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Predictors of depressive symptomatology: Cognitive theories of vulnerability and the relationship of interactional and cognitive styles

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Predictors of Depressive Symptomatology: Cognitive Theories of Vulnerability and the Relationship of Interactional and Cognitive Styles

by

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B. A., State University of New York at Stony Brook, 1996

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Depression imposes negative consequences on an individual interpersonally, financially, and psychologically. Cognitive diathesis-stress theories have been developed to try to explain the etiology of depression. The hopelessness theory (Abramson, Metalsky, & Alloy, 1989), a cognitive model that focuses on vulnerability to depression, has received a great deal of attention and support in recent literature. The hopelessness model indicates that a combination of cognitive diatheses (negative attributional style and hopelessness) and stressors (negative life events) predict the development of the hopelessness subtype of depression. Investigators continue to test the hopelessness theory, examining additional factors that may contribute to depressive etiology, in hopes of extending current understanding of depressive vulnerability. This present study examined factors that may contribute to cognitive diatheses hypothesized to increase vulnerability to depression in the hopelessness model. More specifically, a cognitive style, Need to Evaluate (NtE; Jarvis and Petty, 1996) and an interactional pattern of behavior, Excessive Reassurance-Seeking (Joiner, et al., 1992) are hypothesized to serve as moderators between cognitive diatheses, stressors, and the development of depression.

A second purpose of the study was to examine the impact of other factors (including social support, anxiety, and coping style), found to be highly correlated with depression, to ensure that relationships found to exist between study variables are not better accounted for by these factors. 129 participants completed measures of general and hopelessness depressive symptomatology, attributional style, hopelessness, reassurance-seeking, need to evaluate, negative life events, anxiety, social support, and coping style on two occasions, six weeks apart. Data were analyzed using multiple regression and correlation. Results indicated that initial levels of T2 dependent variables were the best predictors in regression models. Main effects of T1 hopelessness predicted a small amount of variance in models predicting hopelessness depression and anxiety. Interactions between hopelessness and attributional style also predicted a small amount of variance in models predicting hopelessness depression and anxiety. Need to evaluate and excessive reassurance-seeking did not significantly predict any general or hopelessness depression of anxiety. Limitations resulting from reduced power prohibited testing for moderation and of other variables. Implications for these results as well as for future research are discussed.
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Chapter One
Introduction

Depression has been estimated to affect 17.1% of members of the general population of the United States during their lifetime (Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993). According to the DSM-IV criterion for Major Depressive Disorder, a depressive episode must include depressed mood and/or loss of interest and pleasure in almost all activities. In addition, individuals must also experience at least four of the following symptoms: changes in weight, sleep, or appetite; decreased energy; feelings of worthlessness or guilt; difficulty thinking, concentrating, or making decisions; or recurrent suicidal thoughts, plans, or attempts (American Psychological Association, 1994). Recent research indicates that risk for depression is approximately twice as high for females than for males (e.g., Culbertson, 1997; Nolen-Hoeksema, 1987; Weissman & Klerman, 1985). Also research has indicated that cohorts born in the 20th century show higher prevalence rates for each decade (Klerman & Weissman, 1992; Klerman, Lavori, Rice, Riech, Endicott, Andreasen, Keller, & Hirschfeld, 1985). In addition, rates of Major Depressive disorder tend to be highest in men and women between the ages of 25 to 44 and lowest in men and women over 65 years old (American Psychological Association, 1994)

Manifestation of Depressive Symptoms

The broad array of symptoms associated with depression makes it likely that the negative impact on an individual suffering from this diagnosis may extend to many areas of life. While one individual can exhibit mostly physical symptoms (e.g., decreased sleep, appetite, and weight loss), another may suffer primarily from cognitive and
affective symptoms (e.g., irritability, lack of motivation, and dysphoric mood). It is probable that as more symptoms are experienced, consequences whether fiscal, psychological, or interpersonal may become more extensive and possibly more severe.

Depression may significantly impact an individual's health and ability to work. Research indicates that individuals suffering from depression who do not receive treatment for the disorder have 1.5 times the average health care costs than those who are not depressed (Simon, Vonkroff, & Barlow, 1995). For example, individuals with depression may have more physician visits, more days of work missed, need surgery more often, and have higher disability costs than non-depressed individuals (Greenberg, Stiglin, Finkelstein, & Bendt, 1993). These problems could well be greater for individuals who experience frequent depressive episodes. For example, Beshar and Costello (1988) found that 80% of depressed individuals have more than one depressive episode. Frequent depressive episodes are particularly harmful, as those with recurrent depression may never return to premorbid levels of adjustment (Kiloh, Andrews, & Neilson, 1988) and may experience a decrease in the amount of resources available to cope with new episodes (Moos, Fenn, & Billings, 1988). Therefore, it is quite likely that individuals who experience more depressive episodes would incur even higher financial expenses and more debilitation.

In addition to the fiscal consequences previously mentioned, cognitive and affective changes associated with depression can be devastating to depressed individuals. Although symptoms may vary in intensity and duration, there are high rates of comorbidity between major depression and abuse of alcohol (Mueller, Lavori, Keller,

One particularly important relationship is that of depression and suicide (Klerman, 1987). Research indicates that over 30,000 suicide attempts occur each year in the United States (Weissman, 1974) and it has been found to be the second leading cause of deaths among young adults (Meehan, Lamb, Saltzman, & O’Carroll, 1992; Smith & Crawford, 1986). As previously mentioned, depression is a disorder primarily characterized by low mood, decreased interest and pleasure. Although suicidality is often considered a depressive symptom, depression is also thought to be a risk factor for suicide. For example, there is empirical evidence which points to the association between suicide and major depressive disorder (Klerman & Wiessman, 1992) and for an increased risk of suicidality in depressed individuals who suffer from comorbid alcoholism (Cornelius, Salloum, Mezzich, Cornelius, Fabrega, Ehler, Ulrich, Thase, & Mann, 1995).

Several investigations have sought to examine how suicide and depression are linked. One line of research that has addressed the relationship between suicide and depression has examined the influence of cognitive factors. For example, suicide has been hypothesized to be predicted by hopelessness (Beck, 1967; Abramson, Metalsky, & Alloy, 1989), a factor involved in the onset of depression (Abramson, et al., 1989). One study in this area found that hopelessness is the best predictor of suicide attempts and
completions (Spirito, Brown, Overholser, et. al., 1989). Recent work examining cognitive models of depression and suicidality within diverse populations has also supported the role of hopelessness as a contributing factor (e.g., Gibb, Alloy, Abramson, Rose, Whitehouse, & Hogan, 2000).

In addition to the financial and psychological impact on the individual, depression often damages interpersonal relationships. Indeed, a great deal of research conducted in the area of interpersonal relationships and depression show that individuals who experience depressive symptoms, syndromes, or disorders often have difficulty obtaining or maintaining adequate social support (Coyne & Downey, 1991; Joiner, Alfano, & Metalsky, 1992; Joiner, 1994). Furthermore, there is evidence to suggest that depression can be transmitted psycho-socially by depressed individuals to others in their social circle (i.e., friends and/or relatives). Specifically, several investigators (Joiner, 1994; Katz, Beach, and Joiner, 1999) describe a theory based on their research, which states that depression is “contagious”. The theory of contagious depression proposes that one person’s depression induces depressive symptoms in another individual (see Joiner and Katz, 1999 for a review). Thus, interactional difficulties appear to be damaging not only to depressed individuals, but also to those close to them.

Predicting Depression/Dysphoria

Given that depression has been found to be debilitating to individuals physically, mentally, and financially and may have indirect consequences for family, friends, and employers, research aimed at identifying factors that play a role in the etiology, maintenance, and even prevention of depression seems highly important. In the field of
depression, empirically validated treatments such as cognitive behavioral therapy (Beck, Rush, Shaw, & Emery, 1979) are based on theoretical models that specify particular factors that contribute to both the onset and maintenance of depressive symptomatology. Therefore it is reasonable to assume that identifying unique predictors of depressive symptomatology can be useful in the development or improvement of prevention and treatment strategies that could preclude or ameliorate depressive symptoms, prevent the needless suffering of individuals and significant others, and save valuable resources of employers. It seems clear that these types of improvements would benefit depressed individuals as well as society.

Treatment for Depression

Although research examining the use of predictor variables to construct prevention strategies for depression is sparse, there are a few studies that have shown that preventative interventions are beneficial for individuals at risk for developing depression. Indeed some research indicates that preventive interventions can help to reduce the prevalence of depression (Munoz et al., 2000; Munoz, 1993). In addition, some treatments for depression have been modified to serve as preventive interventions. For example, modifications of cognitive-behavioral therapy have been specifically designed to prevent depression in college students (DeRubeis, Seligman, Schulman, Reivich, & Hallon, 1998). A great deal of research has also examined the efficacy of treatments designed to reduce symptoms and prevent relapse in individuals suffering from depression. In the area of depression, treatments that are considered empirically validated treatments include Cognitive Therapy for Depression (Dobson, 1989), Behavior therapy
for depression (Jacobson et al., 1996; McLean & Hakstian, 1979), and Interpersonal therapy for depression (DiMascio et al., 1979; Elkin et al., 1989).

In general, research aimed at validating existing psychotherapies (which are based cognitive and behavioral vulnerability models) has identified specific treatments that are highly effective. Cognitive therapy (CT; Beck et al., 1979) has been found to be at least as effective as pharmacotherapy in decreasing depressive symptoms (Blackburn, Bishop, Glen, Whalley, & Christie, 1981; Hollon et al., 1982; Murphy, Simons, Wetzel, & Lustman, 1984). Also, CT has been found to be quite effective in preventing depressive relapse in patients who were treated to remission with or without medications (Evans et al., 1992). Research into the effectiveness of behavioral therapies such as Lewinsohn’s (1975) use of homework assignments to increase client’s engagement in pleasurable activities has demonstrated improvement in depression (i.e., Gardner & Oei, 1981; Brown & Lewinsohn, 1984; Terri & Lewinsohn, 1986). A study by Hollon et al. (1992) compared the decrease in depression across three conditions including: 1) imipramine and case management, 2) CBT alone, and 3) CBT and imipramine. Although these researchers found no significant improvement between the three conditions there was a trend that suggested that CBT plus imipramine was superior to other treatments. In addition, results from this study indicate that after three years, treatments which included CBT evidenced lower relapse rates. Both behavioral and cognitive therapies have been found to be more effective than no-treatment control or routine psychiatric treatment (Craighead et. al., 1998).
There is also some evidence that provision of psychotherapy is advantageous for health care providers and for depressed individuals. Specifically, psychotherapy reduces depressive and physical symptoms, hospitalization, days of disability, and relapse rates. For example, Strum and Wells, 1995 found that providing psychotherapy to depressed patients resulted in an overwhelming improvement in patient functioning. These authors noted that the implementation of psychological services increased costs only 20 - 30 percent for primary care practitioners. This modest cost increase results in increased patient functioning and quality of life and shows that psychological treatment of depression appears to be a viable alternative to non-treatment, which as noted above, can result in large increases in health care costs for depressed individuals.

For example, an investigation conducted by Miranda and Munoz (1994), investigated the effects of an eight-week cognitive-behavioral course versus a no-treatment control condition using 150 medical patients suffering from minor depression (as specified by RDC criteria; see Miranda & Munoz, 1994 for a description) as well as physical symptoms classified as "somatization". The course provided information related to cognitive-behavioral theories of depressive etiology and treatment. Results indicated that treating medical patients who were experiencing minor depression with the eight-week intervention significantly decreased reported depressive symptoms and this change lasted through a one year follow-up. This change did not occur in the control condition or for those individuals receiving the treatment who did not exhibit minor depression during initial assessment. In addition, participants with minor depression who received the treatment showed a decrease in somatic symptoms and also missed fewer
medical visits with their primary care provider than those who did not receive the intervention. It is important to note that Munoz proposes that decreased utilization of medical services is related to depression (e.g., depressed patients will keep medical appointments less often than will non-depressed patients). Findings which demonstrate an increase in attendance at primary care appointments in depressed participants who received treatment supports this notion. Clearly, treatment based on cognitive theory has been found to be effective for depression.

**Purpose and Goals of the Present Research**

Although existing theories of vulnerability (which will be described below) have resulted in treatments that have received empirical validation, the theories underlying validated treatments (learned helplessness theory, reformulated model of helplessness, and hopelessness theory) have not received consistent empirical support. Accordingly, it is clear that additional work in these areas is needed.

The proposed research attempted to extend the efforts of previous research in the area of vulnerability to depression, proposing a new model that may further the understanding of the etiology and maintenance of depressive symptoms and syndromes. The study focused on existing theories of vulnerability to depression (Abramson et al., 1989; Abramson, Seligman, & Teasdale, 1978; Seligman, 1975). It examined whether the addition of certain predictor variables to cognitive models of vulnerability would increase the ability to predict depressive onset. The answers to these questions could potentially increase the predictability of depression and also our understanding of depressive onset, which are especially important to the continued development of
treatments and preventative interventions for depression.

**Depressive Vulnerability**

The identification of factors believed to predict depressive onset and maintenance continues to be the focus of vulnerability research. This type of research has resulted in the formation of many theories that specify pathways leading to depression. As previously mentioned, models that predict depressive onset can be useful in the development of interventions that may prevent depressive onset and treat depressive symptomatology. It is likely that theories which state a clear etiological path describing how vulnerability factors lead to depressive onset, especially those which are found to be valid and reliable, will be the most helpful in determining which areas should be targeted in treatment and preventative interventions. Vulnerability researchers continue to develop and refine models of vulnerability that strive to capture the etiology of depression. Some theories of vulnerability to depression that have received a great deal of attention will be reviewed in the following sections.

Theories of vulnerability to depression have been proposed and studied within various theoretical perspectives including psychodynamic, behavioral/interactional, interpersonal, biopsychological, and cognitive. Theories of vulnerability developed from cognitive perspectives are particularly relevant, as they have formed the basis for one of the most empirically supported therapies for depression, Cognitive Behavior Therapy (CT) (Beck, 1979). Although models proposed by psychodynamic, interpersonal, behavioral, and biopsychology offer unique perspectives on vulnerability to depression, these perspectives are beyond the scope of this paper, and thus will not be reviewed here.
Cognitive Models of Vulnerability to Depression

Cognitive models used to explain the role of cognitive vulnerability in etiology of depression include Beck's model (Beck, 1967, 1976), the learned helplessness model (Seligman, 1975), the reformulated learned helplessness model (Abramson et al., 1978) and the hopelessness model (Abramson et al., 1989).

Over the past four decades, there has been an enormous amount of research aimed at testing existing cognitive models of depression (see Ingram, Miranda, & Segal, 1998; Barnett & Gotlib, 1988; Haaga, Dyck, & Ernst, 1991, for reviews). Many research investigations conducted in the area of cognitive vulnerability to depression are designed to test only those diatheses specified either by Beck's model (i.e., schemas, dysfunctional attitudes, negative views of the self, world, and future etc) or by the helplessness/hopelessness models noted above (i.e., negative attributional style, and hopelessness). This may be because the pathways hypothesized to lead to depression in the helplessness/hopelessness models and Beck's model hypothesize that different constructs are involved in the development of depression. For example, the reformulated learned helplessness and hopelessness models of depression are essentially outgrowths of Seligman's (1975) learned helplessness theory and include attributional style as a diathesis in the development of depression, while Beck's model was developed separately and specifies different diatheses in the causal chain of depressive onset. Thus researchers may choose to examine Beck's model or an "attributional style" model of depression to avoid confusing the impact of very different constructs hypothesized to result in depression. In an effort to formulate concise hypotheses and clear conclusions about
variables that may predict depressive onset and/or maintenance, only those models that include negative attributional style as a vulnerability factor will be included in this investigation.

**Learned Helplessness Theory**

One theory that explains etiologic processes involved in depression is the learned helplessness theory, developed by Seligman (1975). This theory proposed that prolonged exposure to uncontrollable events causes helplessness. According to Seligman, helplessness is defined as the expectation of not being able to control future outcomes through efforts; Seligman notes that this expectation is not dependent on actual ability. Moreover, helplessness is established only when one learns (through experience) that he or she is unable to control certain outcomes in his or her environment and eventually come to expect that such outcomes will always be uncontrollable.

In addition to explaining how individuals learn helplessness, Seligman’s theory has implications for the development of depression. Specifically, learning that one is helpless (i.e., developing the expectation that the outcome certain events at present and in the future are outside of one’s control) is hypothesized by Seligman to lead to motivational, cognitive and affective deficits associated with depression. The motivational deficit associated with helplessness includes slower and/or decreased responding to controllable situations. The cognitive deficit refers to difficulty learning associations between one’s behavior and an outcome in a given situation where an individual feels helpless. The affective deficit refers to dysphoric affect experienced by the individual.
Helplessness theory originated from research on the effects of uncontrollable events on non-human animals. In a classic experiment, Seligman and colleagues (1967) exposed dogs placed in a shuttle box to electrical shocks that they could not avoid or escape. Once these dogs found that they could not avoid the shocks, they stopped trying to escape. This was true even when escape was again made possible. Seligman hypothesized that this behavior indicates the animals had learned that despite their efforts, they could not control outcomes in their environment (Seligman, 1975).

Similar experiments that have been carried out using a human population have reported similar findings. For example, Hiroto (1974) conducted an experiment exposing human participants to aversive noise. Participants were assigned to one of three conditions including one in which they were able to stop the noise by pressing a button, one in which the noise ended independently of their behavior, and one in which no noise was presented. During a second part of the experiment, all participants were exposed to noise that could be ended by moving a lever. Results indicated that those individuals who were not initially exposed to noise as well as those who were able to stop noise during the initial phase of the experiment, quickly learned to discontinue the noise; however, most participants in the group who were unable to control the noise in the beginning of the experiment failed to stop the noise in the second phase. It should be noted that while the independent variable utilized in Hiroto’s project was different from Seligman’s (e.g. noise vs. shock), Hiroto’s research was valuable in demonstrating that learned helplessness theory could be applied to humans.
The main limitations of the learned helplessness model of depression have been noted by Seligman and colleagues (Abramson et al., 1978) who later re-formulated the theory. These investigators posit that this model does not address several dimensions of perceived uncontrollability (e.g., whether individuals attribute uncontrollability to personal or universal factors, global or specific factors, or whether they perceive uncontrollability to be acute or chronic) and how these dimensions affect subsequent depression; therefore, learned helplessness theory may not being adequate to explain depression in humans. Limitations of learned helplessness theory including those related to perceived uncontrollability are discussed below.

One notable limitation of the original learned helplessness theory is that it does not provide a clear definition of perceived uncontrollability (Abramson et al., 1978). More specifically, the original theory fails to specify whether perceived uncontrollability, which is hypothesized to lead to helplessness and eventually to depressive symptoms, is affected by an individual’s perception of the situation in question. For example, it is unclear whether helplessness evolves from situations that are perceived as out of one’s personal control (personal helplessness) or from situations that appear to be beyond everyone’s control (universal helplessness).

Other limitations relate to the extent to which helplessness is experienced. For example, the original learned helplessness theory does not state whether helplessness learned in a particular situation will occur in a generalized manner (e.g., in a wide variety of situations) or in a specific manner (e.g., only in situations similar to those in which the helplessness was learned). Another limitation relates to the duration of
hopelessness learned from perceived uncontrollability in a particular situation. The original learned helplessness theory does not specify whether the expectation of uncontrollability (helplessness) will be chronic (i.e., will occur on an ongoing basis) or acute (i.e., will be limited to a short duration after helplessness develops; Abramson et al., 1978). The resolution of these limitations is the basis of another theory of cognitive vulnerability, which is a reformulation of the original helplessness model, based on attribution theory (i.e., Weiner, 1972).

The Reformulated Model of Learned Helplessness

Before discussing the specifics of the reformulated model, it is important to define the aspects of attribution theory (e.g., Weiner, 1972) that are applicable. The aspects of attribution theory that are relevant to the reformulated model are the specific types of attributions made following an event, that is, the attributions that a person makes about the cause of the event. When making causal attributions for an event, an individual makes decisions about whether the cause of the event is internal or external, global or specific, or stable or unstable. *Internal and external* attributions are related to an individual’s perception of the cause of an event or situation as either under or outside his or her own control. For example, an individual who fails a test can attribute his or her failure to internal factors (i.e., I did not study enough therefore I failed), or to external factors (i.e., The test was too difficult and that is why I failed). *Global versus specific* attributions addresses how generalizable the individual believes the outcome of an event or situation to be. For example, an individual may attribute the failure of a test to specific factors (I failed this test but will not fail at other tests or activities) or to global factors (I
am not a good at tests or this subject). Stable versus unstable attributions addresses the individual’s perception of the temporal consistency of an outcome of an event or situation. For example, whether the individual who failed the test describes his or her failure as unstable (My failure on this test is not related to my future performance in this course) or as stable (I will never be able to pass a test in this course no matter what I do).

In the reformulation of learned helplessness theory, an individual’s tendency to make internal, stable, and global attributions about negative events or situations (the tendency of individuals to do so is called negative attributional style) puts him or her at risk for depression after he or she experiences uncontrollable negative events. According to Abramson and colleagues, individuals who tend to make internal attributions will experience deficits in self-esteem, those who make stable attributions will likely experience more persistent depressive symptoms, and those who make global attributions should experience pervasive depressive deficits. Thus, negative attributional style serves as the main cognitive mediating variable between negative events or situations and depression. More specifically, The perception of not being able to control future outcomes is hypothesized to be sufficient to produce almost all symptoms of depression (cognitive, motivational, self-esteem, and affective deficits) if the uncontrollable event is attributed to internal, stable, and global factors (Abramson, et al., 1978). Thus when individuals believe that highly desired outcomes and aversive outcomes are beyond their control and make negative attributions for outcomes that are experienced, helplessness, as well as the cognitive, motivational, self-esteem, and affective deficits of depression result. The severity of deficits that are experienced is related to the strength of one’s
beliefs that events are uncontrollable and also on the importance of an outcome to the individual.

Although the reformulated model of learned helplessness (Abramson et al., 1978) addressed several weaknesses of the original learned helplessness model (Seligman, 1975), some research has not supported assumptions or aspects stated in the reformulation. In a review, Barnett and Gotlib (1988) discuss empirical evidence that highlights possible limitations of the reformulated model. They note that previous research has not always demonstrated the presence of negative attributional style in depressed but not in non-depressed individuals, the ability of attributional styles to predict change in depressive symptoms, and that negative attributional style is a stable cognitive trait that differentiates those at risk for depression from those not at risk. Other research that has demonstrated limitations of the reformulated learned helplessness model has proposed that although attributional style in combination with stressful life events does play a role in depressive vulnerability, other factors may be important in explaining depressive onset. For example, Robins and Block (1989) conducted a study which examined diatheses and stressors as specified in the reformulated model as well as those proposed by Beck's (1967, 1976) theory. This study placed cognitive vulnerabilities and stressors specified by each model into a more complex multivariate, interactional model. The model includes the interaction between cognitive diatheses, frequency of negative events, and perception of events as additional vulnerability factors in an attempt to understand better factors which contribute to depressive onset. To test their attributional style model, measures of frequency and perceptions life events, depressive
symptomatology, and attributional style were collected from 83 undergraduate students. Data were analyzed using hierarchical multiple regression. Results indicated the following: (1) a global attributional style was related to level of depressive symptomatology in the presence of negative life events, (2) a stable attributional style was related to depressive symptomatology, but this relationship was not influenced by negative life events, (3) attributions made for life events which resulted in depression were associated with the number of negative life events reported. In sum, these results only partially supported the predictions specified in the reformulated helplessness model and demonstrated a strong relationship between frequency of life events and depression which is not currently included in the reformulated model. The investigators concluded that cognitive theories of depression should incorporate a greater emphasis on the role of life events.

Limitations of the reformulated model of learned helplessness have also been proposed by its authors, who state that the reformulated model is more a theory of helplessness than of depression and have refined the reformulated model of learned helplessness with a new model, termed hopelessness theory (Abramson et al., 1989). These researchers point out that the reformulated model of learned helplessness is “not a clearly articulated theory of depression”, and propose four major revisions that characterize their theory, renamed hopelessness theory.

Hopelessness Theory

Hopelessness theory states that hopelessness rather than helplessness leads to depressive symptomatology in certain individuals when they are exposed to negative life
events. According to hopelessness theory, it is not specifically a negative attributional style that leads to depression. Rather, hopelessness theory proposes that an individual’s overall tendency to make negative inferences about why an event occurred, the consequences that will follow, and about the self in relation to the event, lead to negative processing of specific events when they occur. Moreover, when a negative event is experienced, an individual, considered to be vulnerable according to the theory, assigns stable, global and possibly internal attributions, as well as a high value to the event, and/or makes negative inferences about the consequences of the event, and/or makes negative inferences about characteristics about the self.

