The Dissertation Committee for Jennifer Ann Herren certifies that this is the approved version of the following dissertation:

Investigation of Anxiety Symptoms in a Cognitive-Stress Mediational Model of Depression in Early Adolescent Girls

Committee:

_____
Kevin D. Stark, Supervisor

_____
Deborah J. Tharinger

_____
Janay B. Sander

_____
Susan N. Beretvas

_____
Alexandra Loukas

Investigation of Anxiety Symptoms in a Cognitive-Stress Mediational Model of Depression in Early Adolescent Girls

by

Jennnifer Ann Herren, B.A.; M.A.

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DEDICATION

I am honored to dedicate this body of work to my parents who taught me the importance of persistence, patience, and kindness. Your unwavering support and unconditional love have been my anchors and given me the confidence to pursue my dreams.

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Investigation of Anxiety Symptoms in a Cognitive-Stress Mediational Model of Depression in Early Adolescent Girls

Jennifer Ann Herren, Ph.D.

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Supervisor: Kevin D. Stark

Previous research indicates an increase in the prevalence of depression around adolescence, especially for females. Research suggests depressogenic cognitions play an essential role in the development of depression and may mediate the relation between risk factors and depression. Research has also shown the family environment, negative life events, and maternal depression are all related to the development of depressogenic cognitions. Additionally, few studies have tested models of depression while measuring both anxiety and depressive symptoms despite the high rates of comorbidity between the two disorders. The current study used path analytic techniques to integrate correlates of depression while accounting for comorbid anxiety symptoms in comprehensive model of depression for early adolescent girls. Participants included 203 girls, aged 9-14, along with their mothers. Participants completed self-report measures of the family environment, cognitive triad, and negative life events. Mothers of participants completed a self-report measure of psychopathology. Participants also completed a semi-structured diagnostic interview, which served as the measure for symptoms of depression and anxiety. Results supported previous literature finding a more depressogenic cognitive

triad was significantly associated with higher depressive severity. Family environments, characterized by more cohesive and less conflictual family relationships, more communication, and higher engagement in social/recreational activities, were significantly associated with a more positive cognitive triad. Additionally, more negative life events were significantly associated with a more depressogenic cognitive triad. Both family social/recreational activities and negative life events had significant indirect effects on depression. Results indicated a strong relation between anxiety and depression, with anxiety having a significant positive direct effect on depression. The pathways from maternal depression and anxiety to the cognitive triad, anxiety symptoms to the cognitive triad, as well as family environment variables, maternal depression and anxiety and negative life events to anxiety symptoms were not found to be significant. Results from an exploratory analysis suggest anxiety may moderate the relation between the cognitive triad and depression. Implications of these results, limitations, and recommendations for future research are provided.

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CHAPTER 1

Introduction

Depression during childhood is a common, recurrent, and disabling disorder (Kovacs, 2006; Kovacs, Feinberg, Crouse-Novak, Paulauskas, & Finkelstein, 1984). The prevalence of depression increases from childhood to adolescence (Costello, Erkanli, & Angold, 2006). Prior to adolescence the rates of depression are similar for boys and girls; however, gender differences begin to emerge around the age of 13 (Hankin & Abramson, 2001). Adolescence presents as a time of heightened vulnerability for depression in girls because the female rate doubles the prevalence rate for males between the ages of 13 to 15 (Hankin et al., 1998). This trend continues into adulthood, as adult women are approximately twice as likely to be depressed as adult men (Nolen-Hoeksema & Girgus, 1994). Theories to explain this gender discrepancy are based upon a diathesis-stress framework, suggesting girls have unique vulnerabilities, which put them at risk when faced with the challenges of adolescence (Cyranowski, Frank, Young, & Shear, 2000; Hankin & Abramson, 2001; Nolen-Hoeksema & Girgus, 1994). For instance, girls experience more interpersonal stress than boys and are more reactive to this stress (Rudolph & Hammen, 1999). Thus, early adolescence presents as a critical stage of development directly prior to the emergence of the gender-gap in depression; therefore, it is important to study this time of development in girls. One advantage of this study is it focuses on early adolescent girls, aged 9-14.

Some of the major theoretical approaches regarding the etiology of depression include biological models (genetics and neurochemistry), cognitive models, behavioral/interpersonal models, family models, and life stress models (Hammen & Rudolph, 2003). This body of research highlights risk factors associated with depression in childhood and adolescence, which aid in the development of appropriate prevention programs and interventions. Understanding the etiological pathways of depression is an important area of research because it promotes the development of effective interventions. Given the large number of processes, mechanisms, and risk factors associated with the etiology of depression, an integrative model evaluating these factors simultaneously is needed to understand the etiology of depression in early adolescence (Hankin, 2006). In addition, etiological models of depression should not be adapted from adult models, but should be developmentally sensitive to children (Cicchetti & Toth, 1998). This investigation, therefore, explores a multi-dimensional model of depression integrating several correlates of depression for early adolescent girls.

Environmental and biological influences of the family are important factors to consider when conceptualizing the etiology of depression in children and adolescents. Depression runs in families; however, it is difficult to determine the relative effects of the genetic transmission of depression between generations from the psychosocial effects found in families with a depressed parent (Goodman & Gotlib, 1999). Nevertheless, having a depressed parent is clearly associated with childhood depression and can be considered one of the strongest predictors of childhood and adolescent depression (Beardslee, Versage, & Gladestone, 1998). Additionally, research linking maternal depression and anxiety to internalizing disorders in offspring (Swartz et al., 2005) as well

as the high comorbidity rates between these disorders in women (Kessler et al., 1994) suggests the importance of considering both maternal depression and anxiety when investigating symptoms of depression and anxiety in youth. Research has found depressed children have families that are less cohesive, more controlled and conflictual, communicate less, express emotion more intensely, and engage in fewer social and recreational activities (Barerra & Garrison-Jones, 1992; Cole & McPherson, 1993; Jewell & Stark, 2003; Ostrander, Weinfurt, & Nay, 1998; Stark, Humphrey, Crook, & Lewis, 1990). In general, research indicates multiple family factors are associated with depression in childhood (Sander & McCarty, 2005). This investigation examines maternal depression and anxiety as well as other family environment variables to determine their relation to depression in early adolescent girls.

In addition to familial influences, cognitive models of depression have been widely researched. Cognitive diathesis-stress models of depression are theories that attempt to explain variability in response to stress. These theories suggest depression is the result of the interaction between personal cognitive vulnerability and stress. Beck's theory (1967) focuses on the schemas of individuals, suggesting people who possess depressogenic cognitions have an increased vulnerability to depression when faced with life stress. An important part of the theory suggests cognitions are often defined as distorted negative perceptions of the self, world, and future, which are referred to as the cognitive triad. Several studies have supported Beck's theory of cognitive vulnerability to depression in children and adolescents (Alloy et al., 1999; Epkins, 1996; Garber, Weiss, & Shanley, 1993; Jolly, 1993; Kendall, Stark, & Adam, 1990; Lewinsohn, Joiner, & Rohde, 2001). Some research, however, suggests an interaction between cognitive

vulnerability and stress may not be applicable to children given that cognitive origins are thought to develop in childhood (Cole & Turner, 1993). Research indicates the cognitive diathesis-stress model may not emerge until adolescence or adulthood (Abela, 2001); instead, depressogenic cognitions play an essential role in the development of depression in children by mediating the relation between risk factors and depression (Cole & Turner). This study attempts to replicate and build upon these findings by presenting a cognitive-stress mediational model of depression that is pertinent to early adolescent girls.

Given that depressogenic cognitions likely mediate the relation between some risk factors and depression in children, it is important to understand what factors may contribute to the development of maladaptive cognitions. Beck and colleagues (1979) hypothesize depressive schemata are formed through early learning experiences, especially those within the family. Researchers have shown children develop depressogenic cognitions via their parents through modeling, direct learning from the parents' maladaptive cognitions, or indirect learning through the family environment (Alloy et al., 2001). Research suggests exposure to a depressed mother may be one process by which negative cognitions develop in children (Garber & Flynn, 2001). The family environment has been another avenue through which children develop maladaptive cognitions. Lower levels of maternal acceptance (Garber & Flynn, 2001; Rudolph, Kurlakowsky, & Conley, 2001) and higher levels of rejection (Rudolph et al.) have been associated with negative cognitions in adolescents. Negative parenting, often characterized by harsh, critical and rejecting environments, has been associated with maladaptive cognitions in multiple studies (Gibb et al., 2001; Mezulis, Hyde, & Bilck,

2006; Rose, Abramson, Hodulik, Halberstadt, & Leff, 1994). Additionally, the experience of negative life events is likely one of the most important factors underlying the development of cognition (Garber & Flynn, 2001). Multiple studies have shown a direct relation between negative life events and depressogenic cognition (Cole & Turner, 1993; Garber & Flynn, 2001; Rienemann & Teeter Ellison, 2004; Rudolph et al., 2001; Tram & Cole, 2000). Ingram (2003) suggests cognition is the "final pathway," holding together the vulnerability process to depression in children. This study, therefore, presents a model in which maternal depression and anxiety, family environment variables, and negative life events are directly associated with the cognitive triad in early adolescent girls. In addition, the cognitive triad is predicted to mediate the relation between these risk factors and depressive severity. In other words, an indirect relation between maternal depression and anxiety, family environment variables, and negative life events and depressive severity is proposed.

