone or joint problem (for example, back, knee, or hip) that could be made worse by a change in your physical activity?

☐
☐

6.) Is your doctor currently prescribing drugs (for example, water pills) for you blood pressure or heart condition?

☐
☐

7.) Do you know of *any other reason* why you should not do physical activity?

If YES to item 7, please list:

APPENDIX F

ASRM

Date: _____

Instructions

1. On this questionnaire are groups of five statements; read each group of statements carefully.
 2. Choose the **one** statement in each group that best describes the way you have been feeling for the **past week**.
 3. Circle the number next to the statement you picked.
 4. **Please note:** The word “*occasionally*” when used here means once or twice; “*often*” means several times or more, “*frequently*” means most of the time.
-

- 1) 0 I do not feel happier or more cheerful than usual.
 1 I occasionally feel happier or more cheerful than usual.
 2 I often feel happier or more cheerful than usual.
 3 I feel happier or more cheerful than usual most of the time.
 4 I feel happier or more cheerful than usual all of the time.

- 2) 0 I do not feel more self-confident than usual.
 1 I occasionally feel more self-confident than usual.
 2 I often feel more self-confident than usual.
 3 I feel more self-confident than usual most of the time.
 4 I feel extremely self-confident all of the time.

- 3) 0 I do not need less sleep than usual.
 1 I occasionally need less sleep than usual.
 2 I often need less sleep than usual.
 3 I frequently need less sleep than usual.
 4 I can go all day and night without any sleep and still not feel tired.

- 4) 0 I do not talk more than usual.
 1 I occasionally talk more than usual.
 2 I often talk more than usual.
 3 I frequently talk more than usual.
 4 I talk constantly and cannot be interrupted.

- 5) 0 I have not been more active (either socially, sexually, at work, home, school) than usual.
 1 I have occasionally been more active than usual.
 2 I have often been more active than usual.
 3 I have frequently been more active than usual.
 4 I am constantly active or on the go all the time.

APPENDIX G

BDI-II

Date: _____ **Marital Status:** _____ **Age:** _____ **Sex:** _____

Occupation: _____ **Years of Education** (High School Graduate = 12): _____

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the **one statement** in each group that best describes the way you have been feeling during the **past 2 weeks, including today**. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness

- 0 I do not feel sad.
- 1 I feel sad much of the time.
- 2 I am sad all of the time.
- 3 I am so sad or unhappy that I can't stand it.

2. Pessimism

- 0 I am not discouraged about my future.
- 1 I feel more discouraged about my future than I used to.
- 2 I do not expect things to work out for me.
- 3 I feel my future is hopeless and will only get worse.

3. Past Failure

- 0 I do not feel like a failure.
- 1 I have failed more than I should have.
- 2 As I look back, I see a lot of failures.
- 3 I feel I am a total failure as a person.

4. Loss of Pleasure

- 0 I get as much pleasure as I ever did from things I enjoy.
- 1 I don't enjoy things as much as I used to.
- 2 I get very little pleasure from things I used to enjoy.
- 3 I can't get any pleasure from the things I enjoy.

5. Guilty Feelings

- 0 I don't feel particularly guilty.
- 1 I feel guilty over many things I have done or should have done.
- 2 I feel quite guilty most of the time.
- 3 I feel guilty all of the time.

6. Punishment Feelings

- 0 I don't feel I am being punished.
- 1 I feel I may be punished.
- 2 I expect to be punished.
- 3 I feel I am being punished.

8. Self-Criticalness

- 0 I don't criticize or blame myself more than usual.
- 1 I am more critical of myself than I used to be.
- 2 I criticize myself for all of my faults.
- 3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes

- 0 I don't have any thoughts of killing myself.
- 1 I have thoughts of killing myself, but I would not carry them out.
- 2 I would like to kill myself.
- 3 I would kill myself if I had the chance.

10. Crying

- 0 I don't cry any more than I used to.
- 1 I cry more than I used to.
- 2 I cry over every little thing.
- 3 I feel like crying, but I can't.

11. Agitation

- 0 I am no more restless or wound up than usual.
- 1 I feel more restless or wound up than usual.
- 2 I am so restless or agitated that it's hard to stay still.
- 3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

- 0 I have not lost interest in other people or activities.
- 1 I am less interested in other people or things than before.
- 2 I have lost most of my interest in other people or things.
- 3 It's hard to get interested in anything.

13. Indecisiveness

- 0 I make decisions about as well as ever.
- 1 I find it more difficult to make decisions than usual.
- 2 I have much greater difficulty making decisions than I used to.
- 3 I have trouble making any decisions.