In Abramson and colleagues’ (1989) model, this process leads to hopelessness, a proximal, sufficient cause of depression. It should be noted that this specific causal chain is hypothesized to result in a subtype of depression (hopelessness depression), characterized by symptoms, including retarded initiation of voluntary responses, sad affect, suicidal behavior, lack of energy, apathy, psychomotor slowing, sleep disturbance, difficulty in concentration, and mood-exacerbated negative cognitions. A great deal of recent research has provided support for the hopelessness subtype of depression (Alloy, Abramson, Whitehouse et al., 1999; Alloy & Clements, 1998; Alloy, Lipman, & Abramson, 1992; Joiner et al., 2000). Not unlike the reformulated learned helplessness theory, in this theory, attributions are hypothesized to influence the nature of depressive symptoms. However, in hopelessness theory, if stable and global attributions are made, it is more likely that hopelessness symptoms will be experienced, while stable attributions are hypothesized to increase the severity of symptoms. Finally, if internal, stable, and
global attributions are made, symptoms of lowered self-esteem and dependency may be experienced. It should also be noted that hopelessness depression differs from endogenous depression (a subtype of depression characterized primarily by neurovegetative symptoms) as a result of the defining features of each. For example, hopelessness depression is characterized by hopelessness and while endogenous depression is characterized by anhedonia (Abramson et al., 1989). It is important to note that the reformulation of helplessness theory and hopelessness theory present negative attributions as relatively stable diatheses that exist in vulnerable individuals both in the presence and in the absence of depressive symptoms (Abramson et al, 1978; and Abramson et al, 1989).

**Research Supporting Cognitive Models of Vulnerability**

The evolution of diathesis-stress theories of depression from Seligman’s (1975) learned helplessness theory to Abramson, Metalsky, and Alloy’s (1989) hopelessness theory reflects the efforts of researchers to develop models that best describe pathways of depressive etiology. However, research related to these models has provided mixed evidence (e.g. not all studies of vulnerability have found negative attributional style and hopelessness to be significant predictors of depressive onset). The following sections provide a review of relevant research that has either supported or failed to support cognitive models of vulnerability to depression.

The Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) project (Alloy, Abramson, Whitehouse et al., 1999) is an ongoing investigation of vulnerability factors specified in hopelessness and Beck’s theories of depression. Unlike many
previous studies the CVD uses a prospective behavioral high-risk design, with non-depressed undergraduate populations from two sites. This type of design is advantageous because it allows for both prospective (e.g., a longitudinal type design where measures of cognitive vulnerability and depression are taken at baseline and then again after a specified time period to determine if depression has developed as a result of vulnerability factors) and retrospective tests (e.g., individuals are assessed for the presence of vulnerability factors and based on whether they exhibit high or low levels of these factors, are compared on their likelihood exhibiting depression in the past) of cognitive vulnerability hypotheses (Alloy & Abramson, 1999). The study measures cognitions and cognitive styles (specific to theoretical models), stressful life events, coping, personality, developmental predictors of vulnerability factors, and symptoms and presence of episodes of depression (and also the hopelessness depression subtype), as well as other mental disorders, in individuals determined to be at high or low risk for development of depression over a 5-year period (Alloy et al, 1998; Alloy & Abramson, 1999). In this project, participants were assigned to high or low risk status based on scores on screening measures. Specifically, high-risk participants scored in the highest quartile on the Cognitive Style Questionnaire (CSQ; Abramson, Metalsky, & Alloy, 1990) and on the Dysfunctional Attitude Scale (DAS; Wiessman & Beck, 1978), while low risk participants scored in the lowest quartile on each of these measures. Additionally, participants were excluded from the study if they were over 30 years of age and also if they exhibited any current episodic or chronic mood disorder, any other current Axis I disorder, current psychotic symptoms, or a past history of any bipolar spectrum disorder.
as measured by expanded Schedule for Affective Disorders and Schizophrenia, Lifetime (SADS-L; Endicott & Spitzer, 1978). Also, any serious medical illness resulted in exclusion from participation in the entire study.

As previously noted, the CVD project examined the history and development of depression and depressive symptomatology among individuals classified as either high or low risk. Findings indicate that high risk (HR) participants were more likely to experience a lifetime episode of major depressive disorder (diagnosed using DSM-III R or RDC criteria), minor depression (diagnosed using RDC), of hopelessness depression, and of depressive spectrum disorders (e.g., RDC labile personality and RDC subaffective dysthymia) than were low risk (LR) participants (Alloy, Abramson, Whitehouse, et al., 1999). Interestingly, and in contrast to some previous research (Hollon et al., 1986), there were no group differences found between rates of anxiety, addictive or other Axis I disorders. Also, findings indicate that HR participants had a higher rate of onset of major depressive disorder, minor depressive disorder, and hopelessness depression than did LR participants over the course of the study. Alloy and colleagues (1998) note that this finding, which indicates that cognitive vulnerabilities do lead to more severe forms of depression, is especially important in light of previous criticisms which focus on the limitations of cognitive vulnerability models (i.e., cognitive models are only predictive of mild forms of depression). HR participants with a past history of depression were more likely than LR participants to develop recurrent major, minor, and hopelessness depression. In addition, results demonstrate that HR but not LR participants with a
stress-reactive rumination had a greater likelihood of past and future onset of major depression and also hopelessness depression.

With the same investigation, the CVD project also examined suicidality, information processing, past history of personality disorders, and the impact of parental history of psychopathology, inferential feedback, and maltreatment on children's development of cognitive vulnerability (this was tested using participants' retrospective recall of maltreatment during their own childhood). With respect to suicidality, findings indicate that HR participants assessed at a 2.5-year follow-up exhibited a higher level of suicidality as measured by structured interview assessment and self-report than were LR participants. Results also demonstrated that hopelessness mediated the relationship between cognitive vulnerability and suicidality in even after controlling for the high rates of past history of suicidality. In addition, hypothesized risk factors for suicidality including past history of depressive disorders, borderline personality dysfunction, and parental history of depression were found to be greater for HR participants.

In relation to differences in information processing, HR participants also showed preferential processing of self-referent negative depression-relevant information as compared to LR participants (specific findings will be described later). Assessment of personality disorders demonstrated that HR participants exhibited greater dysfunction on personality dimensions of cluster A, B, and C personality disorders as measured by the Personality Disorder Examination (PDE; Loranger, 1988) administered at the outset of the study.
Assessment of parental factors indicated that parents of HR participants were more likely than those of LR participants to have dysfunctional attitudes and provide negative feedback to their children (the CSQ, ASQ, and PACE; Parents Attributions of Children’s Event’s scale; Steinberg, Tashman, Alloy, & Abramson, 1998 were completed by parents of all participants). HR participants were also more likely than LR participants to have experienced sexual, emotional, and physical abuse or neglect as children. The experience of emotional abuse was most strongly related to the development of depression in these participants.

Given that findings of research testing cognitive models of vulnerability to depression have been inconsistent, the findings of the CVD study are quite encouraging. The design used in this project provides evidence that supports theoretical propositions of cognitive models, strengthens arguments that propose cognitive diatheses are stable diatheses, and provides a replication of previous findings (e.g. Alloy, Lipman, & Abramson, 1992). However, the authors note several areas that have not been tested by the CVD project. Importantly, they suggest that environmental and personal differences protect against development of hopelessness and depression. This suggestion seems particularly salient given their finding that ruminative style and self-referential information processing are related to likelihood of depressive onset (Alloy et al., 2000). The examination of personality characteristics and other factors that may contribute to depressive onset in the CVD including rumination and self-referential processing, coping style, level of social support, personality disturbance, and developmental precursors are encouraging, and suggest that research into personality factors and behavioral styles...
should be examined in future research. This is important because variables which were not examined in this study (e.g., different cognitive and behavioral traits) may in fact be contributing to group differences in the etiology and course of depression.

Other research findings that have provided empirical support for attributional style models of vulnerability to depression are related to the specificity of vulnerability factors to depressive symptoms, syndromes, and disorders. For example, one study examined the diathesis-stress (negative attributional style and negative life events) and causal mediation component (attributions about the cause, consequences, and self) using a prospective design and found that the cognitive diathesis by stress interaction predicted depression, but not anxiety, over a five week interval (Joiner & Metalsky, 1992). This study provides evidence for the specificity of the relationship between components of the cognitive vulnerability factors specified by the hopelessness model and depression.

Alloy and Clements (1998) tested the symptom component of the hopelessness model of vulnerability to depression in a prospective investigation of 100 college students. As predicted by hopelessness theory, hopelessness was found to predict depression but not anxiety and, more specifically, significantly predicted four of eight symptoms related to hopelessness depression. The presence of hopelessness did not predict symptoms any of non-hopelessness depression subtypes or of any anxiety disorders. Although some symptoms of other psychopathology were predicted by hopelessness, it appears that the majority of research evidence supports the specificity of vulnerability factors included in the hopelessness model to depressive symptoms and syndromes.
Research Findings Related to Limitations of Cognitive Vulnerability Models

Over the past few decades, cognitive theories of vulnerability to depression have been challenged by research findings that are inconsistent with theoretical predictions. Although there has been a great deal of empirical support for cognitive models of depressive vulnerability, some studies have demonstrated that cognitive diatheses including negative attributions and dysfunctional attitudes do not differ between depressive individuals and non-depressive individuals. While some researchers conclude that the failure to demonstrate that cognitive vulnerability factors are predictive of depression represents inaccurate models (i.e., that diatheses specified by cognitive models are not predictive of depression), other literature suggests that flaws in methodology prevent accurate measurement of hypothesized cognitive diatheses leading to depression, and thus relationships between cognitive vulnerabilities and depression can not be demonstrated. Finally, some researchers suggest that cognitive diatheses are not specific to depression, but represent vulnerabilities to several disorders. The remainder of this section will present research and conclusions related to each of these possible limitations.

As previously stated, some researchers have concluded that the hypothesized vulnerability factors specified in cognitive models do not lead to depression. For example, a study by Dohr, Rush, and Bernstein (1989), examined Beck's (1967, 1976) model of depression and Seligman's (1975) model of learned helplessness depression. Their study examined attributional biases and dysfunctional attitudes in depressed and non-depressed individuals using both cross sectional and longitudinal methodologies.
Specifically, cognitive vulnerability factors hypothesized to lead to depression or to be present in depressed individuals were measured utilizing a number of self-report questionnaires including the Automatic Thoughts Questionnaire (ATQ; Hollon & Kendall, 1980), the Hopelessness Scale (HS; Beck, Wiessman, Lester, & Trexler, 1974), Dysfunctional Attitudes Scale (DAS; Weissman, 1979), the Attributional Style Questionnaire (ASQ; Seligman, Abramson, Semmel, & von Baeyer, 1979), and the Interpretation of Events Measure (IEM; Dohr, 1987). Results indicated that although symptomatic depressives scored significantly higher than remitted depressives or non-depressives on all measures of attributional biases, dysfunctional attitudes, or in their interpretation of ambiguous events related to existing schemas, no significant differences were found between remitted depressed and non-depressed participants on any of these dimensions. The authors interpreted these findings to mean that attributional biases and dysfunctional attitudes are present only during depressive episodes, and thus are more like states than traits. Thus, they were thought not to constitute vulnerability factors for depressive onset. It is interesting, however, that remitted depressive participants differed significantly from non-depressive controls on a measure of hopelessness. This finding could be interpreted to mean, contrary to the conclusions of Dohr and colleagues (1989), that hopelessness is a stable trait-like vulnerability factor to depression.

While Dohr and colleagues' conclusions related to cognitive models of vulnerability of depression focus on the idea that hypothesized cognitive diatheses including negative attributions and dysfunctional attitudes do not differ between depressive individuals and non-depressive individuals, it is clear that this evidence has
not disproved cognitive theories. For example, if as is indicated by Dohr's et al.'s findings, hopelessness is a stable predictor of depression, then it is plausible that other explanations for inconsistent empirical evidence for cognitive models of depressive vulnerability may exist and should be the subject of future research. Thus it is quite possible that the extension of previous tests of cognitive vulnerability to include constructs specified in more recent cognitive diathesis-stress models (i.e., hopelessness) will result in evidence which supports cognitive vulnerability theory. The examination of non-cognitive predictors (described above) tested in the CVD project (Alloy, Abramson, Whitehouse, et al., 1999) showed that hopelessness and several other factors increased the risk of depression and provided a great deal of evidence supporting cognitive models of depressive vulnerability.

However, as noted above, many criticisms of cognitive diathesis-stress models focus on limitations in methodology as opposed to fundamental flaws in cognitive theory. Critiques of methodology of studies related to cognitive models of vulnerability to depression constitute another challenge to the validity of cognitive vulnerability theories. Specifically, some reviews of the literature on cognitive models of depressive vulnerability indicate that limitations in methodology limits may be limiting the conclusions that can be drawn regarding the presence of cognitive diatheses. For example, Barnett and Gotlib (1988) note that although many studies using cross-sectional designs have found cognitive vulnerability factors do co-occur with depressive symptoms (e.g. attributional style and psychosocial stressors; Metalsky, Haberstadt, & Abramson, 1987), these types of research designs do not allow for statements to be made regarding
the temporal antecedence of vulnerability factors. They propose that prospective (two-wave panel) designs should be used to ameliorate methodological limitations inherent in cross-sectional designs. However, only two studies described in the review which use prospective designs to test vulnerability factors supported cognitive models of depressive vulnerability (e.g., Cutrona, 1983; O'Hara et al., 1982). These results may indicate that methodological flaws are not solely responsible for the absence of consistent support for cognitive models of vulnerability to depression. Indeed, results from the CVD study, which utilizes a longitudinal type design, has demonstrated a great deal of support for the hopelessness model of depression. Methodological considerations in the area of cognitive vulnerability to depression have been addressed frequently in recent research (see Ingram et al., 1998 for a review).

Finally, findings that diverge from existing cognitive theories of depression examine whether hypothesized vulnerability factors are specific to depression or whether they can predispose individuals to other forms of mental illness. For example, Hollon, Kendall, & Lumry (1987) examined dysfunctional attitudes and automatic thoughts in individuals with various mental illnesses including currently depressed bipolar I, currently depressed substance abuse disorder, current unipolar depression, non-depressed substance abuse, schizophrenia, Briquet’s syndrome (now called somatization disorder), obsessive-compulsive disorder and also, individuals who were remitted bipolar and unipolar depressives, and medical patient and normal controls. Participants completed the ATQ and DAS as well as measures of depression and intelligence. Findings indicated that dysfunctional attitudes and automatic thoughts did covary with
depressive symptomatology. However, covariation on the DAS was not limited to depressed (bipolar or unipolar) participants. Also, significant differences were not found for bipolar versus unipolar depressives or for remitted depressives versus non-depressed controls on the ATQ or DAS. Moreover, it appears that the DAS may not measure cognitive diatheses (dysfunctional attitudes) that are specific to people who are depressives, and that cognitions measured by the DAS and ATQ (dysfunctional attitudes and automatic thoughts respectively) are largely state dependent. Thus, in the above example, Hollon and colleagues concluded that dysfunctional attitudes and automatic thoughts do not seem to be measuring factors exclusively involved in the etiology of depression. While more recent research has demonstrated that cognitive diatheses (i.e., negative attributional style and hopelessness) are specific to depression (e.g., Alloy, Abramson, Whitehouse, et al., 1999), it is clear that more research is needed in this area.

**Incorporating Non-Cognitive Variables into Cognitive Vulnerability Models**

As noted above, it is possible that inconsistent findings of research investigating the role of cognitive vulnerability factors in the development of depression mean that cognitive theories of vulnerability are incomplete. Insofar as cognitive therapy for depression targets cognitive predictors (i.e., negative attributions), research that supports the efficacy of cognitive therapies also provides indirect support for cognitive models of vulnerability to depression.

Numerous investigations (see section on empirically validated treatments above) provide supporting evidence for the efficacy of cognitive therapies with depressed clients. However, Miranda and Persons (1992, 1998) note inconsistent support for cognitive
models of vulnerability to depression, and state that many researchers have interpreted this discrepancy to mean that cognitive vulnerability models are flawed (e.g., Hammen, Miklowitz, & Dyck, 1986). These researchers pose the question of why successful interventions work if they are based on theories that are inaccurate. One potential explanation for this difference may be the mood-state hypothesis.

These researchers noted evidence which does not fully support all aspects of cognitive theories, including findings that indicate (1) most longitudinal studies show that dysfunctional attitudes covary with but do not necessarily cause depressive symptoms, (2) that other studies comparing non-depressed and remitted depressive individuals do not find differences in dysfunctional attitudes or attributions, and (3) that the results of prospective longitudinal studies of cognitive vulnerability are mixed, indicating that cognitive diatheses are not stable predictors of depressive onset (see Barnett & Gotlib, 1988, for a review). Conversely, Persons and Miranda hypothesized that cognitive diatheses represented in these theories are stable, but are only accessible in the presence of a negative mood state. Thus, individuals with high levels of dysfunctional attitudes or negative attributions able to disclose these cognitions as their mood become more negative. This hypothesis, referred to as the "mood-state-hypothesis", supports cognitive theory, and provides an explanation for contradictory research findings. According to the mood-state-hypothesis, the reason why studies have failed to validate cognitive theories of vulnerability to depression empirically is that participants who are not in a depressive episode would not be able to report negative cognitions. Persons and Miranda (1992) speculate that to measure diatheses specified by cognitive vulnerability theories in non-
depressed or remitted depressive individuals, it is important to assess these individuals when they are experiencing a negative mood, either arising naturally or through mood induction.

In their 1992 article, these authors cite four studies that provide support for the mood-stated hypothesis (Miranda & Persons, 1988; Miranda, Persons, & Byers, 1990; Teasdale & Dent, 1987; Nolen-Hoeksema, 1987). For example, Teasdale and Dent (1987) examined the affects of an experienced shift in mood on the self-perception of cognitively vulnerable women. Women in this study were asked to complete an adjective checklist after being presented with a sad music mood induction. Results indicated that while neither vulnerable nor non-vulnerable women differed in the number of negative self-descriptive adjectives they endorsed immediately following the mood induction, the vulnerable women exhibited a greater increase in negative self-descriptive adjectives than did non-vulnerable women following a brief delay. According to these authors, negative schemas in the vulnerable women were activated by the negative mood created by exposure to the music. However, this and other studies have been critiqued for methodological limitations. Miranda and Persons (1992) noted that the studies that they described (see citations above) as supporting mood-state hypothesis demonstrated evidence from correlational analyses and thus, do not directly demonstrate that experience of a negative mood state causes negative thinking.

More recent research conducted by Roberts and Kassel (1996) have provided a replication and extension of the mood-state hypothesis by measuring wider range of cognitions that could represent diatheses, including dysfunctional attitudes, automatic
positive and negative thoughts, and self-esteem, as well as by examining the role of positive affect (PA) and negative affect (NA) as possible mood primes (see Roberts & Kassel, 1996 for a description of study variables and procedure). Participants included remitted dysphoric individuals and individuals reporting no history of dysphoria. Results indicated that negative cognitions appeared mood-state dependent in remitted dysphoric (vulnerable) but not in never dysphoric (invulnerable) individuals, and, also, that the relationship between negative thinking and NA was greater among remitted dysphoric participants. Although these results are consistent with the mood state hypothesis, they share an important limitation with Person and Miranda's (1992) study; neither study used a prospective design, and thus, they could not assess the development of depression in individuals with cognitive diatheses (Person & Miranda, 1992).

Interestingly, research which demonstrates that targeting behavioral issues in cognitive therapy leads to better outcome (Hayes, Constanguay, & Goldfried, 1996), as well as research which indicates that cognitive processes can serve as triggers to access negative mood state (e.g. mood induction using self-focus), may indicate that patterns of behavior or cognitions that influence the relationship between negative mood state, experience of dysfunctional thoughts, and depressive onset. For example, if an individual has a behavioral or cognitive style that causes him or her to stay in a negative mood state on a regular basis, they would have greater access to negative cognitions, and thus, according to mood-state-hypothesis (and cognitive theories of depression) would be more likely to develop depression when exposed to meaningful stressors. Here,
remission can be explained by the absence of stressors as opposed to the inaccessibility of dysfunctional attitudes resulting from change in mood.

**Contributory Causal Factors within Cognitive Vulnerability Models**

An alternative explanation for research findings that do not support cognitive diathesis-stress models may also be that cognitive vulnerability hypotheses are incomplete, and do apply to individuals with certain characteristics found to be included in cognitive therapies that are successful. In other words, cognitive therapies are broad in scope and often emphasize behavioral style, social interactions, as well as negative thoughts and beliefs. It is possible that stable stylistic aspects of a person's behavior and thinking, other than those specified in the aforementioned cognitive models, may mediate or moderate the relationship between cognitive diathesis, life stressors, and depressive onset or recurrence. Moreover, it could be that those individuals with certain mediating/moderating characteristics do report cognitive diathesis at higher levels, and are more likely to experience depressive onset or recurrence than non-depressed individuals.

Insofar as specifying what type of variables may add to our understanding and the predictive power of existing cognitive models of vulnerability to depression, it may be helpful to examine research related to cognitive therapy because cognitive treatments are grounded in cognitive theory. Specifically, teasing out what it is about cognitive therapy that leads to a decrease in depressive symptoms can provide information about other factors that may be included in cognitive models of depressive vulnerability. For example, one aspect of CT (Beck et al., 1979) that is not hypothesized to result in direct
change of depressive symptoms is specific behaviors assigned in treatment in order to allow individuals to monitor behavior and related thoughts and feelings (Craighead et al., 1998).

Although cognitive theory indicates that while the performance of specific behaviors (i.e., engaging in social interactions) is not responsible for directly changing symptoms in individuals already experiencing depression, they may be very important in understanding depressive etiology. As cognitive-behavioral therapy for depression (e.g., Beck, 1979) focuses on changing thoughts and behaviors, targeting one or the other may not result in a decrease in depressive symptoms. For example, increasing the frequency of a depressed individual’s social interactions may not decrease his/her depressive symptoms unless depressogenic cognitions (i.e., negative attributions) are changed at the same time. However, this strategy may be effective in preventing depressive onset in a non-depressed, but cognitively vulnerable individual. The behavior of engaging in social interactions may be related to cognitive diatheses, stressors, and depression for a number of reasons. For example, interacting with others could potentially provide access to positive attributions or prevent access to negative ones (i.e., an individual feels competent, as opposed to incompetent, because he or she is able to converse with others), improve one’s ability to cope with negative life events (i.e., talking to others allows for emotional expression related to stressors or via general social support), and provide opportunities to invalidate distorted cognitions (i.e., others provide positive feedback). In addition, some researchers have hypothesized that social support (e.g., material, informational, and emotional) received from others may buffer against depression by
preventing the development of hopelessness (Abramson et al., 1989; Brown, Andrews, Harris, Adler, & Bridge, 1986; Panzarella & Alloy, 1995, 1999). Panzarella and Alloy (1995, 1999) explain that hopelessness may be prevented because support supplied by others contains adaptive inferential feedback. Thus, information given to an individual at risk for developing depression serves to promote the formation of benign (as opposed to depressogenic) inferences about an event's cause, consequence(s), and meaning. Conversely, a lack of social interaction could result in negative attributions related to the self (i.e., that one is not worthy or capable of obtaining other's attention or affection) or in hopelessness (i.e., that one will never be able to have satisfying interpersonal relationships). Although the mechanism that relates behavior and cognitions within this example is unclear, it is reasonable to hypothesize that behavior could interact with cognitive vulnerabilities in a manner that increases the likelihood of experiencing depression. Specifically, it could serve as a contributory cause within an existing cognitive model of depressive vulnerability.

Research and theory in the area of social support may provide some insight into how social interactions are related to cognitions. Social support has been implicated in the prevention of psychological illness (Collins, Dunkel-Schetter, Lobel, & Schrimshaw, 1993; Cohen & Wills, 1985; Barnett & Gotlib, 1988; Gotlib, Whiffen, Wallace, & Mount, 1991; Holohan & Moos, 1991; Norman, Miller & Dow, 1988; Phillips & O'Hara, 1991; Quittner, Glueckuf, & Jackson, 1990; Wilbert & Rupert, 1986) and as previously noted, of hopelessness (Abramson et al., 1989; Brown, et al., 1986; Panzarella & Alloy, 1995, 1999).
For example, two hypotheses have been proposed to explain why social support increases the ability to cope with stress. The *direct or main effect hypothesis* indicates that social support is thought to have a direct effect on an individual’s well-being (e.g. Bell et al., 1982; Moos & Mitchell, 1982; Cohn & Wills, 1985). Hypothesized reasons for the connection between social support and well-being include the following: 1) that social support provides experiences that involve “positive affect, a sense of predictability and stability in one’s life situation, and a recognition of self-worth (Cohn & Wills, 1985), 2) that involvement in a social network provides a source of positive experiences that increase one’s ability to cope (Flannery & Wieman, 1989), and 3) that social interaction or social integration may directly affect one’s sense of well-being (Moos & Mitchell, 1982; Reis, 1984; Cohn & Wills, 1985).