One of the major limitations to current integrative models of depression is the models do not account for psychiatric comorbidity. In fact, there is a methodological need to assess both depression and its' co-occurring symptoms to investigate whether hypothesized vulnerability factors predict the development of the disorder specifically or the comorbid symptoms presented (Hankin, 2006; Seligman & Ollendick, 1998). Without taking comorbid symptoms into account when studying depression, erroneous conclusions and theories may emerge (Keiley, Lofthouse, Bates, Dodge, & Pettit, 2003; Rutter & Sroufe, 2000). Accounting for comorbid symptoms is crucial because research suggests "pure" depression is often less common than depression with a comorbid disorder (Avenevoli, Stolar, Li, Dierker, & Merikangas, 2001), and comorbidity should

be considered the rule rather than the exception in childhood depression (Biederman, Faraone, Mick, & Lelon, 1995). This study, therefore, examines the role of comorbid anxiety symptoms in an integrative cognitive-stress mediational model of depression in early adolescent girls.

Depression is most commonly associated with anxiety disorders, disruptive disorders, and attention deficit hyperactivity disorders (Angold, Costello, & Erkanli, 1999), and the presence of a psychiatric comorbid disorder with depression is associated with a number of negative outcomes (Birmaher et al., 1996). Emerging research suggests rates and manifestations of comorbidity in depression may vary by age and sex. During childhood, girls do not experience comorbidity between depression and disruptive disorders as often as comorbidity between anxiety and depression (Kovacs, Obrosky, & Sherrill, 2003). The prevalence of anxiety and depression in girls is high throughout childhood and adolescence. This study, therefore, examines anxiety symptomatology because it appears to be most salient for girls with depression in childhood or early adolescence.

Depression and anxiety are strongly related in children and adolescents with significant diagnostic overlap. The rates of comorbidity for anxiety and depression can be as high as 70%, but typically range from 20% to 50% (Angold et al., 1999; Brady & Kendall, 1992). Comorbid anxiety and depression is often more common than the "pure" forms of these disorders (Zahn-Waxler, Klimes-Dougan, & Slattery, 2000). The tripartite model, proposed by Clarke and Watson (1991), suggests depression and anxiety share a general factor, negative affect, but each have unique factors distinctive to the disorder. A number of theoretical models have been proposed to account for comorbidity among

psychiatric disorders, including methodological artifacts, diagnostic artifacts, inaccurate boundaries, and substantive causes (Angold et al., 1999; Klein & Riso, 1993). Angold and colleagues ruled out most of the artifactual accounts of comorbidity and suggest comorbidity is a real phenomenon likely accounted for by substantive causes. Comorbidity, therefore, most likely results from shared etiological pathways of having common risk factors or the disorders directly influencing each other (Klein & Riso). Based upon the substantive theories of comorbidity, this study explores whether risk factors associated with depression are common to comorbid anxiety symptoms as well as whether anxiety influences depressive severity.

Given that depression and anxiety commonly co-occur together, it is possible they share similar etiological pathways. As with depression, negative life events have been associated with anxiety symptoms and disorders in many studies (Boer et al., 2002; Cuffe, McKeown, Addy, & Garrison, 2005; Gotheif, Aharonovsky, Horesh, Carty, & Apter, 2004; Phillips, Hammen, Brennan, Najman, & Bor, 2005). Additionally, family factors have been cited as a possible contributor in the development of anxiety in children (Muris, 2006). As with childhood depression, maternal depression has been cited as a risk factor for childhood anxiety. In addition, most anxiety disorders in children of mothers who are depressed have a preadolescent onset, suggesting the onset of anxiety among children of depressed parents may precede the onset of depression (Pilowsky et al., 2006; Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997). Maternal anxiety has also been found to significantly predict anxiety disorders in offspring (McClure, Brennan, Hammen, & Le Brocque, 2001). Aspects of the family environment and parent rearing behaviors have been associated with anxiety in children. Family environments

characterized by low support, more conflict, less sociability, enmeshment, and controlling behaviors have been associated with anxious children (Stark et al., 1990). Parental control and rejection have also been consistently related to childhood anxiety (Rappe, 1997). Research, therefore, suggests stressful events and familial factors are associated with both anxiety and depression in children and adolescents. This study examines the direct effects of maternal depression and anxiety, negative life events, and the family environment on anxiety symptom severity in girls and whether anxiety mediates the relation between these risk factors and depressive severity.

Anxiety has been found to precede the initial onset of major depression in several samples (Avenevoli et al., 2001; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Lewinsohn, Zinbarg, Seeley, Lewinsohn, & Sack, 1997) and is cited as a likely risk factor to depression (Flannery-Schroeder, 2006; Seligman & Ollendick, 1998; Wittchen, Beesdo, Bittner, & Goodwin, 2003). In addition to preceding depression, children with comorbid anxiety and depression tend to have a more severe depression than individuals with depression alone. This research suggests anxiety, therefore, may play a role in the development of depression. Based on the literature, however, it is still unclear how anxiety may influence the development of depression. Researchers hypothesize it may be related to increased stressors due to functional impairment from the anxiety disorder, a possible genetic link between the two disorders, and shared environment or psychosocial factors (Bittner et al., 2004). Research has also found an association between anxiety and the cognitive triad (Jacobs & Joseph, 1997; Laurent & Stark, 1993); however, it is unclear whether anxiety symptoms may contribute to the development of depressogenic cognitions. This study, therefore, investigates the direct effects anxiety has on depressive

severity and the cognitive triad. Considering anxiety typically precedes the onset of major depression, this study examines whether anxiety mediates the relation between risk factors and childhood depression.

In sum, this study examines a model of depression that integrates important theoretical constructs. There are two general goals: (1) to replicate and build upon a cognitive-stress mediational model of childhood depression, with depressogenic cognitions mediating the relation between stressors and depression, and (2) to present a model of childhood depression integrating comorbid anxiety symptoms as a possible mediator and risk factor for childhood depression. Given these goals, the first step was to examine the relation between depressogenic cognitions and depression within a population of early adolescent girls. Secondly, anxiety symptom severity was explored to determine the effect on both the girls' cognitive triad and depressive symptoms. Stress variables related to the family environment (i.e. maternal depression and anxiety, family relationships, family communication, and family social/recreational engagement) were investigated to determine the direct effects on the girls' cognitive triad and severity of anxiety symptoms. In addition, the indirect effects of these family variables were analyzed to determine whether anxiety symptoms and the girls' cognitive triad mediate the relation between these variables and depressive symptoms in early adolescent girls. The direct effects of negative life events on the girls' cognitive triad and anxiety symptoms were analyzed. Indirect effects of negative life events on depressive severity were examined.

An integrative, cognitive-stress model of depression for early adolescent girls that accounts for comorbid anxiety symptoms is examined. Building upon existing research

studying etiological pathways to depression in children and adolescents, this study strives to gain a better understanding of relations among family functioning, cognitions, negative life events, comorbid anxiety, and depression in early adolescent females. By delineating these complex pathways, better intervention and prevention strategies can be explored.

CHAPTER 2

Review of the Literature

Depression in Childhood and Adolescence

Depressive disorders are among the most common worldwide psychiatric disorders, have substantial social costs exceeding those of major health problems, and are considered one of the leading disabling conditions worldwide (Lopez, Mathers, Ezzati, Jamison, & Murray, 2006; Wells, Sturm, Sherbourne, & Meredith, 1996). While there is no definitive number on the economic costs of depression in children and adolescents, the cost is likely to be substantial affecting numerous groups, including healthcare, schools, child serving agencies, and families (Lynch & Clarke, 2006). Depression among children is a common, recurrent and disabling disorder (Kovacs, 2006; Kovacs et al., 1984) associated with significant functional impairment in school and relationships (Birmaher et al., 1996; Puig-Antich et al., 1993). Birmaher and colleagues estimate the lifetime prevalence rate of major depressive disorder in adolescents ranges from 15% to 20% and suggest depression in adults often begins in adolescence. Before the age of 18, approximately 20% of youth in the United States may experience a depressive episode (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993). Despite the high prevalence of depression, the number of depressed children and adolescents not being diagnosed or treated for depression is striking (Beardslee, Keller, Lavori, Staley, & Sacks, 1993; Wells, Kataoka, & Asarnow, 2001).

Clinical Presentation

The Diagnostic and Statistical Manual of Mental Disorders Fourth Edition Text Revised (DSM-IV-TR) recognizes three primary unipolar depressive disorders, including Major Depressive Disorder (MDD) (Appendix A), Dysthymic Disorder (DD) (Appendix B), and Depressive Disorder Not Otherwise Specified (DDNOS) (Appendix C; American Psychiatric Association, 2000). In order to meet criteria for a Major Depressive Episode, individuals must experience a period of at least 2 weeks with a depressed mood or the loss of interest or pleasure in most all activities. Children and adolescents, however, may experience an irritable or cranky mood instead of sadness. Individuals must additionally experience at least four other symptoms from a number of symptoms, including: changes in appetite or weight; sleep disturbance; psychomotor agitation or retardation; fatigue or loss of energy; feelings of worthlessness or excessive guilt; difficulty thinking or concentrating; or recurrent thoughts related to death or suicidal ideation, plans, or attempts. These symptoms need to occur for most of the day, almost everyday, for a minimum of two consecutive weeks. In addition, distress or impairment in social, occupational, or other important areas of functioning must accompany the episode (American Psychiatric Association).