7. Self-Dislike

- 0 I feel the same about myself as ever.
- 1 I have lost confidence in myself.
- 2 I am disappointed in myself.
- 3 I dislike myself.

15. Loss of Energy

- 0 I have as much energy as ever.
- 1 I have less energy than I used to have.
- 2 I don't have enough energy to do very much.
- 3 I don't have enough energy to do anything.

16. Changes in Sleep Pattern

- 0 I have not experienced any change in my sleeping pattern.
- 1a I sleep somewhat more than usual.
- 1b I sleep somewhat less than usual.
- 2a I sleep a lot more than usual.
- 2b I sleep a lot less than usual.
- 3a I sleep most of the day.
- 3b I wake up 1-2 hours early and can't get back to sleep.

17. Irritability

- 0 I am no more irritable than usual
- 1 I am more irritable than usual.
- 2 I am much more irritable than usual.
- 3 I am irritable all the time.

18. Changes in Appetite

- 0 I have not experienced any change in my appetite.
- 1a My appetite is somewhat less than usual.
- 1b My appetite is somewhat greater than usual.
- 2b My appetite is much greater than before.
- 3a I have no appetite at all.
- 3b I crave food all the time.

14. Worthlessness

- 0 I do not feel I am worthless.
- 1 I don't consider myself as worthwhile and useful as I used to.
- 2 I feel more worthless as compared to other people.
- 3 I feel utterly worthless.

19. Concentration Difficulty

- 0 I can concentrate as well as ever.
- 1 I can't concentrate as well as usual.
- 2 It's hard to keep my mind on anything for very long.
- 3 I find I can't concentrate on anything.

20. Tiredness or Fatigue

- 0 I am no more tired or fatigued than usual.
- 1 I get more tired or fatigued more easily than usual.
- 2 I am too tired or fatigued to do a lot of things I used to do.
- 3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

- 0 I have not noticed any recent change in my interest in sex.
- 1 I am less interested in sex than I used to be.
- 2 I am much less interested in sex than I used to be.
- 3 I have lost interest in sex completely.

APPENDIX H

SRLE

Following is a list of experiences which many people have some time or other. Please indicate for each experience how much it has been a part of your life *over the past month*. Put a “**1**” in the space provided next to an experience if it was *not at all part* of your life over the past month (e.g., “trouble with mother in law – 1”); “**2**” for an experience which was *only slightly* part of your life over that time; “**3**” for an experience which was *distinctly* part of your life; and “**4**” for an experience which was *very much* part of your life over the past month.

Intensity of Experience over the *Past Month*

- 1 = *not at all* part of my life
- 2 = *only slightly* part of my life
- 3 = *distinctly* part of my life
- 4 = *very much* part of my life

- | | |
|---|-------|
| 1. Disliking your daily activities | _____ |
| 2. Lack of privacy | _____ |
| 3. Disliking your work | _____ |
| 4. Ethnic or racial conflict | _____ |
| 5. Conflicts with in-laws or boyfriend's/girlfriend's family | _____ |
| 6. Being let down or disappointed by friends | _____ |
| 7. Conflict with supervisor(s) at work | _____ |
| 8. Social rejection | _____ |
| 9. Too many things to do at once | _____ |
| 10. Being taken for granted | _____ |
| 11. Financial conflicts with family members | _____ |
| 12. Having your trust betrayed by a friend | _____ |
| 13. Separation from people you care about | _____ |
| 14. Having your contributions overlooked | _____ |
| 15. Struggling to meet your own standards of performance and accomplishment | _____ |
| 16. Being taken advantage of | _____ |
| 17. Not enough leisure time | _____ |
| 18. Financial conflicts with friends or fellow workers | _____ |
| 19. Struggling to meet other people's standards of performance and accomplishment | _____ |
| 20. Having your actions misunderstood by others | _____ |
| 21. Cash-flow difficulties | _____ |
| 22. A lot of responsibilities | _____ |
| 23. Dissatisfaction with work | _____ |
| 24. Decisions about intimate relationship(s) | _____ |
| 25. Not enough time to meet your obligations | _____ |

26. Dissatisfaction with your mathematical ability _____
27. Financial burdens _____
28. Lower evaluation of your work than you think you deserve _____
29. Experiencing high levels of noise _____
30. Adjustments to living with unrelated person(s) (e.g., roommate) _____
31. Lower evaluation of your work than you hoped for _____
32. Conflicts with family member(s) _____
33. Finding your work too demanding _____
34. Conflicts with friend(s) _____
35. Hard effort to get ahead _____
36. Trying to secure loan(s) _____
37. Getting “ripped off” or cheated in the purchase of goods _____
38. Dissatisfaction with your ability at written expression _____
39. Unwanted interruptions of your work _____
40. Social isolation _____
41. Being ignored _____
42. Dissatisfaction with your physical appearance _____
43. Unsatisfactory housing conditions _____
44. Finding work uninterested _____
45. Failing to get money you expected _____
46. Gossip about someone you care about _____
47. Dissatisfaction with your physical fitness _____
48. Gossip about yourself _____
49. Difficulty dealing with modern technology (e.g., computers) _____
50. Car problems _____
51. Hard work to look after and maintain home _____