Another hypothesis related to the relationship between social support and the ability to cope with stress is the *buffering hypothesis*. Cohen and Wills (1985) buffering model proposes that social support modifies the relationship between stress and psychological illness in two ways. These authors state that social support may decrease perceived stressfulness of a situation by reducing the perceived stressfulness of the event and thus, changing the individual’s coping response. Also, the buffering hypothesis indicates that information from a support network may serve to alter an individual’s interpretation of an event’s magnitude or to provide solutions to perceived difficulty and therefore, represents a moderating relationship. In sum, social support is hypothesized to reduce an individual’s experience of stressors and thus their reaction to them in a given situation.
In addition to social support, other factors have been found to increase the likelihood that individuals will benefit from cognitive therapy. For example, a study conducted by Hayes, Castonguay and Goldfried (1996) found certain factors that seem to be related to the efficacy of cognitive therapies. Their investigation was based on archival data of individuals who received cognitive therapy as part of the Cognitive-Pharmacotherapy Treatment project (Hollon et. al., 1992). It is important to note that this study focused solely on cognitive interventions rather and did not examine comparisons between cognitive and drug therapies. Client-therapist videotaped interactions were coded using the Coding System of Therapist Focus (CSTF; Goldfried, Newman, & Hayes, 1989) to assess the degree to which therapists focused on cognitive, interpersonal, and developmental domains specified in Gotlib and Hammen's (1992) theory of depression. Contrary to the Beck and Colleagues' (1979) cognitive therapy (CT) rationale that changing distorted cognitions and underlying schemas (rather than behavioral changes included in treatment) are responsible for decrease in depressive symptoms, Hayes and colleagues found that interventions used by therapists in CT sessions which addressed interpersonal (e.g., feedback on social functioning, direct change of problematic interactions) and developmental domains (e.g., exploration of experiences with parents) were related to symptom reduction in 30 depressed outpatients. Also, the reduction in symptoms experienced in this group was greater than the reduction experienced by participants who received cognitive therapy that did not address interpersonal and developmental domains.
Another study that examined the role of cognitive change in cognitive therapy demonstrated that change in depression-relevant cognitions were related to change in depressive symptomatology in 32 outpatients with major depressive disorder in a 12-week study (DeRubeis, Evans, Hollon, Garvey, Grove, & Tuason, 1990). In this study, participants in the cognitive therapy group (who received 16 – 20 sessions of CT; Beck et al., 1979) completed measures of depression severity, attributional style, dysfunctional attitudes, automatic thoughts, and hopelessness at the beginning middle and end of the study. As was consistent with cognitive models of depressive vulnerability (e.g. Beck, 1967, 1976; Abramson et al., 1989), change from pretreatment to midtreatment (as measured by ASQ, DAS and HS) predicted change from mid to posttreatment for participants in the CT group. This finding was not replicated in the non-CT group who received pharmacotherapy and medication management only. Authors concluded that cognitive diatheses (e.g. negative attributional style and dysfunctional attitudes) significantly predict change in depression for individuals who received cognitive therapy and that this relationship was stronger for individuals in the cognitive treatment group than for those in the pharmacotherapy treatment group; however, hopelessness did not predict this pattern of change.

The findings of the studies mentioned above seem to indicate that while changing cognitive diatheses (e.g., attributional style) does result in decreased depressive symptomatology after treatment with CT, other factors that are not the focus of CT may influence the relationship between cognitive changes and depression. With respect to cognitive theories of vulnerability to depression, these results may mean that
supplementing existing models of depressive vulnerability with factors that may improve response to treatment would be likely to result in an increased ability to predict and understand onset of depressive symptoms, syndromes, and disorders.

**Description of Moderating and Mediating Variables**

Although cognitive theories of vulnerability have generally been supported in the depression literature, some studies have not demonstrated a relationship between cognitive diatheses, stressors, and depressive onset (see Barnett & Gotlib, 1988 for a review). While this inconsistency does not disprove current cognitive theories of vulnerability to depression, it suggests a need to re-examine and possibly expand these models to include new predictors of depressive vulnerability.

It is possible that certain variables not previously included in cognitive models will influence the causal relationships between cognitive diatheses, stressors, and depressive symptomatology proposed by Beck (1967, 1976) and Abramson and colleagues (1978). The addition of variables to existing cognitive models of depressive vulnerability may account for the absence of consistent findings in prospective and retrospective studies examining the role of cognitive vulnerability in the onset and maintenance of depression. The present project aims to examine variables that may be added to cognitive theories of vulnerability in order to be better able to predict depressive onset and maintenance. Since the cognitive and behavioral factors which will be examined in the present study are hypothesized to strengthen the relationship between cognitive diatheses, stressors, and depression, they will be likely to function as moderator...
variables. Before discussing proposed hypotheses, it is essential to understand the nature of moderating variables.

Definitions of Moderator and Mediator

As defined by Baron and Kenny (1986), “a moderator is a qualitative...or quantitative...variable that affects the direction and/or strength of the relation between an independent or predictor variable and a dependent or criterion variable”. An example of a moderator variable is the presence of negative mood in Person and Miranda’s (1992) mood-state hypothesis. Within this model, negative mood strengthens the relationship between cognitive diatheses (e.g. negative attributions), stressors, and depressive onset. Here negative mood (moderator), cognitive diatheses in combination with life stressors (predictor), and the interaction of moderator and predictor variables are associated with the criterion variable.

According to Baron and Kenny, the moderator hypothesis will be supported if the relationship between the interaction and criterion variable is significant. In addition, these authors indicate that the predictor and moderator should be uncorrelated (as correlation between the predictor and moderator may prevent the delineation of a clear interaction term) and also that moderator variables are not causally related to predictor variables (they are not antecedents or consequences of predictors). If either of these conditions occurs, or if the hypothesized relationship between the predictor and criterion variable dissolves when the proposed moderator variable is not included in the model, the variable in question may be functioning as a mediator.
Mediator variables serve to explain the relationship between the predictor and criterion variables. According to Baron and Kenny, the mediator accounts for the predictor-criterion relationship, and is said to be functioning under the following three conditions: (a) changes in the levels of the independent variable significantly account for changes in the mediator, (b) changes in the mediator significantly affect changes in the criterion, (c) the relationship between the predictor and criterion is significantly reduced, although not eliminated, in the absence of the mediator. These authors also note that since the association specified in condition c (above) is not eliminated, the mediator cannot be a necessary and sufficient causal variable. For example, negative mood-state (specified in mood-state hypothesis) could be functioning as a mediator if it significantly reduced the association between cognitive diatheses-stress variables and depressive onset and if changes in mood-state were directly related to changes in both levels of cognitive diathesis and levels of depressive symptomatology.
Although expanding extant cognitive theories by including new moderator variables may provide a better understanding of processes involved in depressive onset, selecting moderators to add to cognitive models appears to be an extremely complicated task. However, this can be guided by the literature. As noted above, Hayes and colleagues (1996) found that certain behavioral and cognitive factors addressed in cognitive-behavioral therapy contributed to an individual's success in treatment. It is plausible to assume that addressing behavioral and cognitive factors not included in original cognitive vulnerability models lead to increased treatment efficacy because these factors are involved in depressive etiology, perhaps acting as moderators of cognitive diatheses, stressors, and depression.

**Trait Variables as Potential Moderators of the Hopelessness Model**

Thus, while research that increases the validity of cognitive models by adding state-like variables (i.e., negative mood-state) is promising, these variables may not be very helpful in developing treatments because they are transient. It is probable that looking at trait-like variables (which are assumed to be more permanent) would be more useful for this purpose. For example, recent research related to the hopelessness model of vulnerability to depression has demonstrated that a cognitive trait, self-referent information processing, increases the risk of developing hopelessness depression in individuals who are cognitively vulnerable (Alloy et al., 2000). This study was conducted as part of the Cognitive Vulnerability to Depression (CVD) study described previously. All participants (both HR and LR) were presented with a Self-Referent Information Processing task (SRIP) comprised of sub-tasks that resulted in dependent
variables. First, participants were shown adjectives on a computer monitor, which included positive and negative items related or unrelated to depressive cognitions and were asked to rate whether each item was representative of themselves or not by pressing a "me" or "not me" button on the keyboard. Next, participants were given a booklet and were asked to provide examples of behavior that supported each adjective they rated as self-descriptive (e.g., if they endorsed an adjective stating they are worthless, they had to provide an example of a time when they were). In the third task participants were read statements describing hypothetical behaviors and were asked to rate how likely it is that they would engage in each behavior if they encountered that situation in the future. Finally, participants were asked to recall adjectives they had rated in the initial adjective presentation following a two-hour delay.

Consistent with study hypotheses, results indicated that for the first task HR participants endorsed less positive depression-relevant adjectives and more negative depression relevant adjectives than did LR participants. Also, HR participants showed a trend toward responding faster than LR participants at selecting adjectives that were negative and related to depression and at responding slower than LR participants in selecting adjectives that were positive and related to depression (as measured with response time latencies). Importantly, this finding remained after controlling for current symptoms of depression. In the second task, HR participants were more likely to access examples of negative, depression-relevant adjectives that were selected and less likely to access examples of positive, depression-relevant adjectives that were selected. Results from the third task indicated that HR participants were more likely than LR participants
to predict that they would behave in negative, depression-relevant ways and less likely than LR participants to behave in positive, depression-relevant ways. In the fourth task HR participants were more likely than LR participants to recall negative, depression-relevant adjectives after the two-hour delay. Perhaps most importantly, results of the study indicated that for cognitively vulnerable individuals, the presence of negative information processing increased the likelihood of developing hopelessness depression (and also that it served as a moderator in this relationship).

Although findings from Alloy and colleague’s research have not yet been applied to a treatment model, it is plausible that identifying and/or changing thoughts or behaviors related to self-referent information processing may be helpful in reducing risk of depressive onset and possibly in decreasing depressive symptoms. Results related to self-relevant information processing from the CVD project highlights the importance of continued research aimed at identifying other factors that represent vulnerabilities to depression and could be extended to develop existing and new cognitive treatments for depression. Examination of characteristics such as traits or behavioral or cognitive styles would appear to be extremely valuable in this capacity as these factors are somewhat stable and are likely present throughout the course of depression. Isolating these types of factors as predictors of response to particular treatments would be highly beneficial to both treatment and prevention planning. Individuals best suited to a particular form of psychotherapeutic intervention could then receive the best care possible. Perhaps more importantly, knowledge about enduring factors associated with depression could provide
the opportunity to implement preventative interventions in those found to be at risk for depression.

**Interactional and Cognitive Styles as Potential Moderators of Cognitive Models of Depression**

An increased understanding of factors that contribute to depression may aid in the development of treatments and preventative interventions. Research in the area of cognitive vulnerability to depression has provided some support for the cognitive theories that are the basis for validated treatments for depression. The advantage of expanding cognitive models to include additional risk factors is reflected in the results of outcome research of certain cognitive treatments for depression. Some outcome research has indicated that cognitive therapies which treat symptoms and deficits that are not traditionally addressed by cognitive treatment models have beneficial results. For example, interactional components of CBT (i.e., feedback on social functioning and direct change of problematic interactions) described by Hayes and colleagues (1996) are effective in use with depressed clients. If a decrease of depressive symptoms resulting from cognitive treatment that targets cognitive diatheses (i.e., negative attributional style; hopelessness) provides support for cognitive models of vulnerability, then it would appear that a decrease in depressive symptoms resulting from aspects of treatment which focus on ameliorating interactional deficits should provide evidence that interactional factors are involved in depressive etiology. With this in mind it seems that cognitive and interactional styles may influence the onset, maintenance, or recurrence of depression.
In addition to the previously described cognitive processes (specified in cognitive vulnerability models) there appear to be cognitive and interactional styles that are consistent in depressed individuals. It has been demonstrated that highly depressed individuals utilize more wishful thinking, escape-avoidance and confrontative coping, seeking emotional support, and information seeking than less depressed individuals (Folkman & Lazarus, 1986; Coyne et al, 1981). Cognitive theories state that depressed individuals show patterns of thinking such as negative attributional style, helplessness, and hopelessness both before and during depressive episodes (Seligman, 1975; Abramson et al., 1978; Abramson et al., 1989). In addition, interactional theories (e.g., Joiner et al., 1992) posit that in their interactions with others, depressed individuals may act in ways that perpetuate their depressive symptoms. For example, these authors hypothesize that excessive reassurance-seeking influence the course of depression. Thus each of these lines of research has identified factors that contribute to the onset or maintenance of depression. As previously stated, cognitive, behavioral, and interactional factors are addressed in validated therapies for depression (e. g. CBT; Beck 1967; 1976); however, it does not appear that existing cognitive models of vulnerability have examined the contributions of behavioral or interactional styles in depressive etiology. It is plausible that since factors in cognitive, interactional, and behavioral domains are helpful in treating depression, that explanations of depressive onset and maintenance should include aspects from these domains.

The lack of models that examine the complexity of factors that contribute to depressive vulnerability has been addressed in recent literature. For example, Dobson
(2000) discusses limitations of single-factor models of vulnerability (e.g., a model focusing on only one perspective can not capture all of the variance involved in etiology of a psychological disorder) and examines chronic processes that influence depressive onset, maintenance, and relapse/recurrence. Dobson describes processes as factors that represent either internal characteristics (e.g., assertiveness) or external occurrences (e.g. daily hassles) that intervene at a particular stage (e.g. onset, maintenance/duration, remission/recovery, relapse, or recurrence) of a psychological disorder, in this case depression. He provides a brief review of processes that have been incorporated within vulnerability models (e.g. attributional style, excessive reassurance-seeking, helplessness/hopelessness, negative life events, low social support, rumination etc.) and describes these factors influence at particular stages of depression using Joiner et al.’s (2000) framework of erosive processes (processes that are consequences of an episode of depression) and self-propogatory processes (processes that are engaged in by a previously depressed person that increase the risk of future depressive episodes). Finally, Dobson calls for the increased utilization of transactional, multifactorial models to improve understanding of depression throughout the course of the disorder (for a more thorough description of topics addressed in this commentary see Dobson, 2000).

Two stable trait-like variables that may function as moderators in extant attributional style models of vulnerability to depression (e.g. Seligman, 1975; Abramson et al., 1978; Abramson et al., 1989) are presented in the remainder of this section. First, an interactional style, excessive reassurance-seeking (RS; Joiner et al., 1992) will be presented. Excessive reassurance-seeking will first be discussed in terms of its
relationship with attributional style. An explanation of RS along with a review of related research will be provided, as will explanations about how RS may be incorporated into cognitive models of vulnerability to depression.

This study will also examine an additional cognitive style, the need to evaluate (NtE; Jarvis & Petty, 1996). The relation of NtE to attributional style models of depression along with an explanation of this construct and related research will be discussed. Also, implications for the incorporation of NtE into cognitive vulnerability models will be presented. Specifically, the present research will attempt to determine how both RS and NtE function within the hopelessness theory of depression (Abramson et al., 1989).

The Role of Behavior and Interactional Style within Cognitive Vulnerability Models

Some cognitive theories propose that negative attributional style in combination with the development of hopelessness and environmental stressors predispose an individual to depression (Abramson et al., 1989). It is likely that engaging in behaviors or patterns of interaction that increase perceived environmental stress, hopelessness, and focus on negative attributes of the self would result in depressive onset more often in vulnerable individuals. For example, this type of increased risk could occur in a cognitively vulnerable individual who is working in a demanding, highly stressful environment and makes several mistakes over the course of the day. Although mistakes may have occurred as a result of external factors (i.e., not having enough time to check work), they may be attributed to stable, internal, and global causes (i.e., I am a slow worker, I am careless, I will never be as efficient as others will). As a result, the
individual may experience dysphoric affect and hopelessness, especially if mistakes occur on a regular basis and attributions become more stable. This illustration is meant to demonstrate the potential role of behavior (especially stable patterns of behavior) in moderating cognitive diatheses to depression. One behavioral/interactional style which has been associated with depressive onset is excessive reassurance-seeking (Joiner et al., 1992).

**Excessive Reassurance-Seeking**

Reassurance-seeking behavior was first noted in an interpersonal model of depression developed by Coyne (1976). Reassurance-seeking is the tendency of individuals vulnerable to depression to seek consistent reassurance or validation (e.g., as to their self worth or as to whether others truly care about them) from other individuals. According to Coyne, reassurance-seeking behavior is initiated in mildly dysphoric, but non-depressed individuals, after the perception of a loss or change in social structure. At this point, an individual will elicit feedback from others, but doubt the sincerity of this feedback, and thus, continue to seek reassurance. Thus a cycle develops where a dysphoric individual asks for and receives reassurance, doubts the feedback they are given, and therefore needs and requests additional reassurance. Those providing reassurance become increasingly annoyed with this behavior, and may either discontinue contact or continue to give out feedback in a way that conveys their frustration. In either case, the individual seeking reassurance is left with the feeling that they are unloved and rejected. These feelings of frustration and rejection are products of the cycle (described above) and are hypothesized (by Coyne) to lead to depression.
Joiner and colleagues (1992) extended Coyne's (1976) theory by specifying that excessive reassurance-seeking is the most important factor in the development and maintenance of depression in that it transmits distress and depression from one person to another. Thus it appears that the difference between Coyne and Joiner's interactional models is the emphasis placed on excessive reassurance-seeking. A number of recent investigations have demonstrated a high rate of excessive reassurance-seeking exists among clinically and non-clinically depressed individuals (Joiner et al., 1992; Joiner & Metalsky, 1995; Joiner, 1994; Joiner, Katz, & Lew (1999); Joiner & Metalsky, 1998). In addition, there is evidence that the relationship between excessive reassurance-seeking is specific to depression (Joiner & Metalsky, 1995; Joiner & Schmidt, 1998). For example, Joiner and Schmidt (1998) investigated whether excessive reassurance-seeking prospectively predicted changes in depressive and/or anxious symptoms in 1,005 Air Force cadets. Measures of excessive reassurance-seeking, depressive symptoms, and anxious symptoms were administered over a five week period (once at the beginning and once at the end of basic training). Consistent with hypotheses, results indicated that excessive reassurance-seeking did predict depressive but not anxious symptoms, and that this relationship was present even after statistically controlling for initial depressive and anxious symptoms.

In general, results from investigations of excessive reassurance-seeking seem to indicate that excessive reassurance-seeking does play a role in the onset and maintenance of depressive symptoms and also of clinical depression. Other research has demonstrated that there is an interaction between an excessive reassurance-seeking diathesis and
stressors leading to an increase of depressive symptoms (Joiner & Metalsky, 1995).

Joiner (1999) notes that excessive reassurance-seeking is a relatively stable tendency that acts as a contributory causal factor in his model of depression. Given these findings, it is plausible that RS could add to the ability of cognitive diathesis-stress models to predict depression. Specifically, in that RS has been found to be a contributory causal factor of depression in the presence of stressors, it is likely that it could account for some variance in cognitive diathesis-stress models of depression. The addition of RS to cognitive vulnerability models (e.g., the hopelessness model) may provide a more accurate explanation of depressive etiology and may also provide insight into why some research has failed to support cognitive models of vulnerability to depression.

Several theorists and researchers have attempted to create models of depressive vulnerability that include excessive reassurance-seeking, other diatheses (i.e., cognitive factors) and stressors. For example, based on past research related to vulnerability to depression, Schmidt, Schmidt, and Young, (1999) proposed a model, which incorporates cognitive diatheses into interactional theories. According to these authors, cognitive diatheses such as maladaptive schemas, can serve to predict when a person will engage in excessive reassurance-seeking. Also, biases in information processing are thought to influence how reassurance seekers perceive their own excessive reassurance-seeking behavior, and the feedback they receive from others. Insofar as how schemas and reassurance-seeking result in depression, their model posits that either depressogenic information processing (as described by Beck, 1967,1976) or reassurance seeking (as described by Coyne, 1976) are sufficient to predict depression. While the presence of
both depressogenic information processing and reassurance seeking predicts a substantial risk for depression, the absence of both is hypothesized to describe resilient, non-depressed individuals (so long as other risk factors such as biopsychological factors are not present).

Several theories of the role of excessive reassurance-seeking in relation to cognitive models of vulnerability to depression have been proposed. For example, Joiner, Metalsky, Katz, and Beach (1999a, 1999b) discuss ways that excessive reassurance-seeking could be related to attributional style. They state that uncertain attributional style (the tendency to seek from others the explanations for causes of events) may be present in excessive reassurance-seekers and also that reassurance-seeking may be done to obtain explanations for the causes of events (i.e., it is hypothesized in this theory to be a primary motive for reassurance seeking). These authors also reason that depression would occur when significant others withdraw and high reassurance-seekers must create their own explanations for events. Without others to provide feedback, excessive reassurance-seekers are likely to become more uncertain and negative, a process that leads to hopelessness and depression. A related social-cognitive theory of depression provides support for the relationship between cognitive vulnerabilities (schemas and negative attributions), excessive reassurance-seeking, and depression in a social context (Sacco & Nicholson, 1999). Specifically, this theory indicates that social cognition (e.g., schematic person information and causal attributions) may influence a process in which excessive reassurance seeking elicits negative affect, rejection, and eventually, depressive symptoms in an individual.
Preliminary evidence for Joiner's theory was found in one investigation that found an association between uncertain attributional style, excessive reassurance-seeking, and depressive symptoms in a sample of 190 college students (Jacobson & Weary, 1999). Jacobson and Weary (1999) define causal uncertainty as "uncertainty about one's ability to understand cause and effect relations in the social world". Participants were administered the Reassurance-Seeking Scale (Joiner, Metalsky, & Schmidt, 1997), the Beck Depression Inventory (Beck, 1967), and the Causal Uncertainty Scale (CUS; Weary & Edwards, 1994) at one session. Findings from this study indicated that causal uncertainty may mediate the association between excessive reassurance-seeking and depressive symptoms. In addition, analysis of subscales of the CUS indicated that causal uncertainty related to the causes of one's own outcomes, but not related to other's outcomes, partially mediated the association between excessive reassurance-seeking and depression. The specificity of causal uncertainty to one's own outcomes is consistent with the internal causal attributions that serve as a risk factor in attributional style models of depressive vulnerability (Seligman et al., 1978; Abramson et al., 1989). The aforementioned theories and empirical evidence appear to support the integration of aspects of cognitive and interactional models of depressive vulnerability.

Unfortunately, research conducted on the relationship of excessive reassurance-seeking and depression have not included cognitive diatheses specified in the hopelessness model (Abramson et al., 1989); thus, it has not been determined that excessive reassurance-seeking leads to depressive onset in the absence of negative cognitive styles (e.g., negative attributional style, hopelessness, and negative life events.
leading to hopelessness depression). However, several investigations provide indirect support for the placement of excessive reassurance seeking within the hopelessness model.

As previously stated, Joiner and Metalsky (1995) found excessive reassurance-seeking was temporally antecedent to the onset of depressive symptoms and was related to stressors within this model. Joiner and colleagues (1999a) state that excessive reassurance-seeking is a contributory cause of depressive symptomatology. Although not tested in research, it is hypothesized that individuals who engage in frequent excessive reassurance-seeking become depressed because they become demoralized, as reassurance seeking does not produce self-assurance (Joiner et al., 1999a) and also because they tend to withdraw, thus increasing depressive symptoms (Coyne, 1976). Interestingly, the experiences of demoralization and withdrawal described in interactional models appear quite similar to the hopelessness diathesis component of Abramson and colleague's (1989) model. With this in mind it seems plausible to create a diathesis-stress model that includes excessive reassurance-seeking as a contributory causal factor that interacts with stressors (i.e., negative life events), is proximal to negative attributional style and distal to hopelessness in the development of depressive symptoms.

The Role of Cognitive Style within Cognitive Vulnerability Models

An important and implicit assumption of attributional style models of depression is that vulnerable individuals evaluate their environment, taking in information and making negative global, stable, and internal attributions about situations in their lives. However, research conducted using these models has not yet addressed the question of
whether a difference in evaluative responding affects an individual's tendency to make attributions. It is possible that depressed (or vulnerable) individuals are evaluating their environment and using this information to make attributions and also that individuals who evaluate their environment more would be have a greater opportunity to make attributions, both positive and negative. However, it is also possible that an individual's tendency to actively process external evaluation decreases as their attributions become more automatic. For example, if an individual develops a negative attributional style they may evaluate external information in a less active manner, allowing negative attributions to guide their processing of the information. In either case it is likely that the degree to which an individual engages in evaluation will affect vulnerability to depression.