According to the American Psychiatric Association (2000), MDD is characterized by having one or more Major Depressive Episodes without a history of Manic, Hypomanic or Mixed episodes. MDD may be a single episode or recurrent condition. Symptom expression of MDD in children and adolescents is generally similar to MDD in adults (Kovacs, 1996; Mitchell, McCauley, Burke & Moss, 1988); however, the clinical presentation of MDD may differ across different developmental and ethnic groups

(American Academy of Child and Adolescent Psychiatry, 1998). For example, neurovegetative symptoms, such as hypersomnia, are more likely to be reported in older individuals with depression than in children (Kovacs et al., 2003; Ryan et al., 1987). Differences in symptom expression may be related to children's limited cognitive, social, emotional or biological capacities across developmental periods (Hankin, 2006). Children with depression think about and attempt suicide just as often as depressed adolescents and both groups attempt suicide more than adults (Mitchell et al.). Approximately 90% of major depressive episodes remit within two years of onset; however, MDD is a recurrent condition (Birmaher et al., 1996). The majority of individuals, approximately 60%, who experience MDD can expect to have a second episode (American Psychiatric Association).

Dysthymic Disorder (DD) is characterized by a chronically depressed mood for the majority of days for at least 2 years (American Psychiatric Association, 2000). In children, the mood may be irritable rather than depressed and the duration requirement is only 1 year. Individuals must also experience a minimum of two of the following symptoms: appetite disturbance; insomnia or hypersomnia; low energy or fatigue: low self-esteem; poor concentration or difficulty making decisions; or feelings of hopelessness (American Psychiatric Association). In a study of children with DD only, DD was characterized by feelings of being unloved, a gloomy mood, anger, and self-deprecation (Kovacs, Akiskal, Gatsonis & Parrone, 1994). Symptoms of DD may be less recognizable because they are often a part of the individual's day-to-day experiences and may be attributable to "how they are" (American Psychiatric Association). While change in mood in DD is considered to be less intense than MDD, the mood disturbance is more

chronic (American Academy of Child and Adolescent Psychiatry, 1998). A diagnosis of DDNOS is given if individuals are experiencing symptoms of depression that are present more often than not, the symptoms cause significant impairment, and the individual does not meet the criteria for MDD or DD (American Psychiatric Association).

Epidemiology

Overall, the prevalence of depression tends to increase with age until early adolescence when the rate of depression dramatically increases (Lewinsohn, Clarke, Seeley & Rohde, 1994). Epidemiological studies indicate the prevalence rates of depression in children are between 0.4% and 2.5% and between 0.4% and 8.3% in adolescents (Birmaher et al., 1996). The lifetime incidence is considered to be around 20% by the time children reach 18 years of age (Lewinsohn et al., 1993), which is comparable to the adult lifetime rate of MDD (Birmaher et al.). Research investigating depression in preschoolers is relatively new and prevalence rates among this age group are uncertain; however, reported prevalence for preschoolers is approximately 1% (Stalets & Luby, 2006; Luby et al., 2002). A recent meta-analysis reviewing over 30 years of literature suggests the prevalence of depression increases from childhood to adolescence and the rate of depression is higher for adolescent girls than adolescent boys (Costello et al., 2006). Results indicate prevalence rates are 2.8% (standard error (SE) .5%) for children under 13, 5.7% (SE .3%) for adolescents aged 13-18, 5.9% (SE .3%) for adolescent girls, and 4.6% (SE .3%) for adolescent boys (Costello et al.). Results from a large community sample of high school students found a point prevalence rate of 2.9% for unipolar depression, but a lifetime prevalence rate of 20.4% and 25.3% at two different time points (Lewinsohn et al., 1993). The overall results of these studies suggest

the incidence of depression in adolescents is high. In addition, a substantial number of adolescents report experiencing a depressed mood, 20% to 35% of boys and 20% to 45% of girls, which may represent a precursor to the development of depression (Peterson et al., 1993). Overall, the rates of depression steadily increase with age and dramatically increase in adolescence, with up to a sixfold increase from early adolescence to late adolescence (Hankin, 2006).

Research remains unclear as to whether the prevalence of depression is rising. Ryan et al. (1992) suggest the number of children and adolescents with depression is rising and there is a decreasing age of onset. Based on samples of children and adults with depressive disorders, a secular increase in depression has been noted, and individuals born in the late 20th century are at a greater risk for a mood disorder (Birmaher et al., 1996; Ryan et al.). A recent meta-analysis of studies, however, examined prevalence of depression for children born over the past 30 years and found no evidence of an increasing prevalence in later born cohorts (Costello et al., 2006). Costello et al. argue the secular increase reported in Ryan et al's study might be attributable to the use of a clinical sample. The authors suggest there is a societal heightened awareness of an under-diagnosed disorder rather than an increase in prevalence of depression in childhood.

Course

First depressive episodes generally occur in adolescence (Hankin, 2006) and most adult depression is preceded by adolescent or childhood depression (Kim-Cohen et al., 2003; Pine, Cohen, Gurley, Brook & Ma, 1998). Kim-Cohen and colleagues found the majority of adults with adult depression had a psychiatric diagnosis of depression

prior to age 18. In a sample of community adolescents, the mean age of onset for MDD was between 14 and 15 years of age, and the mean duration of the episode was 26 weeks (Lewinsohn, Clarke, et al., 1994). In a review, Birmaher and colleagues (1996) found the mean length for MDD clinical samples to be 7 to 9 months and 1 to 2 months in community samples. MDD episode duration was found to be longer in individuals with an earlier onset of depression (Lewinsohn, Clarke, et al., 1994). Longitudinal research indicates MDD and DD follow different natural courses, in which DD is characterized by a more chronic disturbance (Stark, Laurent, Livingston, Boswell, & Swearer, 1999). Dysthmic Disorder has an earlier age of onset than MDD, with a mean age of onset of 8.7 years, and has a long clinical course of approximately 3 to 4 years (Kovacs et al., 1994).

MDD is considered a chronic, recurrent condition (Birmaher et al., 1996). The majority of individuals, approximately 60%, who experience MDD can expect to have a second episode (American Psychiatric Association, 2000). For youth, 20% to 60% will experience a second episode within two years of remission from an initial episode of MDD, and within five years 70% will experience a second episode (Birmaher et al.). Recurrence of a depressive episode is comparable for boys and girls (Kovacs et al., 2003), and rates are similar for both clinical and community samples (Dunn & Goodyear, 2006). A small but significant number of youth (18%) have a persistent depression, which is characterized as continued, unabated depression into adulthood (Dunn & Goodyear). Childhood DD is a risk factor for developing MDD and other affective disorders with dysthymic children usually having their first episode of MDD 2 to 3 years after the onset of DD (Kovacs et al., 1994).

In addition to being a recurrent condition, depressive disorders are associated with significant psychosocial impairments (Biederman et al., 1995). Depression in youth is associated with impairments in school and relationships, increased suicidal behaviors, homicidal ideation, early pregnancy, and substance use and abuse (Birmaher et al., 1996). Lewinsohn, Clarke, Seeley, et al. (1994) found adolescents with depression to have more psychosocial impairments when compared to adolescents who have never been depressed, including: suicide attempts, negative body image, low self-esteem, emotional dependence, less effective coping strategies, less social support, and increased tobacco use. Suicidal behavior is considered one of the most significant and devastating associations with depression, which has become more of a concern given that the adolescent rate of suicide has quadrupled since 1950 (American Psychiatric Association, 1998). In addition, these psychosocial impairments have been found to persist even after recovery from depression (Lewinsohn, Clarke, Seeley, et al., 1994). The relation between depression and functional impairment is complex, with a likely reciprocal influence. In other words, depression leads to psychosocial impairment, which then becomes a risk factor for developing depression (Klein, Dougherty, & Olino, 2005).

Gender and Vulnerability

Epidemiological data on depression suggests adult women are approximately twice as likely to be depressed as adult men (Nolen-Hoeksema & Girgus, 1994). Prior to adolescence the rates of depression are similar for boys and girls; however, gender differences begin to emerge around the age of 13 (Hankin & Abramson, 2001). Subsequently, the rates of depression increase for both genders around the age of 15; however, the female rate doubles the prevalence rate for males during this time (Hankin

et al., 1998). This gender difference in depression emerging at 13 appears to be unique to depression (Hankin & Abramson, 2001). Adolescence, therefore, presents as a time of heightened vulnerability for depression in girls. Given the significant shift in prevalence rates, which remain into adulthood, this time period is an essential time for depression research in girls.

A significant amount of research has examined the mechanisms that may contribute to the emerging gender differences in depression, which are generally based upon vulnerability-stress models (Cyranowski et al., 2000; Hankin & Abramson, 2001; Keenan & Hipwell, 2005; Nolen-Hoeksema & Girgus, 1994). Nolen-Hoeksema and Girgus present three models accounting for the emergence of gender differences in depression. Based on the literature, they concluded the model receiving the most empirical support was girls carry more risk factors for depression than boys before adolescence, which contributes to higher rates of depression in the context of social challenges of adolescence. Nolen-Hoeksema and Girgus suggest girls as compared to boys tend to be low on instrumentality, high on ruminative coping, and low on aggressive interpersonal interactions prior to adolescence. These risk factors are thought to make girls more vulnerable to depression when faced with the challenges of adolescence. Cryanowski's et al.'s model also focuses on interpersonal aspects of depressive vulnerability in girls. The authors suggest adolescent girls are particularly challenged by the transition of attachment from parents to peers and romantic partners, which makes them more vulnerable to depression. Both these models suggest the way girls are socialized to relate to others may make them more vulnerable to depression.