APPENDIX I

Brief COPE

These items deal with ways you've been coping with the stress in your life. There are many ways to try to deal with problems. These items ask what you've been doing to cope with this one. Each item says something about a particular way of coping. I want to know to what extent you've been doing what the item says – How much or how frequently. Don't answer on the basis of whether it seems to be working or not—*just whether or not you're doing it*. Use these response choices. Try to *rate each item separately* in your mind from the others. Make your answers as true for you as you can.

1 = I haven't been doing this at all

2 = I've been doing this a little bit

3 = I've been doing this a medium amount

4 = I've been doing this a lot

1. _____ I've been turning to work or other activities to take my mind off things.
2. _____ I've been concentrating my efforts on doing something about the situation I'm in.
3. _____ I've been saying to myself "this isn't real."
4. _____ I've been using alcohol or other drugs to make myself feel better.
5. _____ I've been getting emotional support from others.
6. _____ I've been giving up trying to deal with it.
7. _____ I've been taking action to try to make the situation better.
8. _____ I've been refusing to believe that it has happened.
9. _____ I've been saying things to let my unpleasant feelings escape.
10. _____ I've been getting help and advice from other people.
11. _____ I've been using alcohol or other drugs to help me get through it.
12. _____ I've been trying to see it in a different light, to make it seem more positive.
13. _____ I've been criticizing myself.
14. _____ I've been trying to come up with a strategy about what to do.
15. _____ I've been getting comfort and understanding from someone.
16. _____ I've been giving up the attempt to cope.
17. _____ I've been looking for something good in what is happening.
18. _____ I've been making jokes about it.
19. _____ I've been doing something to think about it less, such as going to movies, watching TV, reading, daydreaming, sleeping, or shopping.
20. _____ I've been accepting the reality of the fact that it has happened.
21. _____ I've been expressing my negative feelings.
22. _____ I've been trying to find comfort in my religion or spiritual beliefs.
23. _____ I've been trying to get advice or help from other people about what to do.
24. _____ I've been learning to live with it.
25. _____ I've been thinking hard about what steps to take.
26. _____ I've been blaming myself for things that happened.
27. _____ I've been praying or meditating.
28. _____ I've been making fun of the situation.

APPENDIX J

The POMS

SUBJECT #: _____ **DATE:** _____ **POMS #:** _____

DIRECTIONS: Below is a list of words that describe feelings that people have. Please read each one carefully. Then select the number that best describes **HOW YOU FEEL RIGHT NOW**. Place that number on the small line to the left of each word. Do not skip any items, and print your numbers clearly.

0 = Not at all

1 = A little

2 = Moderately

3 = Quite a bit

4 = Extremely

- | | |
|--|--|
| <p>_____ 1. Tense</p> <p>_____ 2. Unhappy</p> <p>_____ 3. Sorry for things done</p> <p>_____ 4. Shaky</p> <p>_____ 5. Sad</p> <p>_____ 6. On edge</p> <p>_____ 7. Blue</p> <p>_____ 8. Panicky</p> <p>_____ 9. Hopeless</p> <p>_____ 10. Relaxed</p> <p>_____ 11. Unworthy</p> <p>_____ 12. Uneasy</p> | <p>_____ 13. Restless</p> <p>_____ 14. Discouraged</p> <p>_____ 15. Nervous</p> <p>_____ 16. Lonely</p> <p>_____ 17. Miserable</p> <p>_____ 18. Anxious</p> <p>_____ 19. Gloomy</p> <p>_____ 20. Desperate</p> <p>_____ 21. Helpless</p> <p>_____ 22. Worthless</p> <p>_____ 23. Terrified</p> <p>_____ 24. Guilty</p> |
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APPENDIX K