The Need to Evaluate

Jarvis and Petty (1996) have examined differences among individuals' tendency to evaluate their external environment. These researchers propose a trait construct called the Need to Evaluate. The Need to Evaluate can be described as the dispositional tendency to engage in evaluation of one's external environment. Jarvis and Petty propose that NtE may be related to depression in that both constructs are evaluative in nature and call for future research that investigates the role of NtE in the development and maintenance of depression.

NtE is likely to influence the onset of depression for many reasons. First, according to Jarvis and Petty (1996), evaluation is an adaptive, dominant, automatic human response. These authors reason that evaluating one's environment is adaptive
because it enables them to cope with life outcomes, increases control over outcomes, facilitates social interactions, and protects and enhances one's self-image and esteem. Interestingly, these factors are often unavailable in those at risk for development of depression. Thus, high levels of NtE may be a stable factor that serves to protect vulnerable individuals from onset of depression. It is also possible that individuals with negative attributional style do not frequently evaluate their environment, but instead make automatic negative attributions without processing external stimuli. This scenario would also show high levels of NtE as protective, because engaging in evaluation may prevent an individual from making negative attributions automatically. Mid-levels of NtE occur when an individual does not demonstrate a stable tendency to evaluate aspects of their external environment. Mid-levels of NtE do not represent a stable style and thus are not likely to act as a stable diathesis in the prediction of depressive symptomatology. Although instability does not preclude mid-levels of NtE from serving as a diathesis (e.g., much in the way that mood states may influence depressive onset), it is probable that unpredictable variability in one's tendency toward external evaluation would not play a major (or even measurable) role in influencing an individual's risk for depression.

Conversely, it is possible that frequent evaluation of one's environment could be a risk factor for individuals who are vulnerable to depression. As depressed individuals (and those at risk for depression) are hypothesized to make negative causal explanations themselves and their environments (Abramson et al., 1978; Abramson et al, 1989), it is possible that NtE functions in depressed individuals to create or provide access to negative attributions. For example, Jarvis and Petty (1996) hypothesize that there are
individual differences in the propensity to evaluate one’s environment and that individuals who do evaluate their environment may have differences in exactly what they evaluate based on personal experiences. It may be that individuals with negative attributional style derived from early experiences may tend to engage in evaluation of negative aspects in their environment that confirm, and thus perpetuate, these diatheses. It may also be that the mere frequency of evaluation provides more opportunity for negative information to be processed and used to create negative attitudes. In addition, following the logic of the mood-state theory, it may be that while an individual is engaging in evaluation of negative environmental stimulus, negative attributions may be more accessible.

Jarvis and Petty (1996), note that identifying one’s tendency to engage in or avoid evaluative responding may help to determine ability to cope with stress and negative life events. If this is the case, then identifying level of NtE among vulnerable individuals may help to determine the influence of stressors within diathesis-stress models of depression (including Beck, 1967, 1976; Abramson, 1978; Abramson et al., 1989).

It is important to distinguish the need to evaluate from the need for cognition (Cohen et al., 1955; Cacioppo & Petty, 1982), as these constructs are somewhat similar. As described by Jarvis and Petty (1996) Need for Cognition (as measured by the need for cognition scale; Cacioppo & Petty, 1982) refers to the chronic need of individuals to engage in and enjoy effortful thought. Although it may seem that engaging in effortful thought is necessary in order to evaluate one’s external environment, or that motivation to evaluate is likely only in individuals with high levels of Need for Cognition, research has
demonstrated only a moderate association between the two constructs (Jarvis & Petty, 1996). Thus, it is reasonable to assume that the Need to Evaluate and the Need for Cognition are not the same.

As previously noted, NtE has not been tested within cognitive models of vulnerability to depression. Therefore, it is plausible that either high or low levels of this trait variable may serve as either a risk or protective factor in relation to depressive onset and maintenance. All potential roles of NtE will be examined in the present study.

Summary of Potential Moderators

The addition of cognitive and interactional styles to cognitive models of vulnerability may increase our understanding of depressive onset, maintenance, and relapse. Although attributional style theories of depression seem to include behavior in their explanation of depressive onset (behavior must take place for attributions to occur), this is not clearly stated within any cognitive models. The examination of excessive reassurance-seeking style, a factor that has already been associated with depression, may provide a more complete understanding of how interactional styles interacts with cognitive diatheses in relation to depressive etiology.

In addition, attributional style models of depressive vulnerability assume that individuals with negative attributional styles are evaluating their environment. However, it does not appear that existing models of cognitive vulnerability to depression (those that include attributional style as a cognitive diathesis) have examined the impact of the degree to which evaluation is occurring. Variation in an individual’s tendency to evaluate their external environment has been examined (NtE; Jarvis & Petty, 1996), but
this has yet to be applied to theories of depressive vulnerability. It is possible that
differences in evaluative responding affects the frequency or quality of attributions
formed by cognitively vulnerable individuals and thus, it seems plausible to examine
need to evaluate in the context of cognitive models of depression.

Study Hypotheses

Based on past research several hypotheses were developed which predict the
relationship between study variables including cognitive diatheses (attributional style and
hopelessness), stressors (negative life events), proposed moderators including reassurance
seeking and need to evaluate, and depressive symptomatology. Variables that did not
contribute significantly to variance in preliminary analyses were not utilized in further
analyses that examine more specific hypotheses related to relationships among
independent and dependent variables.¹

Hypothesis 1 – Vulnerability Markers

As previously indicated, several cognitive theories propose that negative
attributional style (Seligman, 1975; Abramson et al., 1978; and Abramson et al., 1989)
and hopelessness (Abramson et al., 1989) are involved in the development of depressive
symptomatology. These diatheses have also been associated with the development of
symptomatology related to the hopelessness subtype of depression (Abramson et al.,
1989).

¹ For all hypotheses stated below, factors including coping style, social support, anxiety, attributional style,
hopelessness, excessive reassurance-seeking, and need to evaluate refer to scores on these measures taken
at Time 1. One exception is life events, which will examined using the average of scores taken at baseline
and time 2. Measures of depression will be examined using Time 2 scores while accounting for initial
levels of depression taken at Time 1.
Although the findings of research investigating attributional style and hopelessness as cognitive diatheses are mixed, there are a number of well-designed prospective studies that have demonstrated these factors' involvement in the etiology of depression (e.g. the Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) project; Alloy, Abramson, Whitehouse, et al., 1999). Therefore, it was hypothesized that cognitive vulnerabilities (as measured by scores on the CSQ and BHS at Time 1) would predict level of depressive symptoms (as measured by scores on the Time 2 BDI-II) when entered in a regression model. Specifically, it was predicted that main effects for T1 hopelessness and T1 inferential style, as well as the T1 interaction of these terms, would account for a significant amount of variance in predicting T2 depression, even when controlling for initial levels of depression (as measured by T1 BDI-II scores).

As previously noted, cognitive vulnerabilities have been found to predict the hopelessness sub-type of depression as well as depression in general. Therefore, it was hypothesized (as measured by scores on the CSQ and BHS at Time 1) would predict level of hopelessness depressive symptoms (as measured by scores on the Time 2 HDSQ) when entered in a regression model. Specifically, it was predicted that main effects for T1 hopelessness and T1 inferential style, as well as the T1 interaction of these terms, would account for a significant amount of variance in predicting T2 hopelessness depression, even when controlling for initial levels of depression (as measured by T1 HDSQ scores).
Hypothesis 2 – Full model and moderation

As previously noted, excessive reassurance-seeking and need to evaluate have not yet been tested within attributional style models of depression. However, each factor is likely to contribute to the development of depressive symptoms. Although need to evaluate has not been integrated into existing theories of cognitive vulnerability to depression, excessive reassurance-seeking has been hypothesized to contribute to depressive onset and course. Thus, it is plausible that while need to evaluate and excessive reassurance-seeking may be correlated with depressive symptomatology, they may only elicit depressive symptomatology in the presence of cognitive vulnerabilities (e.g., negative attributional style and hopelessness). In sum, need to evaluate and excessive reassurance-seeking maybe contributory but neither necessary nor sufficient causes of depression. As attributional style models of depression have been found to specifically predict the hopelessness subtype of depression, initial levels of need to evaluate and reassurance-seeking were examined within cognitive vulnerability models of hopelessness depression. Therefore, hopelessness depression as measured by the T2 HDSQ was utilized as the dependent variable in all regression analyses conducted within hypothesis two.

Based on previous literature and research and the rationale stated above regarding the association between need to evaluate, excessive reassurance-seeking, cognitive vulnerability markers, and hopelessness depression, it was predicted that the three-way interaction between for need to evaluate (as measured by T1 scores on the NES) and cognitive vulnerabilities (as measured by T1 BHS and T1 CSQ scores) would predict
hopelessness depressive symptomatology (as measured by T2 HDSQ scores), even when accounting for initial levels of depressive symptoms. It was also predicted that the three-way interaction between excessive reassurance-seeking (as measured by T1 scores on the DIRI-RS) and cognitive vulnerabilities would predict Time 2 hopelessness depressive symptoms when controlling for initial levels of hopelessness depressive symptomatology.

If models from hypothesis one were fully supported, the variance accounted for in each of these interactions would have been compared with the variance accounted for by the interaction of vulnerability markers (BHS and CSQ) in predicting hopelessness depression (from hypothesis one). If one or both of the three-way interactions between vulnerability markers and either need to evaluate or excessive reassurance-seeking were found to account for a greater amount of variance than the regression equation including the interaction of inferential style and hopelessness in predicting hopelessness depression, these variables would have been tested to see whether they met criteria for moderation.

As noted by Baron and Kenny (1986), moderation is supported if three criteria are met. First, the interaction between the predictor and moderator variable should be significantly correlated with the dependent variable. Next, predictor and moderator variables should be uncorrelated (as this may prevent the delineation of a clear interaction term). Finally, these authors state that moderator variables should not be causally related to predictor variables (they are not antecedents or consequences of predictors). Thus it appears that if need to evaluate and excessive reassurance-seeking are to qualify as moderators (as specified by Baron and Kenny), certain relationships between each
moderator and cognitive predictor variables of inferential style and hopelessness (as measured by scores on the CSQ and BHS) can be hypothesized.

With this in mind, it was hypothesized that the interaction between need to evaluate and excessive reassurance-seeking and cognitive diatheses (inferential style and hopelessness) would be significantly correlated with hopelessness depression (scores on the HDSQ at time 1). In addition, it was hypothesized that need to evaluate and excessive reassurance-seeking would contribute significantly to the prediction of hopelessness depression at Time 2 when entered into a regression equation following levels of hopelessness depression at Time 1 and cognitive vulnerability factors (negative attributional style and hopelessness). If these first two conditions were met then it is likely that need to evaluate and reassurance seeking were functioning as moderators. If moderation was indicated then it would have been assumed that need to evaluate and excessive reassurance-seeking were not significantly correlated with cognitive vulnerability (scores on the CSQ and BHS).

**Hypothesis 3 – Main effects of need to evaluate and excessive-reassurance seeking**

Variables proposed to moderate the relationship are likely to be related to the development and course of depressive symptomatology. As noted above, research examining excessive reassurance-seeking has demonstrated that this factor is a contributory cause of depressive symptomatology and major depressive disorder (Joiner et al., 1999a); however, research in this area has not yet tested whether cognitive diatheses such as those specified in attributional style models contribute to the association between excessive reassurance-seeking and depressive onset or course.
Another factor proposed to moderate the relationship between cognitive diatheses and depressive onset and maintenance is the need to evaluate (Jarvis & Petty, 1996). While this variable has never been tested in relation to depression, it is likely to be involved in depressive onset and course as a result of its potential influence on one’s access to negative attributions.

Thus it was probable that need to evaluate and excessive-reassurance seeking would have been predictive of depressive symptomatology both within and independently of attributional style models. The previous hypothesis tested whether need to evaluate and/or excessive reassurance-seeking added to the predictive power of the hopelessness model of depression and also whether they served as moderators within this model. However, results from hypothesis one would not have provided information as to whether main effects of need to evaluate or excessive reassurance-seeking predicted hopelessness depression. In order to replicate previous findings (e.g., Joiner et al., 1999) it was important to determine whether main effects of excessive reassurance-seeking predict general depression. Although a relationship between need to evaluate and general depression has not yet been reported in the literature this variable was also tested for main effects. Findings from past literature suggest that both general depression and the hopelessness subtype of depression can be predicted by multiple etiological factors. Thus, it was expected that significant main effects found for need to evaluate and/or reassurance seeking would account for some, but not all of the variance in predicting symptoms of depression and hopelessness depression.
With this in mind, it was hypothesized that need to evaluate (as measured by scores on the T1 NES) would show main effects when regressed on general depressive symptoms (as measured by scores on the T2 BDI-II) after controlling for initial levels of general depressive symptomatology (as measured by T1 BDI-II scores). In addition, excessive reassurance seeking (as measured by T1 scores on the DIRI-RS) was predicted to show main effects when regressed on depressive symptoms (as measured by scores on the T2 BDI-II) after controlling for initial levels of depressive symptoms (as measured by T1 BDI-II scores).

As previously mentioned, need to evaluate and excessive reassurance seeking are likely to be involved in access or perpetuation of negative attributional style. Since negative attributional style has been associated with symptoms of hopelessness depression, it is plausible that both need to evaluate and excessive reassurance-seeking would also be related to symptoms of this depressive subtype.

As such, it was predicted that need to evaluate (as measured scores on the T1 NES) would yield a significant main effect when regressed on symptoms of hopelessness depression (as measured by scores on the T2 HDSQ) after controlling for initial levels of hopelessness depressive symptoms (as measured by T1 HDSQ). Also, excessive reassurance seeking (as measured by scores on the T1 DIRI-RS) was predicted to result in a significant main effect when regressed on symptoms of hopelessness depression (as measured scores on the T2 HDSQ) after controlling for initial levels of hopelessness depression (as measured by T1 HDSQ scores).
Hypothesis 4 – Specificity

In addition, depression has been found to be highly comorbid with anxiety (Brown & Barlow, 1992). Thus it is plausible that cognitive diatheses specified in attributional style models of depression act as vulnerabilities for anxiety as well as depression. However, as a number of studies have demonstrated, diatheses specified in cognitive models are specific to general depression as well as hopelessness depression (Joiner & Metalsky, 1992; Alloy & Clements, 1998). Thus, it is likely that vulnerability factors (as measured by scores on the CSQ and BHS) in participants would be specific to level of general depressive symptoms or symptoms of hopelessness depression (as measured by scores on the BDI-II and HDSQ respectively).

The role of cognitive vulnerability factors as predictors of anxiety is still a focus of study. Factors that have been found to contribute to depressive etiology such as maladaptive cognitive styles and high levels of stress have been shown to be risk factors for anxiety as well (Beck & Emery, 1985). The need to evaluate, examined in this study, has been found to be predictive of anxiety (Klocek, Carmin, Gillock, Shertzer, & Raja, 1999). Further, some of the same techniques (e.g., identification and modification of negative core beliefs and automatic thinking) utilized in cognitive-behavioral treatments of depression (e.g., Beck, 1976) are also used in the treatment of anxiety and have been found to be effective (Butler, Fennell, Robson, & Gelder, 1991). Therefore, depressive vulnerability markers, hopelessness and inferential style were examined as potential predictors of anxiety. Thus, it was predicted that each factor (T1 hopelessness and T1 inferential style) would yield a significant main effect when entered into a regression
equation predicting anxious symptoms (as measured by T2 BAI scores) after accounting for initial levels of anxiety (as measured by T1 BAI scores). It was also predicted that the interaction between T1 hopelessness and T1 inferential style would predict T2 anxiety after accounting for initial levels of anxiety.

**Hypothesis five - Negative Life Events Within the Full Model**

As previously noted, the attributional style model for depression is a diathesis-stress model. According to cognitive models (e.g., Abramson et al., 1989) the presence of some life stressor is a necessary part of the pathway from cognitive diatheses to depression. As such, it is probable that a high degree of life stressors (as measured by scores on the LES) would strengthen the relationship between cognitive vulnerability factors, need to evaluate and excessive reassurance seeking and change in depressive symptoms. In addition, previous research has found negative life events to be a component of the cognitive diathesis-stress model that leads to hopelessness depression (e.g., Alloy, Abramson, Whitehouse et al., 1999). If hypotheses 1-3 were fully supported, assumptions regarding the influence of negative life events (stated below) would have been tested for interaction effects within the full model. However, as predictions stated below create four-way interactions, sufficient power would have been necessary to proceed with these analyses.

It was assumed that life stressors (as measured by LES scores) would interact with cognitive vulnerability markers (as measured by T1 CSQ and T1 BHS scores) and the need to evaluate (as measured by T1 NES scores) to predict the level of depressive symptoms (as measured by T2 BDI-II scores) and symptoms of hopelessness depression.
(as measured by T2 HDSQ scores) after accounting for initial levels of depressive symptoms (T2 BDI-II and T2 HDSQ scores).

It was also assumed that life stressors (as measured by scores on the LES) would interact with cognitive vulnerability markers (as measured by scores on T1 CSQ and T1 BHS) and excessive reassurance-seeking (as measured by T1 DIRI-RS scores) to predict the level of depressive symptoms (as measured by T2 BDI-II scores) and symptoms of hopelessness depression (as measured by T2 HDSQ scores) over the six-week time period (Time 1 to Time 2) even when controlling for initial levels of general and hopelessness depression symptoms respectively.

Assumptions about Social Support and Coping Style within the Full Model

As a result of the complexity and exploratory nature of hypotheses one through four, (stated above) predictions and analyses regarding social support and coping data collected during the present research will be pursued in subsequent research. At this time background information related to these factors will be discussed and assumptions will be provided in an attempt to guide future research.

A large body of literature demonstrates the influence of social support and coping style in the etiology and maintenance of depression (Finch et al., 1997; Folkman & Lazarus, 1986; Coyne et al, 1981). These factors have been found to be strongly associated with depression (Folkman and Lazarus, 1986; Coyne et al, 1981). However, neither factor has been found to account for more variance than cognitive diatheses (e.g., attributional style and hopelessness) in diathesis-stress models of depression. Thus it was assumed that within cognitive models of general depressive symptomatology, variance
accounted for by cognitive vulnerability factors (attributional style and hopelessness) and by need to evaluate and excessive reassurance-seeking, would not be significantly reduced by differences in level of perceived social support (as measured by scores on the SSQ-6) or coping style (as measured by scores on the COPE).
Chapter Two

Method

Participants

Participants were 129 introductory psychology students recruited through the Psychology 100 subject pool. Power analyses were conducted using the Sample Power statistical software package. Power estimates indicated that a sample of 184 participants should be included to obtain power of .80.

To be consistent with participant criteria of previous research which examines cognitive models of vulnerability to depression (e.g. The Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) project; Alloy, Abramson, Whitehouse et al., 1999), participants ranged in age from 18 – 65 and included both males and females.

Materials

Each participant received two copies of the informed consent form prior to each session. The informed consent form stated the purpose of the project, information regarding limits of confidentiality, benefits of participation (credits for their Psychology 100 course), penalties for failure to attend a scheduled session without informing project staff (deduction of credits for psychology 100 course), legal responsibilities of project staff and the University of Montana, information regarding individuals or agencies to contact in case of concerns, emergencies, distress, or injury, and provided a place for students to sign should they choose to participate in the study. Participants created a unique study identification code on a separate form (the participant identification form). All participants kept one copy of the informed consent form and returned a second,
signed copy following each session. As the participant identification form was the only form that contains the participant's name and identification number, it is kept in a locked filing cabinet separate from all other materials. The participants' informed consent forms are also stored in a locked file cabinet, separate from the participant information form and data. Only project staff had access to study materials.

**Measures**

**Demographic Questionnaire**

This questionnaire was used to compare differences in participant characteristics across depression levels. It consisted of multiple items regarding participant’s identification number, age, race/ethnicity, gender, years of education completed, marital status, current physical illness, and previous or current psychiatric diagnoses.

**Cognitive Style Questionnaire**

The Cognitive Style Questionnaire (CSQ; Abramson et al., 1990) is a 24-item assessment of participant’s styles for inferring causes, consequences, and self-characteristics when faced with negative life events in both achievement and interpersonal domains that are featured as vulnerabilities to depression in the hopelessness model. The CSQ is a revision of the Attributional Style Questionnaire (ASQ; Seligman et al., 1979) with major changes including the addition of more hypothetical life events (12 positive and 12 negative events, six in each content domain). The CSQ also added assessment of dimensions of consequences and self-characteristics to the dimensions of attributional style measured by the original ASQ, internality, stability, and globality. Participants are asked to read each item, write down the one
major cause of the event and then rate the cause on a one to seven scale, separately for each dimension. The scoring procedure for this measure the same as the procedure for scoring the Inferential Style Questionnaire (ISQ) Rose, Abramson, Hodulik, Haberstadt, and Leff (1994). Participant ratings for each for the 12 negative items are summed by dimension resulting in six dimensional scores. Of these scores, dimensions of inferred stable causes, inferred global causes, inferred negative consequences, and inferred negative characteristics of the self are summed to form an aggregate score called “inferential style”. Higher scores correspond to more negative attributional styles. The CSQ has been found to be internally consistent with coefficient alpha’s ranging from .87 (interpersonal subscale) to .89 (achievement subscale) (Alloy et al., 1997).

**Beck Hopelessness Scale**

The Beck Hopelessness Scale (Beck, Weissman, Lester, & Trexler, 1974) measures symptoms of hopelessness (as a vulnerability factor). The BHS is a 20-item inventory that measures pessimistic attitudes about the future (hopelessness). Each item is rated as true or false. Items are scored 1 (for a true response) and 0 (for a false response). Item scores are added and yield a total score ranging from 0 to 20, with higher scores reflecting greater hopelessness. Internal consistencies (using Kuder-Richardson reliabilities; KR-20) for the BHS have been found to be high across several clinical samples including suicide ideators, suicide attempters, alcoholics, heroin addicts, single-episode Major Depression Disorders, recurrent-episode Major Depression Disorders, and Dysthymic Disorders (KR-20 = .92, .93, .91, .82, .92, .92, and .87 respectively; Beck & Steer, 1988). These authors also demonstrated concurrent and construct validity for the
BHS with these populations. Reliability of the BHS has been reported for a college
student population (KR-20 = .65; Durham, 1982).

Need to Evaluate Scale

The Need to Evaluate Scale (NES; Jarvis & Petty, 1996) is a 16-item
questionnaire that measures an individual’s implicit motivation to evaluate his or her
external environment. Each item on the NES is rated on a one to five scale (for each item
a score of one indicates “extremely uncharacteristic” of the participant and five indicate
“extremely characteristic”). Scores on the NES range from 16 to 80. Previous studies
have designated cutoff scores (using tertiary splits) ranging from 46 to 48 and below to
indicate low NtE and 56 to 58 and above to indicate high NtE; Jarvis & Petty, 1996). The
scale has been found to be internally consistent (Chronbach’s alpha = .87; Jarvis & Petty,
of the NES.

Depressive Interpersonal Relationships Inventory – Reassurance-Seeking Sub-Scale

The Depressive Interpersonal Relationships Inventory – Reassurance-Seeking
sub-scale (DIRI-RS; Joiner & Metalsky, 1998) is a four-item sub-scale of the DIRI
measuring the tendency to excessively seek reassurance from others as to whether they
truly care (reassurance seeking). Each item is rated on a one to seven scale and is
averaged across items with higher scores reflecting increasing reassurance seeking.
Studies using the DIRI-RS have reported coefficient alphas ranging from .81 to .88
(Joiner et al., 1992; Joiner, Katz, & Lew, 1999). Joiner and Metalsky (1992) found the
DIRI-RS to be valid using a sample of 353 college students.
Beck Depression Inventory – II

Measurement of depressive symptomatology was be done using the Beck Depression Inventory – II (BDI-II; Beck, Steer, & Brown, 1996). This is a 21 item, self-report questionnaire that measures affective and cognitive aspects of experienced depression. Each item is rated on a 0 – 3 scale with summary scores ranging between 0 and 63. The internal consistency of this inventory has been cited as coefficient alpha = .93 among college students, .92 among depressed outpatients (Beck et al., 1996). Beck et al., (1996) also found support for content, construct, and factorial validity in a large college student sample. Convergent validity has also been demonstrated for this measure in a sample of 1,022 college students (Dozois, Dobson, & Ahnberg, 1998). Item number nine on the BDI-II assesses suicidality. As such, this item was checked for responses indicating suicidal ideation. Upon finding any indication of suicidal ideation, project staff implemented precautionary measures specified in the procedure section of this manuscript.