Research has also focused on how girls respond to stress and demand. Hankin and Abramson (2001) present a cognitively based transactional theory of depression in girls. Their model suggests girls are more likely to encounter negative life events than boys, which results in greater elevations of negative affect. Girls are also found to experience more interpersonal stress compared to boys, which is associated with depressive symptoms (Rudolph & Hammen, 1999). Not only do girls report experiencing more negative events, they encode the negative events in more emotional details than boys (Hankin & Abramson, 2001). In addition, girls are considered to be more cognitively vulnerable than boys, which enhances the possibility of experiencing depressive symptoms when faced with negative life events, negative affect, or both. Hankin and Abramson (2002) found girls' negative cognitive style accounted for elevated depressive symptoms, supporting their theory that greater levels of cognitive vulnerability could account for gender differences in depression. Additionally, negative cognitions regarding body image are particularly salient for girls and should be considered when examining gender differences in depression. Body image has been found to be particularly important to a girl's self-esteem and a significant correlate of depression in adolescence (Allgood-Merten, Lewinsohn & Hops, 1990). Cole and colleagues (1998) found negative cognitions about physical attractiveness and body satisfaction may be more predictive of depression in girls than boys.

Overall, research has explored various mechanisms through which girls may be more vulnerable to depression than boys. Theoretical models with the most support are based upon a diathesis-stress framework, suggesting that girls have unique vulnerabilities which put them at risk when faced with the challenges of adolescence. Specifically, girls

have been found to have a more ruminative coping style, less instrumentality, more negative cognitions and cognitive style, more interpersonal stress, and more body dissatisfaction compared to boys. Research has shown girls experience more interpersonal stress than boys and are more reactive to the stress (Rudolph & Hammen, 1999). The interaction of these vulnerabilities under more life stress likely contributes to the rise in prevalence of depression in girls during adolescence. These findings suggest the domains of interpersonal functioning, cognition, and life stress are important etiological factors when researching the emergence of depression in early adolescent girls.

Assessment of Depression

Klein and colleagues (2005) suggest three phases for the assessment of depression in children, with the initial phase being the diagnosis and prognosis of the child. The assessor must determine if the child meets criteria for a depressive disorder and must rule out any exclusionary diagnoses, such as bipolar disorder. Key symptoms, comorbid conditions, and functional impairment should also be evaluated. Multiple methods may be used to diagnosis and assess symptoms of depression in children. Common assessment methods include: self-report measures, parent and teacher questionnaires, diagnostic interviews, observational methods, and projective techniques. In research, the most common methods of assessment include self-report measures, such as the Children's Depression Inventory (CDI; Kovacs, 1992), parent and teacher reports, such as the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001), and/or diagnostic interviews, such as the Schedule for Affective Disorders and Schizophrenia for School-Age Children- Present State (K-SADS-IVR; Ambrosini & Dixon, 2000).

Despite high use in research, rating scales do not provide sufficient evidence to diagnosis depression as they typically focus only on current symptoms and behaviors (Klein et al., 2005). Self-report rating scales have been found to have adequate reliability but often lack discriminant validity; therefore, self-report questionnaires should be used for screening purposes only (Stark, 1990). Paper- and- pencil rating scales for depression generally have quick administration and may be given individually or in groups and are often useful for screening purposes. Interviews are used for the purposes of diagnosis and understanding the severity of the disorder (Stark). Interview formats are typically unstructured, semi-structured, or structured (Klein et al.). Unstructured interviews vary from clinician to clinician. Semi-structured interviews are "interviewer based" as the interviewer rates symptoms based on his/her questioning, whereas, structured interviews are "respondent based" as the interviewer's role is limited to reading the questions required. Given that semi-structured interviews rely on more clinical judgment, they are designed to be used with mental health professionals or technicians with extensive training. The use of semi-structured interviews, particularly the K-SADS, is considered to be "best practice" in the assessment of depression (Klein et al.).

In the assessment of depression in children, it is essential to gather information from multiple sources in order to obtain adequate diagnostic information (Jensen et al., 1999; Klein et al., 2005). Both parents and children are considered similar in their ability to provide information needed in order to diagnose depression (Jensen et al.). Clinicians, however, are frequently faced with how to integrate ratings given there is often a low consensus between sources (Achenbach, McConaughy, & Howell, 1987; Jensen et al.). In a meta-analysis exploring the consistency of reports across informants, Achenbach and

colleagues found the average correlation of youth adjustment to be .25 between parent and youth reports. It is believed the child may be the better reporter for subjective symptoms of depression such as sadness, feelings of worthlessness, and anhedonia (Kendall, Cantwell, & Kazdin, 1989). On the other hand, younger children may not be reliable reporters of time-related information, such as the duration and onset of specific symptoms (Stark, Sander, Yancy, Bronik, & Hoke, 2000). Klein and colleagues suggest using a "best estimate" procedure when faced with discrepant data, which includes taking all reports into consideration and using clinical judgment to integrate the data received from these multiple informants. The interviewer may need to utilize his or her clinical judgment to discern whether the child or parent is a more accurate reporter for a particular discrepancy. Jensen et al. argues different interviewees are likely to be better raters under particular circumstances.

Given the limitations regarding discriminant validity with self-report measures of depression and the time-consuming nature of diagnostic interviews, a multiple gate procedure may be used to efficiently and effectively diagnosis youth in a school setting (Reynolds, 1986). Reynolds describes a three step multi-gate procedure used to identify children with depression in order to help reduce false-positives. The first step involves using a self-report measure, such as the CDI, as a screening device. The children who score below the predetermined cut-off are considered not depressed and discontinue participation. Those children who score above the cut-off are then administered the same questionnaire within one week as the second stage of the procedure. Those scoring below the cut-off discontinue participation and those scoring above the cut-off again on the second administration move to the third stage of the screening procedure. The final stage

is the completion of a diagnostic interview, such as the K-SADS. The current study uses a modified version of this multi-gate screening and assessment procedure.

Summary of Depression in Childhood and Adolescence

Depression in childhood is a common, recurrent, and disabling disorder (Kovacs, 2006; Kovacs et al., 1984). The rates of depression steadily increase with age and dramatically increase in adolescence, with up to a sixfold increase from early adolescence to late adolescence (Hankin, 2006). While depression usually remits, psychosocial impairment often persists past recovery and the majority of youth will experience subsequent episodes of depression (Birmaher et al., 1996; Lewinsohn, Clarke, Seeley, et al., 1994). Adolescence presents as a time of heightened vulnerability for depression in girls, since the female rate doubles the prevalence rate for males during this time (Hankin et al., 1998). This trend continues into adulthood, as adult women are approximately twice as likely to be depressed as adult men (Nolen-Hoeksema & Girgus, 1994). Given this trend and impact of depression on females, examining the etiology of depression in early adolescent girls is crucial.

Researchers have proposed several possible explanations for why females have higher rates of depression than males starting in adolescence. Most supported theories are based upon a diathesis-stress framework, suggesting that girls have unique vulnerabilities, which put them at risk when faced with the challenges of adolescence (Cyranowski et al., 2000; Hankin & Abramson, 2001; Nolen-Hoeksema & Girgus, 1994). For instance, girls tend to have a more ruminative coping style, less instrumentality, more negative cognitions, more interpersonal stress, and more body dissatisfaction compared to boys. Research has shown girls experience more interpersonal stress than boys and are

more reactive to the stress (Rudolph & Hammen, 1999). The combination of increased vulnerability and greater stress is thought to contribute to the higher rates of depression in adolescent girls.

Various assessment techniques may be used to assess depression; however, certain "best practices" have emerged in the field of depression research. Self-report questionnaires are commonly used to measure depression in research, but these measures often lack discriminant validity and are recommended for screening purposes only (Stark, 1990). The use of semi-structured interviews, particularly the K-SADS, is considered to be "best practice" in the assessment of depression (Klein et al., 2005). The use of multi-informants is important to obtain adequate diagnostic information. Clinical judgment may need to be used because parent and child ratings are often discrepant (Jensen et al., 1999). Given that diagnostic interviews can be extremely time-consuming and costly, the use of a multi-gate screening procedure is recommended (Reynolds, 1986). Overall, depression in youth is a common and prevalent disorder associated with severe impairment that can persist into adulthood. Further research is needed to better understand how various pathways may contribute to depression in early adolescent girls.

Etiological Pathways to Depression

Given the prevalence and recurrent nature of depression in children and adolescents, it is important to understand how depression develops. Depression is a complex disorder with multiple etiological pathways. Various theoretical models have been presented as possible pathways for the development of depression, including biological, cognitive, behavioral/interpersonal, family, and life stress models (Hammen & Rudolph, 2003). Etiological models of depression should not be adapted from adult

models, but should be developmentally sensitive to children incorporating various environmental and child factors (Cicchetti & Toth, 1998). Most etiological models of depression reflect a vulnerability-stress framework (Hankin, 2006; Stark et al., 1999). Stark and colleagues suggest a combination of biological, cognitive, behavioral, familial, and environmental variables reciprocally interact with each other contributing to the development and maintenance of depression in children. Kendler, Gardner and Prescott (2002) used structural equation modeling to develop an integrative, developmental model of depression for adult women and their findings strongly support theory suggesting that depression is a complex, multifactorial disorder. Therefore, developing multi-dimensional developmental models, which integrate these various theories in research, is important. The pathways relevant to this investigation are explored more in depth.