Daily Self Monitoring Form

Ss# _____ Monitoring Day _____ Date: _____

1. Rate your **mood** today:

0	1	2	3	4	5	6	7
Worst Possible Mood						Best Possible Mood	

2. Rate your **stress level** today:

0	1	2	3	4	5	6	7
Least Possible Stress						Most Possible Stress	

3. How many hours of sleep did you get last night? _____

4. Rate the quality of your **sleep** last night.

0	1	2	3	4	5	6	7
Worst Possible Sleep						Best Possible Sleep	

5. What was the theme of your stress (e.g., money, family, social, school)? _____

6. How many steps did you take today (pedometer reading)? _____ steps.

7. What time is it right now? _____ am / pm

8. Did you exercise today? YES NO

Strenuous Exercise = Heart beats rapidly

(e.g., running, hockey, football, judo, weight training, swimming, skiing, biking)

Moderate Exercise = Not Exhausting

(e.g., fast walking, baseball, tennis, easy biking, badminton, dancing, easy swimming)

Mild Exercise = Minimal Effort

(e.g., yoga, archery, fishing, bowling, vacuuming, window washing, golf, easy walking)

9. If you DID exercise today, rate the intensity according to the scale above.

STRENUOUS

MODERATE

MILD

10. If you exercised, what did you do (e.g., jogging, walking, yoga, weight training, etc.)?

11. How long did you exercise?

0-30 min	30-60 min	60-90 min	90+ min
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12. If you exercised, were you alone or with others? *Circle ONE choice.*

ALONE

WITH OTHERS (or in a group)

13. Why did you exercise today? *Circle ALL that apply.*

Exercise makes me feel good about myself.

My body looks better if I exercise.

I can sleep better if I exercise.

Exercise is good for my health.

I get to see my friends when I exercise.

OTHER: _____

Exercise is a part of my daily schedule.

I feel less stress after I exercise.

I have nothing better to do with my time.

Exercise is a part of my job.

I needed to vigorously clean my house.

14. If you did NOT exercise today, why? _____

15. What medication(s) did you take today? Please list: _____

APPENDIX L

Visual Inspection Criteria

The following guidelines outlined by Parsonson and Baer (1978) will be utilized in the visual analysis of data collected during this study. The first set of guidelines address variability in the data. First, stability of baseline data will be inspected, so that reasonable conclusions may be drawn regarding the effect of treatment. Any trends found in baseline data should be opposite of improvement. Next, variability within treatment phases will be examined. The optimal pattern for variability within treatment phases would be that of sudden or steady improvement during treatment. Any findings of high variability within treatment phases may indicate influence of extraneous controlling variables. Third, variability between baseline and treatment phases will be evaluated, where experimental control is demonstrated by findings of relatively stable treatment phase data followed by variable baseline data. Experimental control is less evident when both treatment and baseline phases are equally variable. Finally, baseline and treatment data will be examined for the presence of overlap. Although there are no clear guidelines regarding acceptable amounts of overlap, treatment effects showing less overlap with baseline data are most convincing.

A second set of guidelines (Parsonson & Baer, 1978) addresses patterns in data that may indicate treatment success. First, changes within overall trend relative to treatment phases will be evaluated. For example, a trend toward improvement in final data points may indicate less impact of treatment on well-being (as opposed to natural remission of symptoms). Deterioration in the final stages of treatment would also raise concerns regarding loss of control of treatment effects. Trends may be determined by

creating a trend line through data points: data in the phases are divided into two groups, the mean of each is calculated and plotted, and a straight line is drawn to connect the two points. Next, trend (slope) between baseline and treatment phases will be evaluated. A lack of baseline trend followed by a downward trend during treatment phases would be a strong indication of treatment effect. Baseline trends indicating improvement that increase during treatment would suggest that a trend would have continued in the absence of treatment. Finally, changes in level between phases will be examined. For this evaluation of the data, greater (and more abrupt) changes in level will indicate greater treatment effectiveness.

BIOGRAPHY OF THE AUTHOR

Teresa Edenfield was born in Blountstown, FL on April 4, 1978. She completed her Bachelor's degree in Psychology at Florida State University (FSU) in 2001. Teresa was a scholarship student-athlete at FSU, and played four years with the varsity women's softball team. She completed her graduate training at the University of Maine, earning a Master's degree in 2004, and fulfilling all requirements for the Doctoral degree in 2007. Teresa was awarded a research grant from the Maine Economic Improvement Fund in 2006. She completed her predoctoral internship in Health Psychology at Duke University Medical Center, specializing in promotion of health behaviors in individuals at risk for cardiovascular disease, as well as within transplant populations. She was recently awarded a postdoctoral fellowship through the Duke University Medical Center Department of Psychiatry and Behavioral Sciences. She has five scholarly publications and 24 presentations at professional meetings. She is a member of the APA general association and Divisions of Health and Clinical Psychology, the Advancement of Behavioral and Cognitive Therapies, and the Society of Behavioral Medicine. Teresa is a candidate for the Doctor of Philosophy degree in Psychology from The University of Maine in August, 2007.