Hopelessness Depression Symptom Questionnaire

Symptoms of hopelessness depression was measured using the Hopelessness Depression Symptom Questionnaire (HDSQ; Metalsky & Joiner, 1997). The HDSQ is a 32-item self-report measure of hopelessness depression. Items consist of four statements, rated 0 to 3, regarding a component of hopelessness depression. The scale consists of eight sub-scales with scores for each ranging from 0 to 12 and a total score ranging from 0 to 96 with higher scores indicating higher levels of hopelessness depression. The HDSQ has been found to have high internal consistency for the full scale (alpha = .93)
and for sub-scales ranged from alpha = .70 (motivational deficit) to alpha = .86 (anergia and suicidality) and also to have moderate test-retest reliability over several weeks (r = .58) (Joiner, 1999). Joiner and Metalsky (1997) found demonstrated validity for the HDSQ with a sample of 435 college students. Items 29, 30, 31, and 32 of the HDSQ assess suicidality. As such, these items will be checked for responses indicating suicidal ideation. Upon finding any indication of suicidal ideation, project staff implemented precautionary measures specified in the procedure section of this manuscript.

_Hammen Perception of Negative Life Experiences Survey_

Stressful life experiences were measured using the Hammen Perception of Negative Life Experiences Survey (HPNLES; Hammen, Marks, Mayol, & DeMayo, 1985). This is a 120-item self-report measure of life changes assessed by positive and negative ratings of life events and an impact rating for each reported item that occurred over the past four weeks. Life events are categorized into six sub-sections including the following: 1) work and or school, 2) finances, 3) health, 4) romantic relationships, 5) home, friends, and family life, and 6) personal events. For each item that is endorsed, the participant indicates the valence of that event by rating it on a Likert-type scale ranging from -3 (extremely negative impact) to +3 (extremely positive impact). Test-retest reliability has been reported for the HPNLES over a five-week period (r = .79; Klocek, Oliver, & Ross, 1997). Since previous research has indicated that both number and perceived impact of stressors contribute to individual interpretation of stressful events, this measure appears to be adequate for assessment of this construct.
Social Support Questionnaire-6

Level of social support was measured by the Social Support Questionnaire-6 (SSQ-6; Sarason, Sarason, Shearin, & Pierce, 1987). The SSQ-6 is a 6-item questionnaire that measures perception of and satisfaction with social support. Each item is composed of two parts. The first part indicates the number of individuals that the participant believes are available for support in an array of situations while the second part is a six-point Likert scale that measures satisfaction with perceived support. The questionnaire's internal reliability has been found to be between .90 and .93 (Sarason et al., 1987).

COPE

Measurement of coping style was done using the COPE inventory (COPE; Carver, Scheier, & Weintraub, 1989). The COPE is a 60-item questionnaire that was developed to assess the different ways in which people respond to stress. Items are rated on a four point Likert-type scale (from 1 to 4) and various items are summed to formulate each of the measure's 15 sub-scales (each sub-scale is composed of 4 items from the measure). Sub-scales include positive reinterpretation and growth, mental disengagement, focus on and venting of emotions, use of instrumental support, active coping, denial, religious coping, humor, behavioral disengagement, restraint, use of emotional social support, substance use, acceptance, suppression of competing activities, and planning. Scores on each sub-scale range from 4 to 16 with higher scores reflecting stronger tendencies to utilize a particular form of coping when under stress. Test-retest reliability over eight weeks using an undergraduate student population has been found for
each sub-scale, ranging from $r = .46$ (suppression of competing activities) to .86 (religion coping) (Carver et al., 1989). For this investigation subscales including focus on and venting of emotions, behavioral disengagement, use of instrumental support, planning, active coping, and use of emotional support were utilized to measure coping style at T1 and T2. These subscales were selected because they are similar to types of coping that have been found to either buffer or increase risk for depressive onset in previous literature (e.g., Coyne et al., 1981). The COPE has been found to be highly internally consistent with Cronbach’s Alpha ranging from .63 to .92 across sub-scales (an exception is the mental disengagement sub-scale, with Cronbach’s Alpha equal to .45) (Carver et al., 1989). Convergent and discriminant validity has been demonstrated for this measure (Carver et al., 1989).

Beck Anxiety Inventory

As depressive symptoms are often highly correlated with measures of anxiety (Brown & Barlow, 1992; Sartorius, Ustun, Lecrubier, & Wittchen, 1996), the Beck Anxiety Inventory (BAI; Beck & Steer, 1990; Beck, Epstein, Brown, & Steer, 1988) was utilized to determine if reported symptoms are related to depression, anxiety, or both for each participant. The BAI is a 21-item measure of anxiety developed with a psychiatric population. Each item is rated on a 0 – 3 scale with summary scores ranging between 0 and 63. It has been found to be internally consistent (coefficient alpha ranging from $= .92$ - .94) and reliable (test-retest reliability ranging from $r = .67 - .75$) (Beck, Epstein, Brown, & Steer, 1988; Fydrich, Dowdall, & Chambless, 1992). Support for discriminant validity has also been shown (Fydrich et al., 1992).
Procedure

All participants attended two sessions (six weeks apart) proctored by project staff. Participants were assessed in large groups at previously reserved classrooms on the campus of the University of Montana. They received a questionnaire packet including a participant identification form where they created a study identification code, an informed consent form, CSQ, BHS, HDSQ, BDI-II, BAI, SSQ-6, DIRI-RS, NtE, COPE, HPNLES, and a demographic questionnaire. Questionnaires within each packet were randomized to control for order effects. Each participant also received a number two pencil and custom designed opscan answer sheets prepared on NCS Design Expert. These sheets consisted of sections that correspond to each measure. Proctors provided information related to confidentiality, participant rights, and instructions on how to complete project measures. Participants read and signed informed consent and complete questionnaires. It took approximately 2 hours to complete these forms.

At the end of the first session, all participants handed in completed packets, participant identification form and a signed copy of the informed consent form (participants kept one copy of the consent form). All participants signed a form, which enabled project staff to give them experimental credit for participation. They then chose from one of two days (these days were selected in advance by the PI and were approximately six weeks after the initial session) when they would return to participate in the second session. Each participant was then provided with a form with information including the time, date, and location of their next session, as well as a phone number where the project coordinator could be reached, and the name of the project. All data and
informed consent forms were collected by project staff and returned to the project office located in the department of psychology. All participant answer forms were reviewed by administrators of the assessment sessions for responses indicating suicidal ideation on the Beck Depression Inventory – II, item #9 and on the Hopelessness Depression Symptom Questionnaire, item #29, 30, 31, and 32. As stated in the informed consent, upon finding any indication of suicidal ideation, project staff immediately contacted the PI – a graduate student in clinical psychology (supervised by a licensed clinical psychologist). In accordance with ethical responsibility, the PI then broke confidentiality, contacted the student participant directly, and assessed suicidal risk. Pending the outcome of this evaluation, further referrals or steps were taken. Subsequent to this procedure, all participant identification forms were placed in a locked file cabinet separate from the rest of the data. Only the PI had access to these sheets. Data and informed consent forms were also kept in a locked file cabinet separate from each other and at no time during data analysis were the identifying information contained on participant identification forms associated with the data or with the informed consent forms provided by participants.

Participants returned to complete participation on the date they had selected. As a reminder, general announcements of the times, dates, and locations of the second session along with the project coordinator’s campus phone number were announced in all psychology 100 classes by course instructors the week prior to these sessions. The second session was also proctored by project staff who provided information related to confidentiality, participant rights, and instructions on how to complete the informed consent form, participant identification form, and project measures. Participants received
measures (CSQ, BHS, HDSQ, BDI-II, BAI, SSQ-6, DIRI-RS, NES, COPE, and HPNLES), along with an opscan form recording their answers and provided their identification number, and a number two pencil. Participants once again received the participant identification form and two copies of the informed consent form, which they read and signed prior to participation in this portion of the study. All participants completed study measures, participant identification form, and one copy of the informed consent, which was returned to project staff when they finished. All participants signed a form, which enabled project staff to give them course credit for participation. Participants were thanked and given a debriefing form, which explained the purpose, hypotheses, and potential application of the present study. Procedures described above related to indication of suicidal ideation were followed after the second administration as were procedures related to storage of informed consent forms, participant identification forms, and storage and analysis of data.
Chapter Three

Results

Means and standard deviations for each measure at each time period can be seen in Table 1. 57.4% of participants were male and 42.6% were female. Ages of participants ranged from 17 to 52 years with a mean age of 20.73. All participants reported education levels of 12 years or higher with a mean of 12.96 years. 87.6% of this sample was Caucasian, 4.7% were Native American, 3.9% were Hispanic, 2.3% were other, and 1.6% were African American. The majority of this sample indicated that they were single (89.9%) and that they have not been diagnosed with physical (83.7%) or mental illnesses (85.8%).

To ensure that this sample was representative of other college samples utilized in previous research investigating cognitive models of depression, demographic information provided by this sample was compared with demographics reported by authors of the CVD study (Alloy & Abramson, 1999). It appears that the sample utilized in the current study is comparable to CVD participants on measures of age and identified ethnicity; however, this sample was composed of a greater percentage of males than was the CVD sample. In addition, there were significant, positive, low magnitude correlations between
gender and all dependent measures. As a result of these finding and findings of gender differences in depression among males and females in general (Nolen-Hoeksema et al., 1987), independent samples t-tests were conducted for each dependent variable (T2 BDI-II, T2 HDSQ, and T2 BAI) as well as for all predictor variables (T1 BHS, T1 CSQ, T1 NES, T1 DIRI-RS and T1 interaction between BHS and CSQ) utilizing gender as a grouping variable.

There are significant differences between means for males and females from this sample on all dependent measures (see Table 2). Specifically, females scored significantly higher than males on all dependent measures. Previous researchers have also reported significantly higher mean scores for females on measures of depression and anxiety (for the BDI-II, Beck et al., 1996; for the BAI, Blalock and Joiner, 2000). Analysis of data from Beck and colleagues (1996) entire college sample were conducted during the development of the BDI-II despite reported significant mean differences for gender. There are no significant differences between males and females on mean scores of predictor variables. t-values ranged from $t(104) = -1.679, p > .10$ to $t(103) = .219, p > .10$. Thus, it is likely that gender differences found for this sample are representative. Findings related to significant group differences between males and females on dependent measures are presented in Table 2. As a result of reduced power and small sample size, regression analyses were not conducted separately for males and females.

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Insert Table 2

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In addition, overall mean scores on dependent measures (BDI-II, HDSQ, and BAI) collected at T1 and T2 of this study were compared with means from the college student samples utilized in the development of the BDI-II (Beck et al., 1996) and HDSQ (Joiner & Metalsky, 1997) as well as from a large college sample which utilized the BAI as a dependent measure of anxiety symptoms (Creamer, Foran, & Bell, 1994). This information is summarized in Table 3. It appears that T2 mean scores from this sample (those scores utilized as dependent variables) were lower than those of the comparison group on the BDI-II, higher than the mean scores of the comparison group on the HDSQ (though these scores appear fairly similar), and lower than those of the comparison group on the BAI; however, T1 mean scores for this sample appear to be quite similar to normative samples on the BDI-II and BAI but differ substantially on the HDSQ. Mean scores obtained at T1 and T2 do not demonstrate consistent differences when compared to normative samples. Thus, there is not clear evidence to suggest that this sample is systematically different from normative samples.

There were significant differences between T1 and T2 means for all dependent variables including general depression (T1 BDI and T2 BDI) (t (102) = 5.403; p < .001), hopelessness depression (T1 HDSQ and T2 HDSQ) (t (103) = 6.661; p < .001), and anxiety (T1 BAI and T2 BAI) (t (104) = 5.369; p < .001), with T1 means consistently significantly higher than T2 means. However, there were no significant differences between T1 means for participants who completed the study versus those who did not on measures of general depression (t (124) = .079; p > .10), hopelessness depression (t (125) = .362; p > .10), or anxiety (t (126) = -1.051; p > .10).
Zero-order correlations between measures can be seen in Table 4. Significant and moderate bi-variate correlations were found between all measures utilized in regression analyses including dependent variables of anxiety (BAI) at T1 and T2 ($r (105) = .67; p < .01$), general depression (BDI-II) at T1 and T2 ($r (103) = .67; p < .01$), hopelessness depression (HDSQ) at T1 and T2 ($r (104) = .71; p < .01$), and also predictor variables of hopelessness (BHS) at T1 and T2 ($r (106) = .69; p < .01$), inferential style (CSQ) at T1 and T2 ($r (104) = .57; p < .01$), and excessive reassurance-seeking (DIRI-RS) at T1 and T2 ($r (106) = .59; p < .01$). The zero-order correlation for the NES between T1 and T2 was somewhat lower but still significant ($r (106) = .39; p < .01$).

Significant and moderate correlations were also found between each predictor variable at T1 (hopelessness, inferential style, and excessive reassurance-seeking) with each dependent variable (depression, hopelessness depression, and anxiety) at T2, except for need to evaluate, which was not significantly related to any T2 dependent variable. These correlations tended to support aspects of study hypotheses and thus, are discussed specifically in hypotheses sections below. Some significant correlations were found between T1 predictor variables that were excluded from analyses due to low power (e.g., coping style variables, social support variables, and negative life events) and T2
dependent variables. These relationships tended to be low and less significant than predictors utilized in regression models. Two coping styles were associated with T2 general depression including T1 active coping \( (r(104) = -.20; p < .05) \) and behavioral disengagement \( (r(104) = .28; p < .01) \). One other coping style, behavioral disengagement \( (r(104) = .44; p < .01) \), was related to T2 hopelessness depression. No coping styles were correlated with T2 anxiety. Perceived availability of social support at T1 was negatively related to T2 general depression \( (r(95) = -.26; p < .05) \). T1 social support was not significantly related to T2 hopelessness depression or T2 anxiety. T1 negative life events were positively associated with T2 general depression \( (r(105) = .27; p < .05) \) and also with T2 hopelessness depression \( (r(112) = .23; p < .05) \). T1 negative life events were not significantly correlated with T2 anxiety.

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\text{Insert Table 4}
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\text{Hypothesis One}
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To test hypothesis one, two multiple regression analyses were performed. The first model predicts that negative T1 inferential style (as measured by the CSQ) and T1 hopelessness (as measured by the BHS) will predict T2 depression (as measured by the BDI-II). The second model states that negative T1 inferential style (as measured by the
CSQ) and T1 hopelessness (as measured by the BHS) will predict T2 hopelessness depression (as measured by the HDSQ).

The first model (summarized in Table 5) attempted to predict T2 depressive symptoms utilizing T1 inferential style and T1 hopelessness while accounting for initial levels of depressive symptoms. T1 depression (BDI-II) was entered in the first step to control for initial levels of depression. In the second step, inferential style was entered along with hopelessness. In step three, the interaction between T1 inferential style and T1 hopelessness was entered. The majority of the variance was accounted for by initial levels of depressive symptoms ($t(101) = 5.101; pr = .460; p < .001$). A main effect for T1 hopelessness did not predict T2 depression ($t(101) = 1.552; pr = .156; p > .10$). T1 Inferential style alone ($t(101) = .751; pr = .076; p = >.10$) failed to predict depression. The interaction between T1 inferential style and T1 hopelessness ($t(101) = -1.118; pr = -.113; p > .10$) did not predict depression.

However, significant moderate correlations were found between predictor and dependent variables utilized in this model. T1 hopelessness was significantly correlated with T2 general depression ($r(106) = .59; p < .01$). T1 inferential style was also significantly associated with general depression ($r(105) = .33; p < .01$). Significant correlations were also found for T1 hopelessness and T1 general depression ($r(126) = .62; p < .01$), T1 general depression and T2 hopelessness ($r(103) = .49; p < .01$), T2 hopelessness and T2 general depression ($r(112) = .53; p < .01$), T1 inferential style and T1 general depression ($r(125) = .44; p < .01$), T1 general depression and T2 inferential
style ($r (102) = .25; p < .05$), T2 inferential style and T2 general depression ($r (111) = .32; p < .01$).

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**Insert Table 5**

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For the next model (presented in Table 6), the procedure used in the first model was repeated except that T2 hopelessness depression (HDSQ) was utilized as the dependent variable and T1 hopelessness depression was controlled for in this analysis. The majority of the variance was accounted by initial levels of hopelessness depression symptoms ($t (102) = 7.203; pr = .588; p < .001$). A significant main effect for T1 hopelessness emerged ($t (102) = 2.294; pr = .266; p < .05$). A main effect for T1 inferential style did not predict T2 hopelessness depression ($t (102) = 1.535; pr = .153; p > .10$). The interaction for T1 inferential style and T1 hopelessness did account for a small portion of variance in predicting T2 hopelessness depression ($t (102) = -2.125; pr = -.210; p < .05$); however, this was not in the expected direction. The negative t-value and partial correlation found here indicates that a negative interaction (obtaining high scores on one predictor variable and low scores on the other predictor variable) significantly predicts hopelessness depression. This prediction occurs when scores are high on T1 hopelessness and low on T1 inferential style and when scores are low on T1 hopelessness and high on inferential style.
In addition, significant moderate correlations were also found between predictor and dependent variables utilized in this model. T1 hopelessness was significantly correlated with T2 hopelessness depression ($r (106) = .52; p < .01$). T1 inferential style was also significantly associated with hopelessness depression ($r (105) = .30; p < .01$). Significant correlations were also found for T1 hopelessness and T1 hopelessness depression ($r (127) = .56; p < .01$), T1 hopelessness depression and T2 hopelessness ($r (104) = .53; p < .01$), T2 hopelessness and T2 hopelessness depression ($r (126) = .55; p < .01$), T1 inferential style and T1 hopelessness depression ($r (126) = .44; p < .01$), T1 hopelessness depression and T2 inferential style ($r (103) = .30; p < .01$), T2 inferential style and T2 hopelessness depression ($r (111) = .30; p < .01$).

Hypothesis two:

Hypothesis two was based on the premise that Hypothesis one would be supported. As hypothesis one was not supported, the expansion of the model to test for the potential moderating effects of need to evaluate and excessive reassurance-seeking was not indicated. The need to evaluate and excessive reassurance-seeking were examined as independent predictors of depression and hopelessness depression in Hypothesis three.
Hypothesis three:

Two multiple regression analyses were used to test hypothesis three. The first regression model states that there will be significant main effects for T1 excessive reassurance seeking (as measured by the T2 DIRI-RS) and for T1 need to evaluate (as measured by the T1 NES) in predicting T2 general depression (as measured by the T2 BDI-II). The second model indicates that main effects of T1 excessive reassurance-seeking (as measured by T1 DIRI-RS) and the need to evaluate (as measured by T1 NES) will predict T2 hopelessness depression (as measured by the T2 HDSQ).

The first model (summarized in Table 7) attempted to predict T2 depression symptoms (as measured by T2 BDI-II) utilizing T1 excessive reassurance seeking and T1 need to evaluate while accounting for initial levels of depressive symptoms. T1 depression was entered in the first step to control for initial levels of depression. In the second step, excessive reassurance-seeking was entered along with need to evaluate. Initial levels of depressive symptoms accounted for the greatest amount of variance ($t (102) = 7.471; pr = .600; p < .001$). Main effects for both T1 excessive reassurance seeking ($t (102) = .019; pr = .002; p > .10$) and T1 need to evaluate ($t (102) = .648; pr = .065; p > .10$) were non-significant and did not predict time two depression.

As with hypothesis one, significant correlations were found between predictor and dependent variables utilized in this model. T1 reassurance-seeking was significantly correlated with T2 general depression ($r (106) = .37; p < .01$). Significant correlations were also found for T1 reassurance-seeking and T1 general depression ($r (126) = .48; p < .01$), T1 general depression T2 reassurance-seeking ($r (103) = .27; p < .01$), T2
reassurance-seeking and T2 general depression ($r (112) = .32; p < .01$). Need to evaluate was not significantly associated with general depression.

Insert Table 7

For the second model (presented in Table 8), the procedure used in the first model was repeated except that T2 hopelessness depression (as measured by T2 HDSQ) was utilized as the dependent variable and T1 hopelessness depression was controlled. T1 hopelessness depression was entered in the first step to control for initial levels of hopelessness depression. In the second step, T1 need to evaluate was entered along with T1 excessive reassurance-seeking. As with other models, initial levels of hopelessness depressive symptoms accounted for the greatest amount of variance ($t (103) = 9.237; pr = .679; p < .001$). Main effects for both T1 excessive reassurance-seeking ($t (103) = -1.292; pr = -.128; p > .10$) and T1 need to evaluate ($t (103) = .034; pr = .003; p > .10$) were non-significant and did not predict time two hopelessness depression.

However, significant zero-order correlations were also found between predictor and dependent variables utilized in this model. T1 reassurance-seeking was significantly correlated with T2 hopelessness depression ($r (106) = .31; p < .01$). Significant correlations were also found for T1 reassurance-seeking and T1 hopelessness depression ($r (127) = .47; p < .01$), T1 hopelessness depression and T2 reassurance-seeking ($r (104)$...
= .421; \( p < .01 \), T2 reassurance-seeking and T2 hopelessness depression \( (r (112) = .34; p < .01) \). Need to evaluate was not significantly associated with hopelessness depression.

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Insert Table 8

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**Hypothesis Four**

Previous literature suggests that cognitive vulnerabilities within the hopelessness model of depression (negative inferential style and hopelessness) are vulnerability factors that are specifically involved in the etiology of depression. Hypothesis four tests whether negative T1 cognitive (inferential) style as measured by the CSQ and T1 hopelessness as measured by the BHS will predict T2 anxiety as measured by the BAI. To test this hypothesis these variables were subjected to multiple regression analysis.

This (summarized in Table 9) attempted to predict T2 anxiety symptoms utilizing T1 inferential style and T1 hopelessness while accounting for initial levels of anxiety symptoms. T1 anxiety (as measured by the BAI) was entered in the first step to control for initial levels of anxiety. In the second step, T1 inferential style (as measured by the CSQ) was entered along with T1 hopelessness (as measured by the BHS). In step three, the interaction between T1 inferential style and T1 hopelessness was entered. The majority of the variance was accounted for by initial levels of anxiety symptoms \( (t (103) \)
A main effect for hopelessness emerged \((t = 2.720; \, pr = .566; \, p < .001)\). Inferential style alone \((t(103) = 1.224; \, pr = .122; \, p > .10)\) failed to predict T2 anxiety. The interaction between inferential style and hopelessness \((\eta(103) = -2.262; \, pr = -.222; \, p < .05)\) was a significant predictor of T2 anxiety; however, this was not in the expected direction. This negative t-value and partial correlation indicates that the combination of high scores on one predictor variable and low scores on the other variable interact to predict anxiety. This prediction occurs when scores are high on T1 hopelessness and low on T1 inferential style as well as when scores are low on T1 hopelessness and high on inferential style.

Significant moderate correlations were also found between predictor and dependent variables utilized in this model. T1 hopelessness was significantly correlated with T2 anxiety \((r(128) = .47; \, p < .01)\). T1 inferential style was also significantly associated with T2 anxiety \((r(105) = .25; \, p < .05)\). Significant correlations were also found for T1 hopelessness and T1 anxiety \((r(128) = .41; \, p < .01)\), T1 anxiety and T2 hopelessness \((r(105) = .28; \, p < .01)\), T2 hopelessness and T2 anxiety \((r(111) = .39; \, p < .01)\), T1 inferential style and T1 anxiety \((r(127) = .37; \, p < .01)\), T1 anxiety and T2 inferential style \((r(104) = .28; \, p < .01)\), T2 inferential style and T2 anxiety \((r(110) = .28; \, p < .01)\).
Hypothesis five

Hypothesis five was based on the premise that Hypothesis one would be supported. As hypothesis one was not fully supported, the expansion of the model to test for the potential effects of negative life events was not indicated.