Biological Influences

Clear evidence from family and twin studies suggests depression runs in families, resulting from both genetic and environmental influences (for a review, see Sullivan, Neale, & Kendler, 2000). Sullivan and colleagues found heritability of major depression ranges from 31% to 42%. Using Virginia Twin Study data, Silberg et al. (1999) found genetics accounted for 28% to 30% of the overall variance in depression for adolescent girls. Children of depressed parents are three times more likely to have a lifetime episode of MDD (Birmaher et al., 1996). Beardslee et al. (1998) estimate a child with a depressed parent has a 40% chance of experiencing an episode of MDD by the age of 20. Genetic transmission is likely to be one explanation for the increased risk for depression in children of depressed parents; however, psychosocial factors are also likely to contribute to the higher risk. Goodman and Gotlib (1999) propose four mechanisms that may

account for the transmission of depression between mothers and their children. The mechanisms include: genetics or heritability of depression, dysfunctional neuroregulatory mechanisms, exposure to negative maternal affect, cognitions and behaviors, and stress and the environmental context of the child's life. The last two mechanisms will be discussed in subsequent sections.

In addition to genetic influences, neuroendrocine regulation, neurotransmitters, brain structure, and biological rhythms have been explored as contributors to childhood depression (Hammen & Rudolph, 2003). The monoamine hypothesis suggests reduced monoamine availability contributes to depression, which focuses primarily on serotonin and noreprinephrine. In essence, depression is thought to result from insufficient activity of monoaminergic neurons (Carlson, 2005). Neurobiological stress reactions have been more recently studied, suggesting depression may result from the over-reaction of the neurobiological stress system, specifically dysfunction in the hypothalamic-pituitary-adrenal (HPA) (Birhamer et al., 1996). Although neurobiological dysfunctional responses to stress have been found in adult populations, these patterns have not been consistent in childhood and adolescent research (Birmaher et al.; Kaufman, Martin, King, & Charney, 2001). Abnormalities in brain structures, particularly in the amygdala and prefrontal cortex, have been associated with depression (Carlson); however, limited research with children has been done in this area (Kaufman et al.). Lastly, dysfunction in sleep behaviors has been established in adult samples with depression; however, the same disturbances have not been reported as consistently in children (Birmaher et al.). Overall, Kaufman and colleagues indicate the neurobiological correlates of depression differ in

childhood, adolescence, and adulthood. As a result, more research is needed to better understand the pathophysiology of depression in childhood and adolescence.

Interpersonal Relationships

Lewinsohn (1974) proposed a social-skills-deficit-model of depression, suggesting an individual who lacks these specific skills will often engage in less social interaction thus leading to the development of depressive symptomatology. A cycle seems to develop consisting of poor social skills leading to interpersonal rejection, which then produces depression and social withdrawal. Research has linked deficits in social competency to depressive symptomatology (Cole, Martin, Powers, & Truglio, 1996). Specifically, social rejection and low interpersonal competence have been associated with higher levels of depression. Additionally, it has been proposed that social competency, as it relates to peers, becomes more important as children get older and is associated more with depression (Cole et al.). Other researchers suggest deficits in social competence to be the result of interpersonal conflict between depressed persons and others in their lives (Coyne et al., 1987). Girls, in particular, are found to experience more interpersonal stress, which is associated with depressive symptomatology (Rudolph & Hammen, 1999). These findings suggest behavioral and interpersonal processes may play a role in the development of depression in children.

Family Influences

Contemporary developmental models of depression emphasize the importance of familial contributions in the etiology of depression (Hammen & Rudolph, 2003). Research indicates multiple family factors are associated with depression in youth, including both parent and family environment variables (Sander & McCarty, 2005). In

their review, Sander and McCarty indicate researchers are shifting their perception of a family's influence on depression as a linear influence (i.e. maternal depression to offspring) to an interactive, multidimensional set of family risk factors. Various aspects of parental psychopathology and the family environment, therefore, are reviewed.

Parental Depression and Anxiety. Having a parent with major depression is one of the strongest predictive factors in childhood and adolescent depression (for a review, see Beardslee et al., 1998). Beardslee et al. estimate a child with a depressed parent has a 40% chance of experiencing an episode of MDD by the age of 20. Diagnosis of depression in children was found to be twice as likely among offspring of mothers with depression as compared to never depressed mothers (Hammen & Brennon, 2003). In addition to increasing the risk of depression, parental depression has been associated with a more malignant course of depression in children (Warner, Weissman, Fendrich, Wickramaratne, & Moreau, 1992; Lieb, Isenee, Hofler, Pfister, & Wittchen, 2002). Furthermore, children of a depressed parent were found to have higher rates of alcoholism and poorer functioning in work, family, and marriage as they age (Weissman et al., 1997). As mentioned earlier, Goodman and Gotlib (1999) propose mechanisms that may account for the transmission of depression between mothers and their children, including genetics, neuroregulation difficulties, exposure to negative maternal affect, cognition and behavior, and stress and the environmental context. The last two proposed mechanisms are highlighted in this section.

The influence of maternal depression on youth depression is found in both parent-child interactions and in the overall context. Miller, Warner, Wickramaratne, and Weissman (1999) found the relation between maternal depression and the risk for

childhood depression was associated with a variety of factors, including maternal depression, low maternal emotional availability, and high maternal control. Similarly, children who are exposed to more parental withdrawal and intrusiveness are more likely to experience depressive symptoms (Langrock, Compas, Keller, Merchant, & Copeland, 2002). Results from Langrock et al.'s study found children who used coping strategies to manage these stressors had less depressive symptoms. Children of depressed parents, however, were not able to utilize coping strategies and were more affected by the stressors. Another study found maternal depression was related to more depressive symptomatology and poorer adaptive functioning in adolescence (Hammen, Shih, Altman, & Brennan, 2003). Results also found adolescents with depressive symptoms who had mothers without depression did not have the same interpersonal difficulties as adolescents with a depressed mother. These studies suggest maternal depression not only contributes to a child's depression, but the child's ability to cope and adaptively function.

The influence of mothers' affect and emotional state on the interpretations of children's behavior has been investigated. Researchers have shown mothers with mental health problems make more internal and global attributions for their child's negative behaviors than mothers without depression (Cornah, Sonuga-Barke, Stevenson, & Thompson, 2003). White and Barrowclough (1998) found depressed mothers more often viewed their child's behavior to have a stable cause. In a study investigating communication patterns between depressed women and their children, researchers found depressed women displayed more critical and negative verbal behavior, engaged in less positive behavior, made more off-task comments, and fewer task-productive comments than nondepressed mothers (Gordon et al., 1989). Researchers indicated the interaction

style of mothers with depression is likely to be explained by their depressed mood and chronic stress. Gordon and colleagues proposed three mechanisms that may affect maternal behavior in depressed women. First, depressed women may have more negative judgments about their child. Second, the mother is likely to have a reduced self-esteem and sense of effectiveness. Lastly, the mothers' affect may reduce her tolerance for dealing with a child's problematic behavior.

Evidence shows parental depression is thought to disrupt family functioning, which then leads to negative child outcomes (Elgar, Mills, McGrath, Waschbusch & Brownridge, 2007; Brennan, Hammen, Katz & Le Brocque, 2002). Elgar and colleagues found the child's rearing environment, characterized by parent rejection and low monitoring, mediated the relation between parent depression and child maladjustment. Research has established that families of depressed parents are characterized by less appropriate parenting patterns, greater family discord, lower cohesion, and higher rates of divorce than are families of nondepressed parents (Beardslee & Wheelock, 1994; Downey & Coyne, 1990). Additionally, maternal depression has been found to be associated with negative cognition in offspring, which is discussed in more detail later. Overall, these studies support Goodman and Gotlib's (1999) proposal that maternal depression influences offspring depression through environmental stress and maternal affect, behavior, and cognitions.

Historically, negative interactions between mothers and children and the presence of depression in mothers have been viewed as primary risk factors for depression in childhood; although, more attention is being focused on the role of fathers in the development of depression (Kane & Garber, 2004). Kane and Garber found fathers'

depression was associated with both youth depression and greater conflict levels at home. Similarly, Alloy et al. (2001) found a father's emotional warmth and acceptance was predictive of the development of depression. Another study found parental depression was associated with a number of offspring psychiatric disorders, including depression (Lieb et al., 2002). When Lieb et al. accounted for parent gender and offspring depression, they found no differences in the results, indicating the importance of both maternal and paternal depression. Brennan and colleagues (2002) found the independent presence of paternal depression or maternal depression was associated with the same increased risk for youth depression as the dual presence of depression in the parents. Although maternal depression has been historically associated with youth depression, recent research suggests paternal depression is clearly associated with child depression as well.

Similar to maternal depression, parental anxiety has also been linked to increased risk of psychopathology in offspring (Klein, Lewinsohn, Rohde, Seeley, & Shankman, 2003; Turner, Beidel, & Costello, 1987). The relation between maternal anxiety and internalizing disorders in offspring is thought to be complex, resulting from both genetic and environmental influences (Swartz et al., 2005). Research indicates comorbidity rates of depression and anxiety in adults is high. In the National Comorbidity Survey, 58% of adults with a lifetime diagnosis of MDD qualified for a lifetime diagnosis of an anxiety disorder (Kessler et al., 1994). Using a "bottom up" method of assessing maternal depression, Swartz and colleagues (2005) found 26% of their sample of mothers met criteria for both an anxiety disorder and depressive disorder. In this study, mothers with depression or anxiety were significantly more likely to have children with an

internalizing disorder. Maternal comorbidity has been found to be a risk factor in early development (Carter, Garrity-Rokous, Chazan-Cohen, Little, & Briggs-Gowan, 2001). Carter et al. found mothers with depression and a comorbid disorder, including anxiety disorders, experienced more disruption in the mother-child dyad than mothers with depression only or the no pathology group. Research linking maternal depression and anxiety to internalizing disorders in offspring as well as the high comorbidity rates between these disorders in women suggests the importance of considering both maternal depression and anxiety when investigating symptoms of depression in youth.

Environmental Factors. In addition to parental depression and anxiety, familial influence on depression is related to environmental factors (Sullivan et al., 2000). The parent-child relationship has been one aspect of the familial environment studied. Attachment theory suggests a child's functioning is impacted by the parent-child relationship (Bowlby, 1980). According to attachment theory, children seek out security during times of stress and a lack of felt security may result in increased distress. Research linking Bowlby's theory to youth depression has been supported in the literature. Shirk, Gudmundsen, and Burwell (2005) found child perceptions of a mother as unavailable, unsupportive, and unresponsive were significantly associated with depressive symptoms in a sample of 168 youth. Maternal availability was found to be an important buffer for depression in times of high stress. In times of low stress, however, maternal availability was less associated with risk for depression. As suggested by Bowlby's attachment theory, adolescents who perceived their parent as less available in times of high stress experienced more depressive symptoms. Similarly, other research has found links between impaired parent-child relationship and depression in adolescents (Bergeron et

al., 2007; Brendgen, Wanner, Morin, & Vitaro, 2005; Milne & Lancaster, 2001; Pavlidis & McCauley, 2001).

Research has linked negative family environments to depression in youth. Family support and conflict have been found to be two particularly important factors associated with depression (Sanders & McCarty, 2005). Family environments associated with depression appear to be characterized by a lack of supportive interactions and high levels of conflict (Sheeber, Hops, & Davis, 2001). Normura and colleagues (2002) found family relationship variables of high discord, low cohesion, and high affectionless control were predictive of general psychopathology, including depression. This finding supports the overall family environment as a predictor of depression in youth. Research has shown a consistent inverse relation between familial support and depression (Lewinsohn, Roberts, Seeley, et al., 1994; Sheeber, Hops, Alpert, Davis, & Andrews, 1997; Stice, Ragan, & Randall, 2004; Windle, 1992). Several studies have also found high family conflict to be associated with depression in youth (Lewinsohn, Roberts, Seeley et al., 1994; Sheeber et al., 1997). Family conflict has been found to be a risk factor in both European American and African American samples (Constantine, 2006; Sagrestano, Paikoff, Holmbeck, & Fredrich, 2003). Herman, Ostrander and Tucker (2007), however, found the association between family environment variables and youth depression to vary by ethnic group. The study found low family cohesion significantly predicted depression for Africans Americans, whereas, high family conflict was predictive of depression for European Americans.

Children with depression compared to those without depression have been found to perceive their family environments as being less supportive, more conflictual, and less

engaged in social and recreational activities (Stark et al., 1990). Using multiple sources and multimethod assessment techniques, Sheeber and Sorensen (1998) examined family environments in adolescents with depression and those without depression. Adolescents with depression reported less family cohesion and fewer sources of social support than the nondistressed comparison group. These findings were supported by both behavioral observations and self-report measures. In addition, adolescents with depression reported more conflict than the comparison group. Aydin and Oztutuncu (2001) found a more cohesive family environment was associated with more positive psychological adjustment, primarily less depression. These findings suggest family environments characterized by less support, higher conflict, and less social engagement are associated with depression in children.

The relation between the family environment and depression has been found prospectively (Lewinsohn, Roberts, Seeley, et al., 1994; Sheeber et al., 1997; Stice et al., 2004; Windle, 1992). For example, in a study of 496 adolescent girls, Stice et al. found deficits in perceived family support predicted future increases in depressive symptoms and onset of MDD. Similarly, Sheeber and colleagues found less supportive and more conflictual family environments were associated with depression in adolescents both concurrently and prospectively over a one-year time span. While the family environment was found to predict depressive symptomatology, depression did not predict the deterioration of the family environment in this sample. Sheeber et al.'s longitudinal findings suggest a causal influence of the family environment on depression. Family conflict has also been found to predict subsequent depression and increases in child depressive symptoms over time (Lewinsohn, Roberts, Seeley et al., 1994; Sagrestano et

al., 2003). Results from these studies provide evidence for the role the family environment plays in the onset and maintenance of depression in youth.

Research examining familial influences and youth depression suggests there may be differential effects by gender. Research has demonstrated the family plays an increasingly significant role in adolescent girls' adjustment while peers play a greater role in boys' adjustment (Kavanagh & Hops, 1994). Furthermore, the relation between maternal depression and depression in daughters, aged 8 to 13, was mediated by pre-existing family adversity (i.e. negative life events, marital conflict, and economic stress), though this relationship was not found in boys (Fergusson, Horwood, & Lynskey, 1995). Girls tend to respond to coercive family patterns in a more passive manner than boys, a behavior that often results in internalizing behaviors for girls in the face of family conflict (Compton, Snyder, Schrepferman, Bank, & Shortt, 2003). These findings suggest it is imperative to take the family environment and relationships into account when conceptualizing depression in early adolescent girls.

Assessment of the Family Environment. Assessment of the family context presents unique challenges (Carlson, 2003). According to Carlson, the primary distinction between assessment of an individual and the family is the complexity of the system, given that families are a multi-member, interdependent, hierarchal system. Most approaches to family assessment include semi-structured or structured interviews, clinical ratings of family interactions, and self-report measures of family functioning. Family assessment methods may be further delineated by the level of analysis they measure: the family, triads, dyads, or individual members. Selecting the method of family measurement should be based on the purpose of the assessment. In research, the primary

goal of family assessment is to be able to quantify family behavior or relationships that are being tested. While it is best to measure multiple units of the family, priority should be given to the measurement of the family unit under practical constraints (Carlson, 1989). The use of a reliable and valid measure based upon theoretical constructs is important when assessing family for research (Carlson, 2003).

For the purposes of this study, a self-report measure is used to assess the family environment. Although both interviews and observational techniques may be useful tools in measuring family environments, self-report questionnaires are more efficient, especially in research. Numerous questionnaires have been developed to assess the family environment; however, some of these measures may not be appropriate to use with children and are generally driven by specific theories of family functioning (Schumm, 2001). For example, the Family Environment Scale (FES, Moos & Moos, 1981) measures the family's social climate; the Family Assessment Measure (FAM, Skinner, Steinhauer, & Santa Barbara, 1983) measures family strengths and weaknesses based on a process model; and the Family Adaptability and Cohesion Scales III (FACES III; Olson, Portner, & Lavee, 1985) measures the family as a system that varies across the dimensions of adaptability and cohesion. These assessment measures are based upon unique theories and conceptualizations of family functioning; however, they also have common scales for cohesion, control, and communication. Bloom (1985) developed the Self-Report Measure of Family Functioning in order to create an improved self-report measure of family functioning by trying to maximize the psychometric robustness and integrate various scales. Bloom's instrument integrates items from the FES, FACES III, FAM, and Family-Concept Q Sort (FCQS; Van der Veen, 1965), resulting in a 75-item measure with three

dimensions (relationship, value, and system maintenance) along with multiple subscales. Bloom's dimensions supported Moos's previous assertion that family functioning can be conceptualized into three major dimensions: relationship, personal growth, and system maintenance.

As suggested by Carlson (2003), a researcher must find a psychometrically sound instrument assessing family functioning domains in order to accurately assess the family environment. In addition, the measure needs to be developmentally appropriate to use with children. The Self-Report Measure of Family Functioning was adapted and revised for use with children and adolescents (Stark et al., 1990). Overall, the Self-Report Measure of Family Functioning – Child Revision (SRMFF-CR) offers a reliable method of assessing youth perspectives on the dimensions of family functioning common across family theories (Stark, 2002). The SRMFF-CR consists of six subscales: Communication, Conflict, Social/Recreational Orientation, Cohesion, Laissez-Faire Style, and Authoritarian Style. This child-appropriate instrument is utilized to assess the family environment in the present investigation.

Summary of Family Influences. In general, research indicates multiple family factors are associated with depression in youth (Sander & McCarty, 2005). Environmental and biological influences of the family are important factors to consider when conceptualizing the etiology of depression in children and adolescents. Having a depressed parent is clearly associated with childhood depression and can be considered one of the strongest predictive factors in childhood and adolescent depression (Beardslee et al., 1998). It is difficult, however, to determine the relative effects of the genetic transmission of depression from the psychosocial effects found in families with a

depressed parent (Goodman & Gotlib, 1999). In addition to biological mechanisms, Goodman and Gotlib propose maternal depression may influence offspring depression through environmental stress and maternal affect, behavior, and cognitions. Additionally, research has shown depression in adults is associated with comorbid anxiety (Kessler et al., 2004). Maternal comorbid depression and anxiety is suggested to be a risk factor in early development (Carter et al., 2001), and is associated with internalizing disorders in offspring (Klein et al., 2003).