Power

As noted above, none of the hypotheses tested with regression analyses were fully supported. Although some study variables and interactions have not yet been integrated into existing cognitive theories, such as the hopelessness model, they have received strong support in the literature on vulnerability to depression. Investigation of power is one way to explain additional information regarding this pattern of results. Although observed power for some variables utilized in regression equations was below .80, power for many of these variables included in regression analyses was quite reasonable (see Tables 5 – 9). For variables with observed power ranging from .50 to .75, an increase in sample size may have resulted in significant main effects and/or interactions within regression models. This is especially likely given significant main effects and interactions even after controlling for initial levels of dependent variables. This possibility should be tested in subsequent research using a larger sample. Other variables such as the need to evaluate and excessive reassurance-seeking, had extremely low observed power. For these variables, it is unlikely that an increase in power would have resulted in significant predictions in regression models. With this in mind, possible reasons for low power will now be discussed.
As this investigation was exploratory, adequate power estimates could not be obtained from previous literature. Initial power analyses were based on an estimated $R^2$ for the full model (the hopelessness model with the addition of need to evaluate and excessive reassurance-seeking) calculated from correlations between study predictor and dependent variables utilized in several other studies using college student samples. However, these assumptions led to the underestimation of power for study regressions. This underestimation was the result of two problems, one of which was due to the absence of published correlations between some study measures and the BDI-II and HDSQ. For example, there are no published investigations that include analyses of both need to evaluate (as measured by the NES) and depression (as measured by the BDI-II). For this measure $r^2$ was estimated utilizing a study which reported correlations between the NES and a depression related construct, negative affectivity, which was much larger than the relationship between NES and depression as well as between the NES and hopelessness depression observed in the present study.

Another problem that contributed to low power is that estimated effect sizes of variables were incorporated into power analyses without controlling for the effect of initial symptom level. For example, estimated variance accounted for by hopelessness, as well as other predictor variables, were calculated from zero-order as opposed to partial correlations. Thus, any variance accounted for by initial levels of the control variable (T1 BDI-II) was unknowingly added to the expected effect sizes of other predictor variables. This type of estimation resulted in an overall estimate of effect for the predictors that was too high and in turn, a suggested sample size that was too low.
Initial power analyses suggested that an $N$ of 184 would be sufficient to detect a small effect within regression equations. Although these participants were recruited for this study beginning two weeks before the time 1 data collection, only 129 participants took part in this collection. In addition, despite adding additional times when participants could return to complete the time 2 part of the investigation, only 112 participants completed the study. In addition, due to incomplete data for some of these participants, only between 103 and 104 participants were included in regression analyses. Low power also prohibited testing additional such as negative life events, coping style, social support, and gender within regression models. These variables will be tested within subsequent research. For a power of .80, regression analyses conducted in the present research required sample sizes ranging from 140 to 519 to detect proposed main effects and interactions. Observed power for predictors within regression equations is presented in Tables 5 – 9.
Chapter Four

Discussion

The current study attempted to replicate an existing cognitive vulnerability model (the hopelessness model; Abramson et al., 1989), which has been shown to predict the hopelessness depression subtype. A second goal of this study was to examine the roles that other factors (the need to evaluate, excessive reassurance-seeking) may play within the hopelessness model of depression, as well as, within in a model including hopelessness and inferential style (and their interaction) hypothesized to predict general depression; specifically, it was designed to test whether these variables met criteria for moderation as specified by Baron and Kenny (1986). In addition, this study tested the independent contributions of hypothesized vulnerability factors (hopelessness, the need to evaluate, inferential style, and excessive reassurance-seeking) in predicting hopelessness depression as well as depression. This study also included measurement of other factors that have been shown to be related to depressive etiology including negative life events, coping style, and social support to determine if they would have significant effects within the two models from hypothesis one.

The sample utilized in the present investigation appear to be roughly equivalent to other college student samples who have participated in studies which have supported the hopelessness model utilizing similar dependent and predictive measures on demographic variables including age, ethnicity, and level of education (e.g., Alloy & Abramson, 1999). There were significant differences between males and females on mean scores of all dependent variables, with females consistently scoring higher; however, similar gender
differences have been reported in research examining depression and anxiety in general as well as within vulnerability models (e.g., Beck & Steer, 1993; Beck, Steer, & Brown, 1996; Blalock & Joiner, 2000). Also, the time period between T1 and T2 measurement is consistent with several longitudinal investigations of cognitive vulnerabilities to depression (e.g., Joiner & Schmidt, 1998; Joiner, Alfano, & Metalsky, 1992). The representativeness of this sample provides additional support for study findings. The patterns of results in general provide both direct and indirect support for the hopelessness model and yield interesting information related to the prediction of anxiety. The remainder of this paper will discuss the implications of findings from the present study.

Study hypotheses were guided by previous research findings related to the etiology of depression. It was predicted that 1) initial levels of specific vulnerability factors included in the hopelessness model (e.g., inferential style and hopelessness) would predict levels of depression and hopelessness depression measured after six-week period, 2) the need to evaluate and excessive-reassurance seeking would account for additional variance in predicting hopelessness depression when added to the hopelessness model and would function as moderators within this model, 3) initial levels of the need to evaluate and excessive reassurance-seeking would be independently predictive of both depression and hopelessness depression measured after a six-week period, 4) initial levels of inferential style, hopelessness, the need to evaluate, and excessive-reassurance seeking would each be predictive of anxiety measured after a six-week period. In addition, several assumptions were made regarding the potential influence of negative life events, coping style, and social support. Most coping style and social support variables
did not significantly correlate with dependent measures of depression or anxiety, and for those that did, correlations were typically low. This was also true for negative life events. Although participants’ scores on these measures were not analyzed in regression models due to insufficient power, it is assumed that none would significantly decrease the observed variance accounted for by relationships specified in the aforementioned hypotheses. Hypotheses were tested using multiple regression analyses, and intercorrelations among the variables were examined.

Although none of the aforementioned hypotheses were fully supported by the data, findings suggest that hopelessness, inferential style, the need to evaluate, and excessive-reassurance seeking are related to and in some cases predictive of dependent variables. Specific findings related to each of these variables will be discussed throughout the remainder of this paper. Overall, it appears that findings from regressions conducted within this study supported the most basic assumptions of the hopelessness model: that hopelessness is the proximal, sufficient cause of hopelessness depression. This was evidenced by a significant main effect that emerged for hopelessness when predicting hopelessness depression. In addition, regression analyses provided evidence that hopelessness was independently and in an interaction with inferential style, predictive of anxiety. This speaks to the specificity of the model and will be discussed later. Despite findings that indicated only partial support for regression equations, most of the variables in the models that were not supported by regression analyses were found to be significantly correlated. As reported in the results section, significant correlations between predictor and dependent variables were found for relationships between T1 and
T2 as well as within each time period (e.g., T1 and T1 or T2 and T2). For example, there were significant correlations between hopelessness and each of the dependent variables (hopelessness depression, general depression and anxiety), between inferential style and general as well as hopelessness depression, and between excessive reassurance-seeking and general and hopelessness depression as well as anxiety. Only the need to evaluate failed to predict or to be associated with any dependent variables. The presence and magnitude of these correlations appear to support the hopelessness model and will be discussed below.

Hopelessness and inferential style as vulnerability factors

Results of this study failed fully to support hypothesis one, which stated that main effects of hopelessness and inferential style as well as the interaction of these variables would predict depression and hopelessness depression. As a great deal of literature has supported the hopelessness model (see Abramson, Alloy, Hogan, et al., 1999 for a review), this finding is highly unexpected. According to hopelessness theory, attributional style and hopelessness are theorized to be involved in the etiology of depression and both of these variables are necessary components of the model and therefore must each be present if hopelessness depression is to develop. Although some findings from regression equations tested in hypothesis one fail to reject the null hypotheses, partial support from regressions and correlational analyses produce a pattern of findings, which partially support the hopelessness model of depression.
Hopelessness

Significant main effects and correlations for hopelessness provided a great deal of support for the hopelessness model. As previously noted, a significant main effect for T1 hopelessness accounted for a small amount of variance in predicting T2 hopelessness depression. This finding provides support for the hopelessness model even though the variance accounted for by the main effect for hopelessness is small in comparison the magnitude of prediction resulting from of initial levels of hopelessness depression. Specifically, theory states that hopelessness is considered to be a sufficient proximal cause of hopelessness depression and thus should be highly predictive of hopelessness depressive symptoms; however, in Abramson and colleagues' (1989) model, hopelessness develops when an existing negative attributional style interacts with significantly stressful or negative life events. Although inferential style did not produce a significant main effect on hopelessness depression and negative life events were not included in regression equations, hopelessness still predicted hopelessness depression. This highlights the importance of hopelessness within this model.

Additional support for the hopelessness model can be derived from the finding that the interaction between hopelessness and inferential style is significantly predictive of hopelessness depression over and above initial levels of hopelessness. As previously noted, this interaction produced a negative $\beta$ and $t$ values and is interpreted to mean that hopelessness depression is predicted by the combination of high scores on one measure with low scores on the other. As the combination both factors in this interaction influence the ability to predict hopelessness depression, these results partially replicate
the relationship between these factors from Abramson and colleagues' (1989) hopelessness model. For example, in the hopelessness model negative attributions are activated in individuals when these individuals are exposed to negative life events, and this process is theorized to lead to hopelessness, a proximal sufficient cause of the hopelessness subtype of depression. Within the hopelessness model, however, it is high levels of both negative attributions and hopelessness that are believed to predict hopelessness depression. Thus, the negative interaction between hopelessness and inferential style is unexpected. However, the present investigation tested only the diathesis components of the hopelessness model. As the presence of stressors (negative life events) are considered to be important in the path from negative inferential style to hopelessness, it is possible that attributional style and hopelessness are somehow differently related when measured in the absence of these events. Although some research has failed to find an interaction between cognitive vulnerability and negative life stressors (e.g., Barnett & Gotlib, 1988; Persons & Rao, 1985; Robins & Block, 1989; Robins, Block, & Peselow, 1990), more recent research indicates that life stressors do interact with cognitive vulnerabilities to predict general and hopelessness depression (e.g., Alloy et al., 1999; Joiner, Metalsky, Lew, and Klocek, 1999). Negative life events were not included in regression equations due to reduced power. Thus, it is plausible that the addition of the LES to the interaction of hopelessness and inferential style could have increased the ability of this model to predict hopelessness depression. This is likely in light of significant correlations between initial levels of negative life events and initial levels of inferential style, between initial levels of negative life events and hopelessness,
and between initial levels of negative life events and T2 levels of hopelessness 
depression. The negative interaction found in this model between hopelessness and 
inferential style and the potential contribution of negative life events can be examined 
using ANOVA procedures and will be the focus of future research.

In addition to replicating aspects of the hopelessness model, the findings of 
significant main effects for hopelessness and for the interaction between hopelessness 
and inferential style are important because they provide support for the idea that 
cognitive vulnerabilities are predictive of depression even when controlling for initial 
levels of depression. As previously stated, this idea has been challenged by Persons and 
Miranda’s mood state hypothesis which indicates that cognitive vulnerability factors, 
such as negative attributions, are not stable and lead to depressive symptoms only when 
activated by a negative mood state such as depression that is specifically induced by 
mood induction procedures (e.g., imagined stressful life events). The finding that 
negative attributions and hopelessness predicted hopelessness depression regardless of 
initial levels of hopelessness depression suggests that activation of negative attributions 
by inducing a negative mood state is not necessary for cognitive vulnerability factors to 
predict or possibly to cause hopelessness depression. This supports the idea that 
individuals with negative inferential style and hopelessness are cognitively vulnerable 
every day and do not require activation (e.g., by a negative mood induction) to develop 
depression. In addition, since T1 hopelessness and the T1 interaction between 
hopelessness and inferential style significantly predicted T2 hopelessness depression,
without entering negative life events into the regression equation, it is plausible that
negative life events or stressors may not be essential for the development of hopelessness depression. These possibilities highlight the stability and importance of cognitive diatheses (hopelessness and inferential style) in the hopelessness model.

Perhaps the most substantial support for the hopelessness model derived from this study is that hopelessness alone and in an interaction with inferential style failed to predict general (as opposed to the hopelessness type of) depression (T2 BDI-II scores). This finding, although unexpected, is extremely important when considering hopelessness theory. For example, while factors such as inferential style and hopelessness are similar to the negative schemas and pessimism from Beck’s (1967, 1976) theory, which predicts general depression, factors within the hopelessness theory are hypothesized to lead specifically to the hopelessness subtype of depression. These findings from regression analyses were further supported by patterns of zero-order correlations among inferential style, hopelessness, and both general and hopelessness depression. For example, the magnitude of correlation between T1 inferential style and T2 hopelessness depression and between T1 hopelessness and T2 hopelessness depression were greater than the magnitude between T1 inferential style and T2 general depression and between T1 hopelessness and T2 general depression. It should be emphasized that significant correlations between cognitive vulnerabilities from the hopelessness model with general depression may exist because of some overlap between symptoms of general and hopelessness depression. In this study, findings of significant moderate correlations between vulnerabilities and general depression in the absence of
significant main effects or interactions provides support for the idea of overlapping but different (heterogeneous) types of depression.

*Inferential Style*

As inferential style is a component of the hopelessness model of depression, findings related to its interactions with hopelessness in regression equations are presented in the hopelessness section above. However, findings related to the absence of significant main effects for inferential style in predicting both hopelessness depression and general depression are discussed in this section. Although these findings were not expected, they were actually consistent with the hypothesized role of inferential style within the hopelessness model. Specifically, inferential style is proposed to be necessary but not sufficient in the etiology of the hopelessness subtype of depression. When examined in this context, the failure of inferential style to predict hopelessness depression does not diverge from hopelessness theory, especially given the significance of the hopelessness X inferential style interaction.

More support for the role of inferential style within the hopelessness model is demonstrated by significant, moderate, positive correlations between inferential style and hopelessness and also between inferential style and hopelessness depression. These correlations are important for two reasons. First, inferential style is considered necessary for the development of hopelessness within the hopelessness model. Although correlations do not provide information related to directionality or causality, significant associations between these variables both within and between time periods of the study suggests that both factors are related and relatively stable within study participants. In
conjunction with significant correlations between both hopelessness and inferential style and depression, these findings appear to increase support for the hopelessness model provided by regression analyses.

Finally, a main effect for inferential style failed to predict general depression. This finding failed to reject the null hypothesis; however as with hopelessness, the failure of inferential style to predict general depression supports the specificity of the hopelessness model (e.g., predicting only the subtype of depression). Although unexpected, this is consistent with hopelessness theory, which states that inferential style is a vulnerability factor specifically predictive of hopelessness depression.

At this point, the discussion has focused on evidence obtained from study data that has demonstrated partial support for the hopelessness model of depression. It appears that patterns of findings are consistent with hopelessness theory of depression and also provide support for heterogeneity of depression and for the specificity of etiological factors. Implications of this evidence are important when considering the development of future research and clinical practice and these will be discussed later in this section. Emphasis of this discussion will now turn to findings related to the roles of need to evaluate and excessive-reassurance seeking in predicting depression and hopelessness depression.

**Excessive Reassurance-Seeking and the Need to Evaluate as Vulnerability Factors**

As noted in the results section, excessive reassurance-seeking and the need to evaluate were not tested for moderation because models from hypothesis one were not fully supported by the data; however, both variables were tested within regression models.
to determine if they predicted depression or hopelessness depression. Overall, results from regression equations indicate that both factors failed to predict dependent variables of hopelessness depression and general depression.

_Excessive Reassurance-Seeking_

Main effects of reassurance-seeking failed to predict general depression. This result is unexpected, especially given the substantial body of literature which has demonstrated that excessive-reassurance seeking is involved in the etiology of depression (e.g., Joiner, 1999). This is not to say that reassurance-seeking and general depression were found to be completely unrelated. Significant, moderate zero-order correlations were found between reassurance-seeking and general depression both within and between time periods. This finding provides some support for the relationship between reassurance-seeking and depression. In addition, as previously stated some of the work in the area of excessive-reassurance seeking suggests that the relationship between this factor and general depression is moderated by causal uncertainty, the inability of an individual to understand cause and effect relations in the social world (Weary & Edwards, 1996; Weary et al., 1993; Jacobson and Weary, 1999); however, this possibility was not tested in the present investigation. Thus, it is plausible that reassurance-seeking failed to predict depression because uncertain attributional style does strengthen the relationship between these factors, but was not measured and/or incorporated into the regression equation. This possibility should be examined in future research.

It is important to note that reassurance-seeking has never been tested within the hopelessness model or with hopelessness depression. Results also indicated that
excessive reassurance-seeking failed to predict hopelessness depression. As with general
depression, excessive reassurance-seeking correlated with hopelessness depression both
within and between data collections. Thus, it appears that there is some relationship
between these variables. As the magnitude of correlations between excessive
reassurance-seeking and general as well as hopelessness depression are similar, it is
possible that reassurance-seeking is related to several types of depression and highlights
the importance of this construct as a risk factor for depression. This implication is
important for the development of treatments for depression that excessive and, as this
study is exploratory in nature, should be addressed in future research. Research and
clinical applications of this finding will be discussed below.

Need to Evaluate

Main effects for need to evaluate failed to predict general depression as well as
hopelessness depression significantly. This finding is unexpected, especially as
evaluation of one’s external environment is an implicit assumption of attributional style
models of depression and also because evaluation seems intuitively linked with
attributional (inferential) style. As previously noted, tendencies to engage in external
evaluation could provide more opportunities to develop negative attributions, while not
engaging in external evaluation decreases opportunities for new information to
disconfirm existing negative patterns of thinking. Also, it is possible that external
evaluation decreases with an increasing reliance on negative attributions to guide
processing of new information. In all of these cases, however, either high or low levels
of external evaluation would be associated with attributions, and thus would also likely
be associated with depression. The lack of a relationship between need to evaluate and general as well as hopelessness depression was also reflected by non-significant, low magnitude zero-order correlations between these variables. These results may suggest that need to evaluate is completely unrelated to depression and the hopelessness subtype of depression; however, it is also possible that these constructs were unrelated because participants were not consistently evaluating their external environment. As previously noted, mid-levels of need to evaluate indicate that individuals are not engaging in external evaluation in a stable way, which suggests that it should not be able to function as a stable predictor of another construct. This possibility would explain the lack of predictive and associative relationships between need to evaluate and depression measures. Results from this investigation yield mean scores on need to evaluate that do indeed fall within the mid-level range. Thus, the findings may not truly fail to support the hypothesized relationship between need to evaluate and general and hopelessness depression. This possibility should be tested in future research. One other possibility that remains to be tested is whether a curvilinear relationship exists between need to evaluate and depression. This type of relationship was not tested in these analyses but may reveal significant, albeit non-linear relationships, among these constructs. Although it is unlikely that a curvilinear relationship existed between need to evaluate and dependent variables given the restricted range of T1 NES scores in this sample, it is possible that a non-linear relationship would emerge if a sample with more variability (e.g., more high and low scores on the NES) was tested. At this time, the exact
association between the need to evaluate and depression remains unclear and research examining predictive or associative relationships is needed.

In summary, this examination of the need to evaluate and excessive reassurance-seeking represents an initial attempt to explore possible associations with the hopelessness subtype of depression and provides indirect support for literature which has demonstrated predictive relationships between reassurance-seeking and general depression. Findings of significant associations between excessive reassurance-seeking and general and hopelessness depression provides support for examining these variables in more complex cognitive models in future research studies. This reflects one of the original goals of this study that could not be carried out due to insufficient sample size and power (this will be discussed in the limitations section below). In addition, possible reasons for findings of non-significant associative relationships between the need to evaluate and general and hopelessness depression provide questions for future study. So far, only results, which test relationships between predictor variables and general depression and hopelessness depression, have been discussed. The next section discusses the specificity of study findings to depression and explains findings related to anxiety.

Specificity

Hypothesis four was included in this study to determine whether or not cognitive vulnerability factors included in the hopelessness model of depression (hopelessness and inferential style) would also be predictive of anxiety. Analyses indicate that main effects for hopelessness but not inferential style significantly predicted anxiety. In addition, the interaction between hopelessness and inferential style was predictive of anxiety but this
was not in the expected direction. As previously noted, this finding suggests that the combination of low levels of inferential style with high levels of hopelessness (and vice-versa) predicted anxiety. This finding is unexpected given numerous studies which suggest that etiological factors for depression and anxiety are unique (e.g., Alloy & Clements, 1998). It should be noted that initial level of anxiety was the best predictor of anxiety at time 2.

The finding that the main effect for inferential style did not predict anxiety is in line with specificity hypothesized in hopelessness theory. However, the finding of a negative interaction between inferential style and hopelessness which is predictive of anxiety is unusual because there is a great deal of literature which states that the effects of inferential style and hopelessness (along with their interaction) is specific to the hopelessness subtype of depression (Alloy & Clements, 1998; Alloy, Abramson, Whitehouse, & Hogan, 2000). As there is strong support for the specificity of the hopelessness model to hopelessness depression, the finding that hopelessness and inferential style predict anxiety may be explained by limitations this investigation. It is possible that other factors or confounds may have contributed to the prediction of depression within this regression equation. As this study is correlational in nature, it is impossible to rule out this possibility. Another explanation for this finding is that hopelessness does predict anxiety. It is possible that the interaction between inferential style and hopelessness are specific to depression but that like depression, etiological factors that lead to hopelessness are heterogeneous. Thus, it may be that hopelessness is
predictive of anxiety when it develops as a result of factors other than negative patterns of thinking such as inferential style.

Although the finding that hopelessness predicts anxiety is inconsistent with previous literature in the area of depressive vulnerability, it does not necessarily contradict hopelessness theory. It is important to note that hopelessness was predictive of hopelessness depression as well as anxiety. These results suggest that hopelessness may serve as a risk factor for mood as well as anxiety symptoms and possibly, disorders. The extent of the relationship between hopelessness and depression and anxiety is unclear at this time and should be the focus of future research; however, it does appear to be a potential risk factor and thus should be incorporated into existing treatments for depression and anxiety. These implications will be discussed later in this discussion.

Other findings such as correlations between measures of depression and anxiety both within and between time periods and similar patterns of mean scores for depression, hopelessness depression, and anxiety (a substantial decrease from the first to the second data collection) fit with other literature related to high levels of comorbidity between depression and anxiety (e.g., Brown & Barlow, 1992). Ingram and colleagues (1998) postulate that comorbid anxiety and depression may be prevalent because factors which activate cognitive vulnerabilities to depression may also activate cognitive vulnerabilities to anxiety (e.g., a disruption in the attachment process). Their explanation relates to factors, which may represent extremely distal contributory causes (e.g., activating agents) not currently included in cognitive models of depression. The some common factors may play a role in the activation, or perhaps etiology of an array of cognitive vulnerability
factors (some which are unique to depressive vulnerability and some which are unique to anxiety) provides a parsimonious explanation for the comorbidity of these distinct disorders.

**Limitations of the Present Study**

Results of this investigation partially supported existing models of cognitive vulnerability to depression and provided new information about the prediction of anxiety. Most of these results were expected; however, as previously noted hypotheses were not fully supported and some variables and models were not tested as a result (e.g., whether the need to evaluate and excessive reassurance-seeking moderate the relationship between cognitive vulnerabilities and depression). There are limitations related to sample size/power and also to correlational studies in general that may have affected study findings.

As previously noted, a small sample size decreases resulting power for study analyses, which in turn may increase the likelihood of type II errors. It is possible that variables found to be related (e.g., those that demonstrated significant zero-order correlations) may have been found to account for variance within regression models if the sample was larger. As previously noted, increasing the sample size may result in significance for some predictions but not others (e.g., for main effects of variables with observed power above .50 but below .80); however, predictor variables with low power (e.g., power below .50) may not be related to dependent variables, or may be related in to dependent variables in non-linear models or using samples with more variable range of scores on some study measures (e.g., the NES). Although reduced power may have
caused some type II errors and prohibited testing of additional such as negative life events, coping style, social support, and gender within the hypothesized models, it does not appear to be the paramount reason for the lack of significant findings for the main effects of need to evaluate or excessive reassurance-seeking. These variables will be tested within subsequent research.

In addition to small sample size and reduced power, the correlational nature of this study does not control for potential threats to internal validity such as statistical regression, maturation, or history. All of these threats are suspect within the present study because of the large decrease in mean scores from time 1 to time 2, common to all dependent variables (significant differences between mean scores for T1 and T2 general depression, T1 and T2 hopelessness depression, and T1 and T2 anxiety). Specifically, it is possible that participants became less depressed and anxious from the beginning to the end of the study because they were impacted by factors unrelated to study variables. For example, it is possible that mean scores for depression and anxiety measured decreased because the initial phase of the study occurred during the week of mid-term examinations while the second phase was one to two weeks before finals when participants were experiencing less stress. In addition, it is also possible that participants who were highly depressed and/or anxious to begin with became less so as a result of the natural course of their symptoms or disorders or simply as a result of regression to the mean. Unfortunately, due to the nature of this study, the impact of these factors cannot be determined. However, it was determined that there were no differences in T1 mean scores between completers and non-completers of the study on depression, hopelessness
depression, or anxiety, and thus, it is unlikely that confounds related to attrition (e.g. that the most depressed and anxious participants did not return to complete the study and thus statistically decreased sample means from T1 to T2) influenced findings.