In addition to parental depression and anxiety, familial influence on depression is also related to environmental influences (Sullivan et al., 2000). Research has found depressed children have families that are less cohesive, more controlled and conflictual, communicate less, express emotion more intensely, and engage in fewer social and recreational activities (Barerra & Garrison-Jones, 1992; Cole & McPherson, 1993; Jewell & Stark, 2003; Ostrander et al., 1998; Stark et al., 1990). The relation between the family environment and depression has been found prospectively (Lewinsohn, Roberts, Seeley, et al., 1994; Sheeber et al., 1997; Stice et al., 2004; Windle, 1992) demonstrating the family environment influences the onset and maintenance of depression in youth. Research suggests the family environment and relationships may have an even more critical role in the development of depression in girls; therefore, it is important address familial variables when conceptualizing childhood depression in girls. Maternal depression and anxiety as well as other family environment variables are examined to determine their relation to depression in this study.

Assessing the family context presents unique challenges, and the method of family measurement should be based on the purpose of the assessment (Carlson, 2003).

Self-report questionnaires are an efficient tool to use in research to assess the family environment. As suggested by Carlson, a researcher must find a psychometrically sound instrument that is developmentally appropriate to use with children. The SRMFF-CR offers a reliable method of assessing youth perspectives on the dimensions of family functioning common across family theories (Stark, 2002) and is utilized in this study.

Cognitive Diathesis-Stress Theories

The major cognitive models (Abramson, Metalsky, & Alloy, 1989; Abramson, Seligman, & Teasdale, 1978; Beck, 1967) are stress-diathesis perspectives in which cognitive vulnerability is believed to interact with stressful life events to produce depression. Cognitive theorists highlight the role of negative or maladaptive beliefs have in the development of depression. Two of the major cognitive theories of depression, the hopelessness theory (Abramson et al., 1989; Alloy, Abramson, Metalsky, & Hartlage, 1988) and Beck's cognitive theory (Beck, 1967, 1972), are vulnerability-stress theories that attempt to understand individual differences in response to stress in terms of maladaptive cognitive patterns. Both theories suggest specific negative cognitive styles increase an individual's likelihood of developing episodes of depression (Abramson et al., 1989; Beck, 1967). Beck's information processing model indicates individuals engage in errors in thinking, exhibit negative cognitive schemas, and possess negative thoughts about the self, world and future, which are associated with depression. Abramson's (1978) model suggests individuals may possess a "depressive attributional style," which is a predisposition to attribute negative events as internal, global, and stable. Both of these theories suggest people who possess depressive cognitive styles are more vulnerable to depression because they tend to formulate interpretations of their

experiences that have negative implications for themselves and their futures (Abramson et al., 1989; Beck, 1967). These theories are extremely similar in their presentation of cognitive vulnerability as a pathway of depression and both models have been found to overlap and significantly predict depression in research (Hankin, Abramson, Miller, & Haeffel, 2004). This study defines cognitive vulnerability to depression via Beck's model in order to test hypotheses using one theoretical model.

Beck's Theory of Depression. Beck's information-processing model of depression suggests negative events activate maladaptive schema, which results in a cognitive disturbance (Beck, 1976). The schema then serves as a filter to process information from the environment. The information processed, however, is usually distorted and leads to negative cognitions. These negative cognitions, in turn, lead to depressive symptoms. Thus, there are three aspects of cognitive functioning in Beck's theory (Beck et al., 1979). One, depressed individuals experience errors in their thinking. Thus, these individuals typically have an inaccurate interpretation of a situation leading to an "automatic thought." These automatic thoughts usually relate back to a person's negative core schema, which are an individual's central rules of life. Depressed individuals are thought to have schemas associated with being inadequate, unlovable, and unacceptable. The second aspect, the self-schema or core belief, therefore, guides the information processing. Depressed individuals are likely to encode, store, and retrieve information related to their negative self-relevant information. Prieto, Cole, and Tageson (1992) found self-schemas in children were associated with the attention, encoding, and retrieval of information, supporting Beck's theory. Beck's theory argues people with negative self-schemas consequently possess a cognitive vulnerability to depression (Beck, 1967, 1972,

1987). In other words, individuals who interpret events through this negative lens are more likely to experience depression when encountering stress.

The third aspect of Beck's model is the negative cognitive triad, or having a negative perception of the self, world, and future. When individuals encounter negative life events they are hypothesized to develop negatively biased views of the self (low self-esteem), world, and future (hopelessness) and, ultimately, depressive symptoms (Beck, 1967). The first component of the triad, view of self, is reflective of perceiving the self in a negative way. The second component, view of the world, suggests an individual perceives the world as full of burdens, obstacles, or traumatic situations. The last component consists of viewing the future in a negative way. Individuals may believe their current difficulties will never get better and they will continue to suffer (Beck).

Support for Beck's Theory. Extensive evidence supports Beck's cognitive vulnerability theory to depression. Depressogenic cognitions and/or automatic thoughts are associated with an increase in depressive symptoms in multiple studies (e.g. Abela & D'Allesandro, 2002; Hankin et al., 2004; Joiner, Metalsky, Lew, & Klocek, 1999; Kendall et al., 1990; McDermut, Haaga, & Bilck, 1997; Stark, Schmidt, & Joiner, 1996). Depressogenic cognitions have been shown to be related to depressive symptoms in various populations, including adults (Jolly, Dyck, Kramer, & Wherry, 1994; Lewinsohn, Hoberman & Rosenbaum, 1988), adolescents (Garber et al., 1993; Jolly, 1993), and children (Epkins, 1996; Kendall et al., 1990; Stark et al., 1996).

A few studies have examined the cognitive diathesis-stress model of depression in children and adolescents, producing mixed results. Cole and Turner (1993) examined negative attributional style, cognitive errors, life events, peer-nominated competence, and

depression in 356 fourth, sixth, and eighth graders. Results generally did not support that attributional style moderated the relation between life events and depression. Weak support was found for cognitive errors moderating the relation between life events and depression. Findings from the study, however, supported a theoretical model in which attributional style and cognitive errors mediated the relation between life stress and depression. This mediational model will be examined more in depth in the following section. The Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) Project utilized a prospective behavioral high-risk design testing the cognitive vulnerability hypotheses of depression (Alloy et al., 1999). The CVD project started with a large sample of first year college students (*N*=5,378) with no current Axis I disorder. Two groups emerged from this sample, a high-risk group (*n*=173) and a low risk group (*n*=176) based on their cognitive risk scores. These two groups were monitored over the next 3 years. Findings from the CVD Project supported cognitive vulnerability hypotheses. Cognitively high-risk participants were significantly more likely than cognitively low-risk participants to develop first onsets and recurrences of episodes of depressive disorders during the first two-and-a half years of follow-up (Alloy, Abramson, & Hogan, 2000). In contrast, there were no risk differences in the development of anxiety disorders during the prospective follow-up period. These results indicate that depressogenic cognitions present specific risk for first onsets of depression. Lastly, Lewinsohn and colleagues (2001) also examined cognitive vulnerability in a longitudinal design with 1,507 adolescents. Results found evidence for the Beckian version of the diathesis-stress hypothesis. Dysfunctional attitudes, therefore, were found to be a risk

factor for adolescent depression under times of stress, even when controlling for other variables.

Mediational Model of Life Stress and Cognition. Cognitive diathesis-stress models of depression represent a moderational model, suggesting stress alone does not evoke depression but is influenced by cognition (Cole & Turner, 1993; see upper panel of Figure 1 for a general form). Beck's model, for instance, suggests cognitions are stable and in latent form, which are triggered by life events in depressive-prone individuals. Given most cognitive origins are believed to develop in childhood (Beck, 1972), the use of moderational models in childhood is questionable (Cole & Turner). Research indicates cognitive vulnerability may not interact with stress to predict depression in younger ages (Abela, 2001), but cognitive variables and stress have direct effects on depression in children (Ostrander et al., 1998). A child's self-schema is believed to be less structuralized than adolescents or adults. Cole, Jacquez and Maschman's (2001) research supported this theory, finding stability of a child's self-appraisal increased from 3rd to 6th grade, suggesting self-appraisals begin to solidify into relatively stable constructs of the one's self concept as children age. The use of a mediational model suggests a child may internalize life stress and develop negative self-schemata (see lower panel of Figure 1 for a general form). In other words, adverse environments or events may be internalized as negative cognitions, which then contribute to the development of depression. Instead of interacting with stress, as proposed by moderational models, depressogenic cognitions would arise from the stress. Comparisons of mediational models and moderational models are relatively few with youth; however, preliminary studies have found the support for a mediational pathway (Hammen & Rudolph, 2003).