**Future Directions**

Research

Study findings and limitations suggest some future directions for research. It would be helpful to replicate this study in the future with a larger sample, perhaps utilizing several overlapping data collections to assist with the identification of potential confounds such as history or attrition. This type of investigation would give more certainty to results related to the prediction of depression, hopelessness depression, and anxiety and would decrease the potential for type II errors. Increasing the study sample would also make it possible to include potentially related variables such as coping style and social support in regression equations. This may yield valuable information, especially as many of these variables were significantly correlated with measures of depression and anxiety in the present study. It would also be interesting to determine the role of study variables that were not tested in regression equations such as negative life events, social support, and coping style using analysis of variance procedures. For example, participants could be into split into groups based on high or low scores on predictor variables that were tested in regressions (e.g., initial symptoms, hopelessness, and inferential style) and then untested variables such as negative life events could be covaried. This type of testing could be conducted in an exploratory way to guide future research hypotheses.
Additional hypotheses generated from study findings could also be tested in subsequent research. For example, it would be interesting to investigate variables that may predict hopelessness when anxiety develops in individuals across time. Further, as the need to evaluate failed to predict any of the dependent variables within this investigation, it would be beneficial to examine whether a curvilinear relationship exists between need to evaluate and measures of depression and anxiety. Also, significant correlations between excessive reassurance-seeking and both general and hopelessness depression suggest future research aimed at examining the utility of this variable within existing etiological theories, perhaps using path models or structural equation modeling approaches.

Clinical Applications

As previously noted, findings related the ability of hopelessness to predict both hopelessness depression and anxiety suggest that this construct should be incorporated within existing cognitive treatments for these disorders. In addition, if hopelessness is shown to be a consistent predictor of anxiety in future research, it would be interesting to utilize screening measures which include hopelessness to identify at risk individuals and hopefully prevent the development of anxious or depressive symptoms of disorders. This type of preventive screening has been suggested in previous literature and is supported by outcome data.

As stated by Ingram and colleagues (1998), it is probable that efforts towards prevention could be helpful to prevent development of depression in individuals who have never been depressed, as well as to prevent relapse of depressive episodes in non-
depressed individuals who have a history of depression. These authors also integrate vulnerability literature from both cognitive and biological theory that suggests that processes involved in the etiology of depression become increasingly incorporated into patterns of thinking and into neurological pathways with each additional depressive episode. This idea is similar to Lewinsohn and colleagues' (1981) scar hypothesis, which suggests that processes involved in a depressive episode may create lasting changes in cognitions thus increasing vulnerability to subsequent depressive episodes. The likelihood that future depression is influenced by past depressive episodes is consistent with study findings that indicate that initial levels of depression were the best predictors of subsequent levels of depression and that initial levels of hopelessness depression were the best predictors of hopelessness depression at the second time period. Although cognitive and biological processes involved in depressive onset may be different (e.g., more complex in relapse than at initial onset), prevention would be useful in both cases.

As previously noted, treatments related to preventing depressive onset in college students and other at risk populations have been designed and have received some support in the literature (e.g., DeRubeis et al., 1998; Munoz, 1993, 2000; Munoz et al., 1996). Although there is a great deal of evidence for the effectiveness of existing empirically validated treatments for depression with currently depressed populations (with the exception of booster sessions commonly used in CBT), there appears to be less work validating efforts toward prevention of depression in recovered individuals (e.g., those who have experienced at least one depressive episode but are currently non-depressed).
As discussed by Ingram and colleagues (1998), techniques utilizing attentional distraction to prevent a focus on depression related negative cognitions (e.g., Teasdale, Segal, & Williams, 1995). This attentional distraction is similar to ideas proposed within the current study, such as that a focus on external environmental events may prevent the over-reliance on negative attributions to guide information processing in individuals with high levels of need to evaluate. Although this idea was not supported by study results, attentional distraction could be applied to persistent thought patterns that maintain hopelessness once it has developed. Distracting individuals from demoralizing ruminations and potentially negative affect associated with hopelessness could possibly prevent depressive onset or relapse. Thus, the finding that hopelessness an important aspect in the prediction of depression could guide the application of distraction techniques specifically aimed at hopelessness, especially within existing therapies for depression, such as CBT.

Given findings which indicate that hopelessness predicts anxiety as well as hopelessness depression, it is likely that efforts to address hopelessness in existing validated treatments for anxiety (e.g., Butler, Fennell, Robson, & Gelder, 1991) may improve the overall quality of care for individuals experiencing anxiety. Although the sample from this investigation utilized a college student sample and may not generalize to clinical populations, findings related to the predictive relationship between hopelessness and anxiety suggest that targeting hopelessness in preventive interventions (e.g., with vulnerable but non-disordered individuals) may help to decrease rates of anxiety in general. Screening procedures, such as those utilized in depressed and primary
care patients (Munoz, Le, and Ippen, 2000) could be adapted to screen for anxiety itself as well as for risk factors related to anxiety. The high comorbidity of anxiety and depression may suggest that screening should be done simultaneously for both of these conditions.

It is probable that adaptation of screening procedures might also be important to address different types of depression. For example, the addition of hopelessness as well as symptoms of hopelessness depression to depression screening checklists would be meaningful in that these additions might tap important areas missed by measures of endogenous depression. This idea is supported by findings from the present study (e.g., that hopelessness predicted hopelessness depression but not general depression) as well as by literature that suggests that the presence of suicidal symptoms and hopelessness should be addressed when reported in depression screenings (Jacobs, 1999). Adjusting brief screening inventories to include items regarding hopelessness would thus increase the number of individuals who could benefit from subsequent preventive interventions.

In conclusion, hopelessness and to some extent, the interaction between hopelessness and inferential style appear to be predictive of the hopelessness subtype of depression as well as anxiety. As noted in the discussion above, the hopelessness model was partially supported by findings from regression equations and zero-order correlations. Due to limitations of this investigation, it was not determined whether other factors such as social support, coping style, or negative life-events would have predicted depression, hopelessness depression, or anxiety, independently or in interactions with other variables. An increase in sample size and additional power may have provided a
clearer picture of the influence of variables included in regression analyses (e.g., hopelessness, inferential style, need to evaluate, and excessive reassurance-seeking). Inclusion of a larger sample would also allow for a test of the proposed moderation hypothesis. Findings related to the predictive power of hopelessness as well as initial symptom levels of depression, hopelessness depression, and anxiety speak to the importance of providing the best possible preventive interventions and treatments. Future research should be aimed at replication and extension of study findings as well as at the development of screening measures that are sensitive to the presence of hopelessness and of hopelessness depression.
Reference


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### Table 1

**Means and Standard Deviations For Study Measures, Excluding Demographic Variables.**

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<td>32</td>
<td>2.33</td>
<td>1.37</td>
<td>2.33</td>
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</table>

All variable names followed by 1 and 2 indicate scores from T1 and T2 respectively; SUMBAI = Beck Anxiety Inventory; SUMBDI = Beck Depression Inventory 2nd edition; SUMNES = Need to Evaluate Scale; SUMBH SR = Beck Hopelessness Scale; COPEFV = the focus on venting of emotions sub-scale of the COPE; COPEIS = the use of instrumental social support sub-scale of the COPE; COPEAC = the active coping sub-scale of the COPE; COPEBD = the behavioral disengagement sub-scale of the COPE; COPEES = the use of emotional social support sub-scale of the COPE; COPEPL = the Planning sub-scale of the COPE; SUMHDSQ = the Hopelessness Depression Symptom Questionnaire; PASSQ = the perceived sub-scale of the Social Support Questionnaire - 6th edition; OSSSQ = the actual sub-scale of the Social Support Questionnaire - 6th edition; SUMLESR = the Hammen Perceived Life Events Scale; INFSTYL = Cognitive Styles Questionnaire; MNDIRI = Reassurance-Seeking sub-scale of the Depressive Interpersonal Relationships Inventory.
Table 2

**Correlations with Gender and Mean Scores and Group Differences for Males and Females on Dependent Measures at Time 1 and Time 2**

<table>
<thead>
<tr>
<th>Variable</th>
<th>$r$</th>
<th>Group Mean for Males</th>
<th>Group Mean for Females</th>
<th>$t$</th>
<th>df</th>
</tr>
</thead>
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<tr>
<td>BDI-II (T1)</td>
<td>.258**</td>
<td>9.77</td>
<td>15.33</td>
<td>-2.891**</td>
<td>124</td>
</tr>
<tr>
<td>BDI-II (T2)</td>
<td>.307**</td>
<td>5.38</td>
<td>11.24</td>
<td>-3.047**</td>
<td>104</td>
</tr>
<tr>
<td>HDSQ (T1)</td>
<td>.203*</td>
<td>16.24</td>
<td>21.11</td>
<td>-2.289*</td>
<td>125</td>
</tr>
<tr>
<td>HDSQ (T2)</td>
<td>.336**</td>
<td>9.05</td>
<td>17.13</td>
<td>-3.443***</td>
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<td>BAI (T1)</td>
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<td>8.73</td>
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</table>

Note. BDI-II = the Beck Depression Inventory, 2nd edition; HDSQ = the Hopelessness Depression Symptom Questionnaire; BAI = the Beck Anxiety Inventory. For this analysis, males were coded “0” and females were coded “1”.

* $p < .05$. ** $p < .01$. *** $p < .001$. 

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Table 3

Summary of Mean Scores From T1 and T2 Dependent Variables From This Sample Compared With Normative Sample Means

<table>
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<tr>
<th>Sample</th>
<th>BDI-II</th>
<th>HDSQ</th>
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<tr>
<td>T1</td>
<td>12.11</td>
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<td>8.36</td>
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<tr>
<td>2.</td>
<td>12.56 (120)</td>
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<tr>
<td>3.</td>
<td>11.38 (435)</td>
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<td></td>
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<tr>
<td>4.</td>
<td></td>
<td>11.80 (326)</td>
<td></td>
</tr>
</tbody>
</table>

Note. 1 = sample from the present study; 2 = sample from Beck et al. (1996); 3 = sample from Metalsky and Joiner (1997); 4 = sample from Creamer, Foran, and Bell (1994). Numbers in parentheses are N’s for sample means. All samples were undergraduate college students from the United States, Australia, and/or Canada.
Table 4

Zero-Order Correlations Among Study Variables

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<th>Variables</th>
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### Table 4 continued

**Zero-Order Correlations Among Study Variables**

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Table 4 continued

Zero-Order Correlations Among Study Variables

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Zero-Order Correlations Among Study Variables

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<td>.233*</td>
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Table 4 continued

Zero-Order Correlations Among Study Variables

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<td>.316**</td>
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<td>32. MNDIRI2</td>
<td>.132</td>
<td>.201*</td>
<td>.590**</td>
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Note. All variable names followed by 1 and 2 indicate scores from T1 and T2 respectively; SUMBAI = Beck Anxiety Inventory; SUMBDI = Beck Depression Inventory 2nd edition; SUMNES = Need to Evaluate Scale; SUMBHRSR = Beck Hopelessness Scale; COPEFV = the focus on venting of emotions sub-scale of the COPE; COPEIS = the use of instrumental social support sub-scale of the COPE; COPEAC = the active coping sub-scale of the COPE; COPEBD = the behavioral disengagement sub-scale of the COPE; COPEES = the use of emotional social support sub-scale of the COPE; COPEPL = the Planning sub-scale of the COPE; SUMHDSQ = the Hopelessness Depression Symptom Questionnaire; PASSQ = the perceived sub-scale of the Social Support Questionnaire - 6th edition; OSSSQ = the actual sub-scale of the Social Support Questionnaire - 6th edition; SUMLESR = the Hammen Perceived Life Events Scale;
Table 5

Summary of Multiple Regression Analysis for Variables Predicting T2 Depressive Symptoms (as measured by T2 BDI-II) (N = 102)

<table>
<thead>
<tr>
<th>Predictor Variables Entered</th>
<th>β</th>
<th>Cumulative Change in R²</th>
<th>Change in R²</th>
<th>F Change in R²</th>
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<td>.453</td>
<td>82.882***</td>
<td>.673***</td>
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<tr>
<td>T1 BHS</td>
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<td>.021</td>
<td>2.003</td>
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<td>T1 CSQ</td>
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<td>T1 BHS X T1 CSQ</td>
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<td>.460</td>
<td>.007</td>
<td>1.250</td>
<td>-.113</td>
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Note. BDI-II = the Beck Depression Inventory, 2nd edition; BHS = the Beck Hopelessness Scale; CSQ = Cognitive Style Questionnaire. Observed power for T1 BDI-II = .999, T1 BHS = .366, T1 CSQ = .115, T1 BHS X T1 CSQ = .198

*p < .05. **p < .01. ***p < .001.
Table 6

Summary of Multiple Regression Analysis for Variables Predicting T2 Hopelessness Depressive Symptoms (as measured by T2 HDSQ) (N = 103)

<table>
<thead>
<tr>
<th>Predictor Variables Entered</th>
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<th>Change in $R^2$</th>
<th>$F$ Change in $R^2$</th>
<th>Partial Correlation</th>
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<td>T1 HDSQ</td>
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<td>.505</td>
<td>103.191***</td>
<td>.588***</td>
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<td>.005</td>
<td>.530</td>
<td>.226*</td>
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<td>T1 CSQ</td>
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<td>.153</td>
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<td>Step 3</td>
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</tr>
<tr>
<td>T1 BHS X T1 CSQ</td>
<td>-.958</td>
<td>.513</td>
<td>.022</td>
<td>4.515*</td>
<td>-.210*</td>
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</table>

Note. HDSQ = the Hopelessness Depression Symptom Questionnaire; BHS = the Beck Hopelessness Scale; CSQ = Cognitive Style Questionnaire. Observed power for T1 HDSQ = 1.000, T1 BHS = .622, T1 CSQ = .330, T1 BHS X T1 CSQ = .557

* p < .05. ** p < .01. ***p < .001.
Table 7

Summary of Multiple Regression Analysis for Variables Predicting T2 Depressive Symptoms (as measured by T2 BDI-II) \( (N = 103) \)

<table>
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<th>( F ) Change in ( R^2 )</th>
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<td>T1 NES</td>
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<td></td>
<td>.065</td>
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<tr>
<td>T1 DIRI</td>
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Note. BDI-II = the Beck Depression Inventory, 2nd edition; NES = Need to Evaluate Scale; DIRI = the Excessive Reassurance Seeking sub-scale of the DIRI. Observed power for T1 BDI-II = 1.000, T1 NES = .098, T1 DIRI-RS = .050

\* \( p < .05 \). ** \( p < .01 \). *** \( p < .001 \).
### Table 8

**Summary of Multiple Regression Analysis for Variables Predicting T2 Hopelessness Depressive Symptoms (as measured by T2 HDSQ) (N = 104)**

<table>
<thead>
<tr>
<th>Predictor Variables Entered</th>
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<th>Cumulative $R^2$</th>
<th>Change in $R^2$</th>
<th>$F$ Change in $R^2$</th>
<th>Partial Correlation</th>
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<td>Step 1</td>
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<td>.505</td>
<td>104.263***</td>
<td>.679***</td>
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<td>Step 2</td>
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</tr>
<tr>
<td>T1 NES</td>
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<td>.499</td>
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<td>.003</td>
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<td>T1 DIRI</td>
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<td>-.128</td>
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</table>

Note. HDSQ = Hopelessness Depression Symptom Questionnaire; NES = Need to Evaluate Scale; DIRI = the Excessive Reassurance Seeking sub-scale of the DIRI. Observed power for T1 HDSQ = 1.000, T1 NES = .050, T1 DIRI-RS = .249

* $p < .05$. ** $p < .01$. *** $p < .001$. 

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Table 9

Summary of Multiple Regression Analysis for Variables Predicting T2 Anxiety Symptoms (as measured by T2 BAI) (N = 104)

<table>
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<th>Change in R²</th>
<th>F Change in R²</th>
<th>Partial Correlation</th>
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<td>.566***</td>
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<tr>
<td>Step 2</td>
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<td>.468</td>
<td>.031</td>
<td>2.976</td>
<td>.264**</td>
</tr>
<tr>
<td>T1 BHS</td>
<td>1.145</td>
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<tr>
<td>T1 CSQ</td>
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<td>.122</td>
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<tr>
<td>Step 3</td>
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<td>.489</td>
<td>.025</td>
<td>5.119*</td>
<td>-.222*</td>
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<td>T1 BHS X T1 CSQ</td>
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</tbody>
</table>

Note. BAI = Beck Anxiety Inventory; BHS = the Beck Hopelessness Scale; CSQ = Cognitive Style Questionnaire. Observed power for T1 BAI = 1.000, T1 BHS = .768, T1 CSQ = .228, T1 BHS X T1 CSQ = .610

*p < .05. **p < .01. ***p < .001.
Figure 1

Moderator Model

Predictor → A → Outcome

Moderator → B → Variable

Predictor X Moderator → C

(Baron & Kenny, 1986)
Figure 2

Mediator Model

(Baron & Kenny, 1986)
APPENDIX A

SUBJECT INFORMATION AND CONSENT FORM

**TITLE:** Predictors of Depressive Symptomatology: Cognitive Theories of Vulnerability and the Relationship of Interactional and Cognitive Styles

**Principle Investigator:** Erica L. Shertzer, B.A.
Department of Psychology
Skaggs Building 368
(406) 243-5647

**Supervisor:** John W. Klocek, Ph.D.
Department of Psychology
Clinical Psychology Center 131
(406) 243-5647

**Special instructions to the potential subject**
This consent form may contain words that are new to you. If you read any words that are not clear to you, please ask the person who gave you this form to explain them to you.

**Purpose**
You are being asked to take part in a research study examining a number of areas that have been identified in previous research as potentially important factors in predicting who becomes depressed. The purpose of this research is to determine the role that patterns of thinking and behavior may play in the development of depression.

**Procedures**
If you agree to take part in this research study you will be given a questionnaire packet that asks you about some experiences you may have had, some thoughts and feelings you may have experienced, ways you may act in various situations, and about whom you rely upon for support. You will be asked to complete each questionnaire and to record your answers on the sheets provided. You will also be required to return in approximately six weeks and fill out these questionnaires again. The study will take place at the University of Montana and each session will last for approximately two hours.

**Risks/Discomforts**
The questionnaires ask you about some experiences you may have had, some thoughts and feelings you may have experienced, ways you may act in various situations, and about whom you rely for support. It is possible that some of the questions may elicit uncomfortable feelings. Should this be the case please contact the principle investigator, Erica L. Shertzer (243-5647) or the Counseling and Psychological Services Center (243-4711). Should you have any other questions about the study, please feel free to contact Erica Shertzer at 243-5647.
Benefits

You may benefit from this study during the debriefing by learning more about the research concerning vulnerability to depression.

Confidentiality

Your records will be kept private and will not be released without your consent except as required by law*. Your identity will be kept confidential. If the results of this study are written in a scientific journal or presented at a scientific meeting, your name will not be used.

*There is one condition under which confidentiality may be breached. Should you indicate active suicidal ideation, this form will be given to the researcher, Erica Shertzer, who will contact you. Because of this, we also require that you provide your name and phone number below. Please note that this form will be stored in a locked file cabinet separate from the data. Only the researcher and her faculty supervisor will have access to the files.

Name (print)______________________________

Phone______________________________

Compensation for Injury:

Although we believe that the risk of taking part in this study is minimal the following liability statement is required in all University of Montana consent forms. "In the event that you are injured as a result of this research you should seek appropriate medical treatment. If the injury is caused by the negligence of the University or any of its employees, you may be entitled to reimbursement or compensation pursuant to the Comprehensive State Insurance Plan established by the Department of Administration under the authority of M.C.A., Title 2, Chapter 9. In the event of a claim for such injury, further information can be obtained from the University’s Claim Representative or University Legal Counsel (Reviewed by University Legal Counsel, July 6, 1993)."

Voluntary Participation/Withdrawal

Your decision to participate in this project is entirely voluntary. You may refuse to take part in or you may withdraw from the study at any time without penalty or loss of benefits to which you are normally entitled. You may leave the study for any reason.

Questions

You may wish to discuss with others before you agree to take part in this study. If you have any questions about the research now or during the study contact the project investigator, Erica L. Shertzer at 243-5647. If you have any questions regarding your rights as a research subject, you may contact Dr. Rudbach through the Research Office at the University of Montana at 243-6670.
Subject's Statement of Consent

I have read the above description of this research study. I have been informed of the risks and benefits involved, and all my questions have been answered to my satisfaction. Furthermore, I have been assured that any future questions I may have will also be answered by a member of the research team. I voluntarily agree to take part in this study. I understand I will receive a copy of this consent form.

Printed Name of Subject____________________________________

Signature____________________________________ Date: ____________

Thank you for your time and effort. Please take a copy of this form with you.
APPENDIX B

Institutional Review Board Proposal for the Experimental Study

Predictors of Depressive Symptomatology:
Cognitive Theories of Vulnerability and the Relationship of Interactional and Cognitive Styles

Investigator: Erica L. Shertzer, B.A.
Supervisor: John W. Klocek, Ph.D.

1) Depression has been estimated to affect between 8 and 18% of the general population during their lifetime (Kessler, McGonagle, Swartz, Blazer, and Nelson, 1994). Given that depression has been found to be debilitating to individuals physically, mentally, and financially and may have indirect consequences for family, friends, and employers (Ingram et al., 1998), research aimed at identifying factors that play a role in the etiology, maintenance, and even prevention of depression seems highly important. In the field of depression, validated treatments such as cognitive behavioral therapy (Beck et al., 1979) are based on theoretical models that specify particular factors that contribute to both the onset and maintenance of depressive symptomatology. Therefore it is reasonable to assume that identifying unique predictors of depressive symptomatology can be useful in the development or improvement of prevention and treatment strategies that could preclude or ameliorate depressive symptoms, prevent the needless suffering of individuals and significant others, and save valuable resources of employers.

The purposed research project is designed to identify factors that may add to current understanding of depression and to existing cognitive models of vulnerability to depression. This investigation will examine the ability of factors including attributional style, hopelessness, negative life events, the tendency to seek reassurance from others, the tendency to evaluate one's external environment, coping style, social support, and anxiety to predict the development or maintenance of depressive symptomatology.

2) Participants of the proposed investigation will be approximately 300 male and female students from the University of Montana – Missoula. They will be between the ages of 18 and 65 and will be enrolled in Psychology 100 during the Spring Semester of 2001. Eight experimental credits will be given to those students who complete participation in this study. Although the present study is meant to assess factors which contribute to depressive vulnerability, participants are selected from a non-specific undergraduate population, and thus are not considered a vulnerable population.

3) Participants will consist of introductory psychology students recruited through the Psychology 100 subject pool. To recruit participants, an advertisement for the study including the project's location, duration, date, time, title, phone number of the principal investigator, and the number of experimental credits that will be provided for
participation will be posted along with a sign-up sheet will be posted on the second floor of the Skaggs building at the University of Montana for one week prior to administration. In addition, the above information will also be announced in all psychology 100 classes during the week prior to administration.

4) Participants will be assessed in large groups at previously reserved classrooms on the campus of the University of Montana.

5) All participants attend two sessions (six weeks apart) proctored by project staff. Participants will be assessed in large groups at previously reserved classrooms on the campus of the University of Montana. They will receive a questionnaire packet including an informed consent form (this will include the purpose of the project, information regarding limits of confidentiality, benefits of participation, penalties for failure to attend a scheduled session without informing project staff, legal responsibilities of project staff and the University of Montana, information regarding individuals or agencies, including the principal investigator and the Counseling and Psychological Services Center at the University of Montana to contact in case of concerns, emergencies, distress, or injury and will provide a place for participants to sign their name should they choose to participate in the study). A separate sheet will provide a place for students to write their name and create a study identification number. This packet will also include the Cognitive Style Questionnaire (CSQ), Beck Hopelessness Scale (BHS), Hopelessness Depression Symptom Questionnaire (HDSQ), Beck Depression Inventory-II (BDI-II), Beck Anxiety Inventory (BAI), Social Support Questionnaire-6 (SSQ-6), Depressive Interpersonal Relationships Inventory – Reassurance-Seeking Subscale (DIRJ-RS), Need to Evaluate Scale (NES), COPE, Hammen Perceived Negative Life Events Survey (HPNLES), and a demographic questionnaire (requesting information about the participants age, gender, race, ethnicity, marital status, education, physical illness, and past or present psychiatric diagnoses). Each participant will also receive a number two pencil and opscan answer sheets prepared on NCS Design Expert. These sheets will consist of sections that correspond to each measure. Proctors will provide information related to confidentiality, participant rights, and instructions on how to complete project measures. Participants will read and sign informed consent and complete questionnaires. It will take approximately 2 hours to complete these forms (copies of all measures are attached at the end of this document).

At the end of the first session, all participants will hand in completed packets and a signed copy of the informed consent form (participants will keep one copy of the informed consent form). All participants will sign a form that will enable project staff to give them experimental credit for participation (participants will receive four experimental credits for completing this half of the study and an additional four after completing the second session). They will then choose from one of two days (these days will be selected in advance by the PI and will be approximately six weeks after the initial session) when they will return to participate in the second session. Each participant will then be provided with a form with information including the time, date, and location of their next session, as well as a phone number where the project coordinator can be reached, and the name of the project. All data, informed consent forms, and
Identification sheets will be collected by project staff and returned to the project office located in the department of psychology. All participant answer forms will then be reviewed by administrators of the assessment sessions for responses indicating suicidal ideation on the Beck Depression Inventory - II, item #9 and on the Hopelessness Depression Symptom Questionnaire, item #29, 30, 31, and 32. As stated in the informed consent, upon finding any indication of suicidal ideation, project staff will immediately contact the PI - a graduate student in clinical psychology (supervised by a licensed clinical psychologist). In accordance with ethical responsibility, the PI will then break confidentiality, contact the student participant directly, and assess suicidal risk. Pending the outcome of this evaluation, further referrals or steps may be taken. Subsequent to this procedure, all informed consent forms will be placed in a locked file cabinet separate from the rest of the data. Only the PI will have access to these sheets. Data will also be kept in a locked file cabinet and at no time during data analysis will the identifying information contained on informed consent forms or identifier sheets be associated with the data provided by participants.

Participants will return to complete participation on the date they had selected. As a reminder, general announcements of the times, dates, and locations of the second session along with the project coordinator's campus phone number will be announced in all psychology 100 classes by course instructors the week prior to these sessions.

The second session will be conducted similarly to the initial session. This session will also be proctored by project staff, who will provide information related to confidentiality, participant rights, and instructions on how to complete the informed consent form and project measures. Participants will receive measures (CSQ, BHS, HDSQ, BDI-II, BAI, SSQ-6, DIRI-RS, NES, COPE, and HPNLES), along with an opscan form where they will record their answers and provide their identification number, and a number two pencil. Participants will also receive two copies of the informed consent form where which they will read and sign prior to participation in this portion of the study. All participants will complete study measures and one copy of the informed consent and will hand these materials in to project staff when they have finished. All participants will sign a form that will enable project staff to give them course credit for participation. Participants will receive a debriefing form that will explain the purpose, hypotheses, and potential application of the present study. Participants will be thanked for their participation in the study. Administrators of assessment sessions will immediately check for responses indicating suicidal ideation on the Beck Depression Inventory - II, item #9 and on the Hopelessness Depression Symptom Questionnaire, item #29, 30, 31, and 32. As stated above and in the informed consent, upon finding any indication of suicidal ideation, project staff will immediately contact the PI - a graduate student in clinical psychology (supervised by a licensed clinical psychologist). In accordance with ethical responsibility, the PI will then break confidentiality, contact the student participant directly, and assess suicidal risk. Pending the outcome of this evaluation, further referrals or steps may be taken. Procedures described above related to storage of informed consent forms and storage and analysis of data will also be followed after the second administration. Procedures described above related to storage of informed consent forms and storage and analysis of data will also be followed after the second administration.

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6) Participants may benefit from this study during the debriefing by learning more about the research concerning vulnerability to depression. The proposed research may benefit scientific knowledge by increasing the current understanding of the factors involved in the etiology of depression.

7) Although risks to participants of the proposed research are few, some aspects of the study may be perceived as uncomfortable. First, two of the measures, the Beck Depression Inventory – II and the Hopelessness Depression Symptoms Questionnaire contain items (BDI-II, item #9 and HDSQ, items 29, 30, 31, and 32) which assess suicidal ideation. Also, study questionnaires ask participants about some experiences they may have had, some thoughts and feelings they may have experienced, ways they may act in various situations, and about whom they rely upon for support. It is possible that some of the questions may elicit uncomfortable feelings. No physical harm is expected to result from this investigation.

8) Because the Beck Depression Inventory – II and the Hopelessness Depression Symptoms Questionnaire contain items (BDI-II, item 9 and HDSQ, items 29, 30, 31, and 32) which assess suicidal ideation, special procedures have been devised. When signing the informed consent form, subjects will be required to print their name, phone number and the unique identifying code that will identify their data sheets. All participant answer forms will be reviewed by administrators of the assessment sessions for responses indicating suicidal ideation on the Beck Depression Inventory – II, item 9 and/or the Hopelessness Depression Symptoms Questionnaire, items 29, 30, 31, and 32). As stated in the informed consent, upon finding any indication of suicidal ideation, project staff will immediately contact the PI – a graduate student in clinical psychology (supervised by a licensed clinical psychologist). In accordance with ethical responsibility, the PI will then break confidentiality, contact the student participant directly, and assess suicidal risk. Pending the outcome of this evaluation, further referrals or steps may be taken. If participants do experience uncomfortable feelings after completing study measures they will be able to contact the principle investigator, Erica L. Shertzer (243-5647) or the Counseling and Psychological Services Center (243-4711). If participants have any other questions about the study, they will be able to contact Erica Shertzer at 243-5647.

9) Participant’s names and identification codes not be will be recorded on informed consent forms. Instead identification codes and names will be recorded on a separate form. All informed consent forms will be placed in a locked file cabinet separate from the rest of the data. The identification code sheets will also be placed in a locked file cabinet and will be separate from informed consent and data. Only the PI will have access to these sheets. Data will also be kept in a locked file cabinet. At no time during data analysis will the identifying information contained on informed consent forms be associated with the data provided by participants. Participants will not be identified in any way in subsequent analyses, presentations, or publications emanating from this data. Information related to claims of injury which may be the result of participation will be provided on the informed consent form (see attached form for specific information).
10) See *Informed Consent* form attached

11) A waiver of informed consent is not applicable.

I HAVE READ THE ABOVE AND AGREE THAT IT IS AN ACCURATE
REPRESENTATION OF THE PROCEDURES TO BE USED IN THIS STUDY.

______________________________

John W. Klocek, Ph.D.

Assistant Professor of Psychology

Chairperson of Thesis Committee
APPENDIX C

Information Regarding the Study Titled Predictors of Depressive Symptomatology: Cognitive Theories of Vulnerability and the Relationship of Interactional and Cognitive Styles

Thank you for participating in this research. The questionnaires you have just completed ask about a number of areas that have been identified in previous research as potentially important factors in predicting who becomes depressed. In addition, the investigation of the role that two new constructs (the Need to Evaluate and excessive reassurance-seeking) may play is also being investigated.

The model that is being tested is that of a cognitive vulnerability to depression. This model hypothesizes that individuals who engage in particular thinking styles may be more susceptible to depression when encountering negative life events. In the face of many stressors, this susceptibility may be buffered by the presence of social support or may be exacerbated by the presence of a number of vulnerable cognitive styles. Previous research has found a substantial amount of evidence suggesting that there is a relationship between thinking styles, negative life events, and depressive symptomatology, including a subtype of depression called hopelessness depression. However, many questions remain. This research will hopefully shed further light on how these vulnerabilities operate both independently and in conjunction. In addition, the role of social support, the role of anxiety, the role of coping style, the role of Need to Evaluate and the role of excessive reassurance-seeking will be assessed in a longitudinal fashion.

The Need to Evaluate is a construct which attempts to describe the degree to which an individual feels it necessary to form opinions about the world around them. While psychology initially assumed that everyone engages in evaluative thinking at all times, this appears not to be the case. In addition, excessive reassurance-seeking is a construct that attempts to describe the interpersonal process whereby an individual feels it necessary to seek validation about how others feel about them, but are not satisfied even when feedback is provided. Individuals who engage in excessive reassurance-seeking have been found to be at risk for depression. This research will attempt to discover any role that Need to Evaluate and excessive reassurance-seeking might play in vulnerability to depression and the hopelessness depression subtype.

Thank you once again for participating in this research. Should you have further questions about this research, its findings, or theories of vulnerability to depression, please feel free to contact Erica L. Shertzer at 243-5647.

Primary Investigators: Erica L. Shertzer
Contact: Erica L. Shertzer
Department of Psychology
University of Montana
Missoula, MT 59812
(406) 243-5647
APPENDIX D

NES Q#2

Instructions:
Below are a number of descriptive statements. Using the scale below, please indicate on the bubble sheet how well each statement describes you.

1 = Extremely Uncharacteristic
2 = Somewhat Uncharacteristic
3 = Uncertain
4 = Somewhat Characteristic
5 = Extremely Characteristic

1. I form opinions about everything. 1 2 3 4 5
2. I prefer to avoid taking extreme positions. 1 2 3 4 5
3. It is very important for me to hold strong opinions. 1 2 3 4 5
4. I want to know exactly what is good and what is bad about everything. 1 2 3 4 5
5. I often prefer to remain neutral about complex issues. 1 2 3 4 5
6. If something does not affect me, I do not usually determine if it good or bad. 1 2 3 4 5
7. I enjoy strongly liking and disliking new things. 1 2 3 4 5
8. There are many things for which I do not have a preference. 1 2 3 4 5
9. It bothers me to remain neutral. 1 2 3 4 5
10. I like to have strong opinions even when I am not personally involved. 1 2 3 4 5
11. I have many more opinions than the average person. 1 2 3 4 5
12. I would rather have a strong opinion than no opinion at all. 1 2 3 4 5
13. I pay a lot of attention to whether things are good or bad. 1 2 3 4 5
14. I only form strong opinions when I have to. 1 2 3 4 5
15. I like to decide that new things are really good or really bad. 1 2 3 4 5
16. I am pretty much indifferent to many important issues. 1 2 3 4 5
Instructions:
The following questions ask about people in your environment who provide you with help or support. Each question has two parts. For the first part, list directly onto the questionnaire all the people you know, excluding yourself, whom you can count on for support in the manner described. List the persons' initials and their relationship to you (e.g., parent, sibling, friend, teacher, etc.). Do not list more than one person next to each of the letter beneath the question.

Next, on your bubble sheet, record the total number of persons using the following scale:

**HOW MANY:**

- **A** = no one
- **B** = one person
- **C** = two people
- **D** = three people
- **E** = four people
- **F** = five people
- **G** = six people
- **H** = seven people
- **I** = eight people
- **J** = nine people

For the second part, indicate how satisfied you are with the overall support you have using the following scale:

**HOW SATISFIED:**

- **A** = very dissatisfied
- **B** = fairly dissatisfied
- **C** = a little dissatisfied
- **D** = a little satisfied
- **E** = fairly satisfied
- **F** = very satisfied

**NOTE:** If you had no support for a question, bubble in "A" for "no one" but still rate your level of satisfaction. Please answer all the questions as best you can.

1. Who can you really count on to be dependable when you need help?

   - **A) NO ONE**
   - **B)**
   - **C)**
   - **D)**
   - **E)****
   - **F)**
   - **G)**
   - **H)****
   - **I)**
   - **J)**

2. How satisfied?

   **Very Dissatisfied**
   **A**  **B**  **C**  **D**  **E**  **F**  **Very Satisfied**
3. Who can you really count on to help you feel more relaxed when you are under pressure or tense?

A) NO ONE  E)    H)  
B)  F)    I)  
C)  G)    J)  
D)  

4. How satisfied?

<table>
<thead>
<tr>
<th>Very Dissatisfied</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>F</th>
<th>Very Satisfied</th>
</tr>
</thead>
</table>

5. Who accepts you totally, including both your worst and best points?

A) NO ONE  E)    H)  
B)  F)    I)  
C)  G)    J)  
D)  

6. How satisfied?

<table>
<thead>
<tr>
<th>Very Dissatisfied</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>F</th>
<th>Very Satisfied</th>
</tr>
</thead>
</table>

7. Who can you really count on to care about you, regardless of what is happening to you?

A) NO ONE  E)    H)  
B)  F)    I)  
C)  G)    J)  
D)  

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8. How satisfied?

Very Dissatisfied   A   B   C   D   E   F   Very Satisfied

9. Who can you really count on to help you feel better when you are feeling generally down-in-the-dumps?

A) NO ONE   E)   H)
B)   F)   I)
C)   G)   J)
D)

10. How satisfied?

Very Dissatisfied   A   B   C   D   E   F   Very Satisfied

11. Whom can you count on to console you when you are very upset?

A) NO ONE   E)   H)
B)   F)   I)
C)   G)   J)
D)

12. How satisfied?

Very Dissatisfied   A   B   C   D   E   F   Very Satisfied
Instructions:
We are interested in how people respond when they confront difficult or stressful events in their lives. There are lots of ways to deal with stress. This questionnaire asks you to indicate what you generally do and feel, when you experience a stressful event. Obviously, different events bring out somewhat different responses, but think about what you usually do when you are under a lot of stress. Then respond to each of the following items by blackening in one number on your answer sheet using the following choices:

1 = I usually don't do this at all  
2 = I usually do this a little bit  
3 = I usually do this a medium amount  
4 = I usually do this a lot

Please try to respond to each item separately in your mind from each other item. Choose your answers thoughtfully, and make your answers as true for you as you can. Please answer every item. There are no "right" or "wrong" answers, so choose the most accurate answer for you – not what you think "most people" would say or do. Indicate what YOU do when YOU experience a stressful event.

1. I try to grow as a person as a result of the experience.  
2. I turn to work or other substitute activities to take my mind off things.  
3. I get upset and let my emotions out.  
4. I try to get advice from someone about what to do.  
5. I concentrate my efforts on doing something about it.  
6. I say to myself "this isn't real."  
7. I put my trust in God.  
8. I laugh about the situation.  
9. I admit to myself that I can't deal with it, and quit trying.  
10. I restrain myself from doing anything too quickly.  
11. I discuss my feelings with someone.  
12. I use alcohol or drugs to make myself feel better.  
13. I get used to the idea that it happened.  
14. I talk to someone to find out more about the situation.  
15. I keep myself from getting distracted by other thoughts or activities.  
16. I daydream about things other than this.  
17. I get upset, and am really aware of it.  
18. I seek God's help.  
19. I make a plan of action.
20. I make jokes about it.

21. I accept that this has happened and that it can't be changed.
22. I hold off doing anything about it until the situation permits.
23. I try to get emotional support from friends or relatives.
24. I just give up trying to reach my goal.
25. I take additional action to try to get rid of the problem.
26. I try to lose myself for a while by drinking alcohol or taking drugs.
27. I refuse to believe that it has happened.
28. I let my feelings out.
29. I try to see it in a different light, to make it seem more positive.
30. I talk to someone who could do something concrete about the problem.

31. I sleep more than usual.
32. I try to come up with a strategy about what to do.
33. I focus on dealing with this problem, and if necessary let other things slide a little.
34. I get sympathy and understanding from someone.
35. I drink alcohol or take drugs, in order to think about it less.
36. I kid around about it.
37. I give up the attempt to get what I want.
38. I look for something good in what is happening.
39. I think about how I might best handle the problem.
40. I pretend that it hasn't really happened.

41. I make sure not to make matters worse by acting too soon.
42. I try hard to prevent other things from interfering with my efforts at dealing with this.
43. I go to movies or watch TV, to think about it less.
44. I accept the reality of the fact that it happened.
45. I ask people who have had similar experiences what they did.
46. I feel a lot of emotional distress and I find myself expressing those feelings a lot.
47. I take direct action to get around the problem.
48. I try to find comfort in my religion.
49. I force myself to wait for the right time to do something.
50. I make fun of the situation.

51. I reduce the amount of effort I'm putting into solving the problem.
52. I talk to someone about how I feel.
53. I use alcohol or drugs to help me get through it.
54. I learn to live with it.
55. I put aside other activities in order to concentrate on this.
56. I think hard about what steps to take.
57. I act as though it hasn't even happened.
58. I do what has to be done, one step at a time.
59. I learn something from the experience.
60. I pray more than usual.
DIRI-RS Q# 6

Instructions:
This questionnaire consists of 4 groups of statements. Please read each group of statements carefully and then pick out the one statement in each group that is the most appropriate to you using the following scale.

1 = no, not at all
2 = no, hardly ever
3 = not really
4 = I'm not sure
5 = yes, somewhat
6 = yes, quite often
7 = yes, very much

Bubble in the number of the statement you have chosen on your answer sheet. If several statements in the group seem to apply equally well, bubble in the highest number for that group. Be sure that you do not choose more than one statement for any group.

1) In general, do you find yourself often asking the people you feel close to how they truly feel about you?

1 2 3 4 5 6 7
no, not no, hardly not really I'm not yes, yes, quite yes, very
at all ever sure somewhat often
much

2) In general, do you frequently seek reassurance from the people you feel close to as to whether they really care about you?

1 2 3 4 5 6 7
no, not no, hardly not really I'm not yes, yes, quite yes, very
at all ever sure somewhat often
much

3) In general, do the people you feel close to sometimes become irritated with you for seeking reassurance from them about whether they really care about you?

1 2 3 4 5 6 7
no, not no, hardly not really I'm not yes, yes, quite yes, very
at all ever sure somewhat often
much

4) In general, do the people you feel close to sometimes get "fed up" with you for seeking reassurance from them about whether they really care about you?

1 2 3 4 5 6 7
no, not no, hardly not really I'm not yes, yes, quite yes, very
at all ever sure somewhat often
much

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Instructions:
Listed below are a number of events that sometimes bring about change in the lives of those who experience them. We ask that you consider the last four weeks when answering this questionnaire. Please indicate the extent to which you view each event as having either a positive or negative impact on your life. That is, indicate the type and extent of impact that the event had according to the scale below. If you have not experienced the event in the last four weeks, please bubble in "H."

Extremely Negative  Moderately Negative  Somewhat Negative  No Impact  Slightly Positive  Moderately Positive  Extremely Positive  Did not Occur
A     B     C     D     E     F     G     H

WORK AND/OR SCHOOL
1. Starting a new job in a new line of work.
2. Starting a new job in the same line of work.
3. Increase in hours of job and/or schoolwork.
4. Decrease in hours of job and/or schoolwork.
5. Not getting an expected advancement. (e.g., promotion, raise, acceptance to a better school, etc...).
6. An outstanding personal achievement.
7. Changing to a new school at the same academic level (e.g., transferring to a new school to continue undergraduate work).
8. Changing your major.
9. Failing a course.
10. Dropping a course.
11. Being expected to take over more without a promotion.
12. Promotion with increase of responsibilities at work.
13. Being downgraded or demoted at work.
15. Problems in finding desired work.
16. Problems in choosing appropriate work.
17. Being laid off.
18. Re-entering school after a break of at least one year.
19. Graduation from high school.
20. Dropping out of school due to financial difficulties.
21. Dropping out of school due to academic difficulties.
22. Dropping out of school for other reasons.
23. Being put on academic probation.
24. Failing an important exam.
25. Trouble with your boss.
26. Trouble with your co-workers.
27. Trouble with your professor.
28. Other events concerning work or school.

FINANCES
29. Having a major unexpected expense (e.g., hospital bill, car repairs, etc.).
30. Income decreased substantially.
31. Income increased substantially.
32. Other events concerning finances.

HEALTH
33. Sudden and serious impairment of your vision or hearing.
34. Having an operation.
35. Learning that your operation was not helpful.
36. Women only: Unwanted pregnancy.
37. Women only: Wanted pregnancy.
38. Women only: Miscarriage.
39. Women only: Abortion.
40. Men only: Girlfriend has unwanted pregnancy.
41. Men only: Girlfriend has wanted pregnancy.
42. Men only: Girlfriend has miscarriage.
43. Men only: Girlfriend has an abortion.
44. Serious physical illness requiring hospital treatment.
45. Serious physical injury requiring hospital treatment.
46. Illness which did not require hospitalization, but which did keep you in bed or at home for a week or more.
47. An injury which did not require hospitalization, but which did keep you in bed or at home for a week or more.
48. Problems related to alcohol or drugs.
49. Other events concerning your health.

ROMANTIC RELATIONSHIPS
50. Began a new relationship.
51. Became engaged.
52. Increase in number of arguments with girlfriend/boyfriend.
53. Relationship with girlfriend/boyfriend changed for the better.
54. Sexual difficulties.
55. You learn that your girlfriend/boyfriend has been unfaithful.
56. You have an affair.
57. Break up of affair.
58. Break up with girlfriend/boyfriend.
59. Steady girlfriend/boyfriend moves to a new city or new area
60. Death of girlfriend/boyfriend.
61. Girlfriend/boyfriend develops serious physical illness that requires hospitalization.
62. Girlfriend/boyfriend has a serious injury that requires hospital treatment.
63. Learning that an operation was not helpful for girlfriend/boyfriend.
64. Girlfriend/boyfriend develops serious psychiatric problem that requires hospital treatment.
65. Girlfriend/boyfriend starts drinking heavily, attempts suicide, or is sent to prison.
67. Girlfriend/boyfriend is raped.
68. Girlfriend/boyfriend is robbed.
69. Girlfriend/boyfriend is physically threatened, attacked, or assaulted.
70. Other events concerning a romantic relationship or dating.
HOME, FRIENDS, AND FAMILY LIFE

71. Move to a different city or area.
72. Move within same city or area.
73. Leaving home for the first time.
74. Period of homelessness.
75. Parent moves away to another city.
76. A close friend moves away to another city or area.
77. Parents get divorced or separated while you are living with them.
78. Parents get divorced or separated. You have not been living with them.
79. Death of immediate family member with whom you are living.
80. Death of immediate family member. You were not living with him/her at the time.
81. Death of a close friend.
82. Death of an acquaintance (e.g., neighbor, co-worker, etc.).
83. Immediate family member develops serious physical illness that requires hospital treatment.
84. Close friend or roommate develops serious physical illness that requires hospital treatment.
85. Immediate family member has a serious injury that requires hospital treatment.
86. Close friend or roommate has a serious injury that requires hospital treatment.
87. Immediate family member starts drinking heavily, attempts suicide, or is sent to prison.
88. Close friend or roommate starts drinking heavily, attempts suicide, or is sent to prison.
89. Trouble with parents.
90. Quarrel with neighbor or roommate.
91. Joining a fraternity or sorority.
92. Being turned down from a desired fraternity or sorority.
93. Substantial increase in social activities (e.g., parties, movies, visiting friends).
94. Substantial decrease in social activities (e.g., parties, movies, visiting friends).
95. Learning that an operation (surgery or other major treatment) was not helpful for immediate family member.
96. Learning that an operation (surgery or other major treatment) was not helpful for a close friend.
97. Immediate family member is sent to a nursing home.
98. Immediate family member develops a psychiatric problem that requires hospitalization.
99. Close friend develops a psychiatric problem that requires hospitalization.

100. Having a pet become seriously ill.
101. Having a pet die.
102. A close friend or family member was raped.
103. A close friend or family member is robbed.
104. A close friend or a family member is physically threatened, attacked, or assaulted.
105. Begin a new close friendship.
106. A "falling out" of a close personal friendship.
107. Other events concerning home, friends, or family.
108. Victim of auto accident.
109. Victim of natural disaster (e.g., fire, mudslides, etc.).

110. Having something you own otherwise damaged (e.g., vandalism).
111. Having your home robbed.
112. Having your car stolen.
113. Being robbed.
114. Being physically threatened, attacked, or assaulted.
115. Being raped.

PERSONAL EVENTS
116. Being found guilty of a minor legal violation (e.g., traffic ticket, jay walking, etc.).
117. Being involved in a lawsuit.
118. Being arrested or detained by legal authorities.
119. Appearing in court.
120. Other personal events.
Demographic Information

Please record the following information on your answer sheet in the space marked DQ by blackening in the numbers that correspond with your answer.

1) Identification Code (5 digit-letter-number combination)

2) Age

3) Gender 0 = Male 1 = Female

4) Race/Ethnicity 1 = African American 2 = Native American 3 = Caucasian 4 = Hispanic/Latino(a) 5 = Asian 6 = Pacific Islander 7 = Other

5) Marital Status 1 = Single 2 = Married 3 = Cohabitating 4 = Separated 5 = Divorced

6) Physical Illness 1 = Presence of Physical Illness (chronic e.g., diabetes, arthritis) 2 = Presence of Physical Illness (non-chronic e.g., cold, flu, broken leg) 3 = No Illness Present

7) Past or Current Psychiatric Diagnoses 1 = Current diagnosis 2 = Past diagnosis 3 = No diagnosis

8) Years of Education Completed
The following measures could not be included in APPENDIX D due to copyright laws:

1) The Beck Depression Inventory – II (BDI-II)

2) The Beck Anxiety Inventory – II (BAI)

3) The Beck Hopelessness Scale (BHS)

The following measure could not be included in APPENDIX D because the authors have not given permission to do so:

1) The Cognitive Style Questionnaire (CSQ)

* Please note that references for each of these measures are included in the reference section of this document.