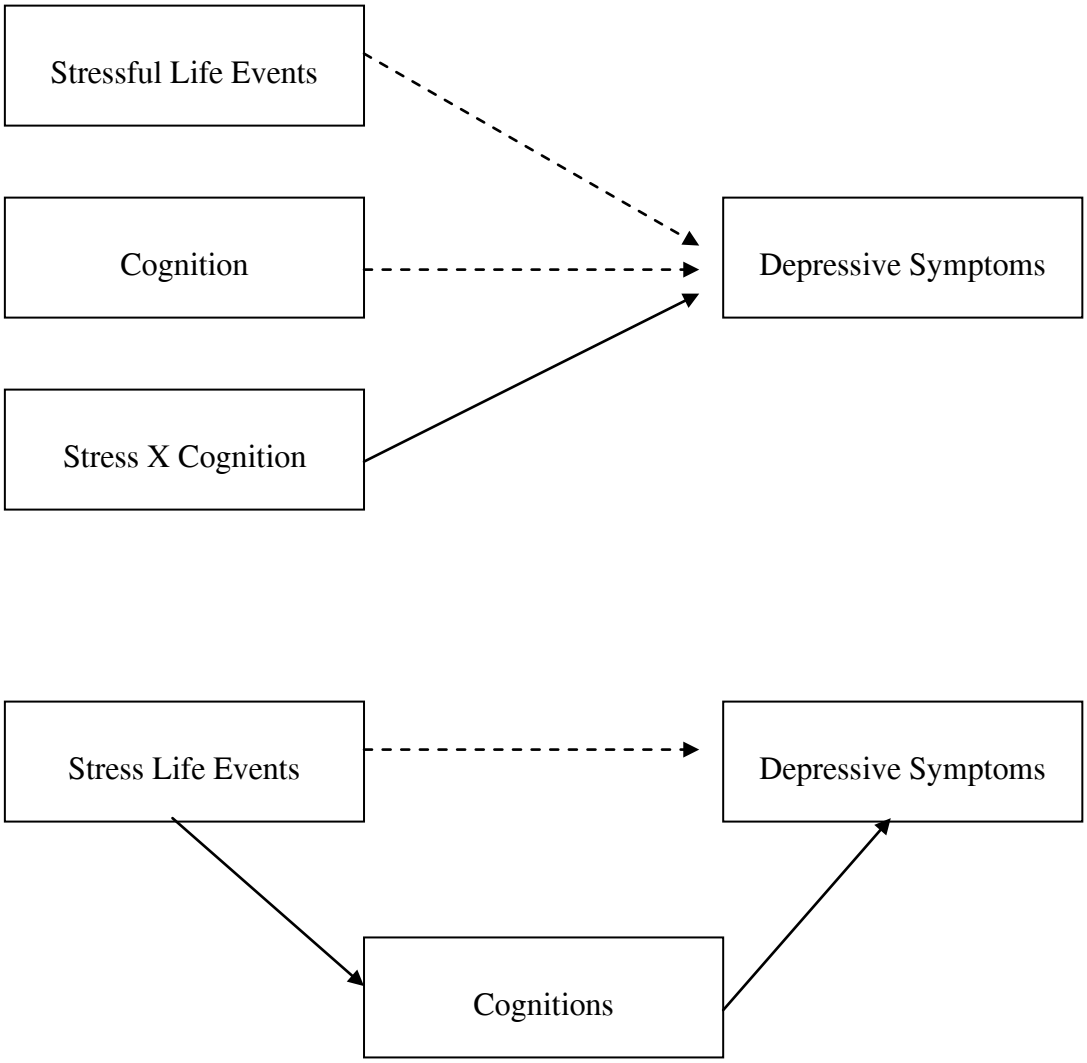


Figure 1. A schematic diagram of general forms of cognitive moderational and mediational models of depression

As mentioned previously, Cole and Turner (1993) examined the relation between attributional style, cognitive errors, peer-related competencies, and life events in 356 elementary and middle school age children. Using structural equation modeling, Cole and Turner found support for the mediation between cognitive variables and depression, but

only weak support for a moderational model with negative life events and cognitive errors. Specifically, results indicated attributional style and the tendency to make cognitive errors mediated the relation between peer-rated competencies and depression. Similarly, the relation between positive and negative events and depression was partially mediated by the cognitive variables. Overall, these findings show that adverse environmental and interpersonal factors may influence cognition, which in turn may influence depression. Tram and Cole (2000) utilized a longitudinal design to examine the relation between negative events, self-perceived competency, and depression in 468 ninth graders. The authors found relatively little support for a moderational model of these factors, but found a significant partial mediational model. Results indicated the relation between negative events and changes in depression was partially mediated by self-perceived competence. In other words, negative events influenced self-perceived competence, which was associated with greater depression. Reinemann and Teeter Ellison (2004) examined whether cognitive variables mediated or moderated the relation between negative life events and depressive symptoms in 6th graders. Results provided primary support for the mediational model, finding cognitive variables mediated the relation between negative life events and reports of negative mood, ineffectiveness, and anhedonia on the Children's Depression Inventory. These studies indicate cognitive diathesis may not emerge until adolescence or adulthood and a mediational model may be more applicable to children (Reinemann & Ellison; Tram & Cole).

Summary of Pathways and Need for Integrative Models of Depression

Given the large number of processes, mechanisms, and risk factors associated with the etiology of depression, an integrative model evaluating these factors

simultaneously is needed to understand the development of depression in early adolescence (Hankin, 2006). The research reviewed in this section emphasizes the importance of considering family influences, biological factors, interpersonal relationships, cognitive vulnerability, and life stress when studying the onset and maintenance of childhood depression. Research shows depression tends to run in families; however, it is difficult to determine the relative effects of the genetic transmission of depression from the psychosocial effects common to families with a depressed parent (Goodman & Gotlib, 1999). Nevertheless, having a depressed parent is clearly associated with childhood depression and can be considered one of the strongest predictive factors in childhood and adolescent depression (Beardslee et al., 1998). Research has found depressed children have families that are less cohesive, more controlled and conflictual, communicate less, express emotion more intensely, and engage in fewer social and recreational activities (Barrera & Garrison-Jones, 1992; Cole & McPherson, 1993; Jewell & Stark, 2003; Ostrander et al., 1998; Stark et al., 1990). Thus, research indicates multiple family factors are associated with depression in youth, including both parent and family environment factors (Sander & McCarty, 2005).

In addition to familial influences, cognitive models of depression have been widely researched. Cognitive diathesis-stress models of depression are theories that attempt to explain variability in response to stress. These theories suggest depression is the result of the interaction between personal cognitive vulnerability and stress. Beck's theory (1967) focuses on the schemas of individuals, suggesting people who possess depressogenic cognitions are at increased vulnerability to depression when faced with life stress. An important part of the theory suggests cognitions are often defined as distorted

negative perceptions of the self, world, and future (cognitive triad). Several studies have supported Beck's theory of cognitive vulnerability to depression in children and adolescents (Alloy et al., 1999; Epkins, 1996; Garber et al., 1993; Jolly, 1993; Kendall et al., 1990; Lewinsohn et al., 2001). Some research, however, suggests an interaction between cognitive vulnerability and stress may not be applicable to children given that cognitive origins are thought to develop in childhood (Cole & Turner, 1993). Research indicates cognitive diathesis may not emerge until adolescence or adulthood; therefore, a mediational model may be more appropriate when studying the relation between stress, cognitive vulnerability, and depression (Tram & Cole, 2000). Overall, further research is needed focusing on complex models of childhood depression evaluating individual, family, and environmental factors simultaneously to help further delineate these complex pathways.

Origins of Depressogenic Cognitions

As addressed in the previous section, depressogenic cognitions present specific risk for the onset of depression (Alloy et al., 1999). Despite the strong association between cognitive vulnerability and depression, research has just recently started to better understand how a person's depressive schemas develop. Beck and colleagues (1979) hypothesize depressive schemata are formed through early learning experiences, especially those within the family. As suggested by Cole and Turner's (1993) research, a child's self-schema is not as structuralized as adults; therefore, the self-schema may be shaped by life experiences. Thus, a child's self-schema is not only affecting how they process information, it is being simultaneously shaped by the experience as well. Through repeated learning experiences, a child's self-schema becomes more

structuralized over time. Children exposed to chronic stress or unhealthy environments, will integrate these experiences into their self-schema. Various aspects of the child's environment, therefore, may contribute to the development of the self-schema. In middle childhood (ages 8 to 12), children develop the ability to conceptualize and make judgments about their self worth (Garber & Flynn, 1998). During this time, other people's opinions and interactions become salient and are integrated into their self-concept. The age of this sample, 9 to 14, seems to be a critical age at which children may be developing their self-schemas. The following section, therefore, addresses possible developmental origins of the cognitive triad.

Negative Life Events

Negative cognitive schemas associated with depression are hypothesized to develop in response to stressful events in childhood (Beck, 1967). Negative life events, therefore, are thought to play a crucial role in the onset of depression. Negative events have been found to have long-term, short-term, and rapid effects on the onset of psychiatric disorders in children and adolescents (Goodyear, Kolvin, & Gatzanis, 1987). In addition, depressive disorders have been found to be associated with a broad range of adverse events (Tiet et al., 2001). In longitudinal studies, negative life events have been shown to predict depression in adolescence and adulthood. In a study of 9th graders, Tram and Cole (2000) found life events assessed during the fall semester predicted depression in the spring semester. Studies have shown adolescent life events increase the risk of having depression into adulthood (Franko et al., 2004; Pine, Cohen, Johnson, & Brook, 2002). In a five-year longitudinal study, negative life events were found to predict depression in young children and continued to predict depression across 5 years (Nolen-

Hoekesma, Girgus, & Seligman, 1992). The direct effect of negative life events on depression was found even when controlling for previous depression. These studies indicate adverse life events are associated with the development of depression.

In addition to influencing depression, negative life events have been shown to directly impact cognitive vulnerability in children. Cole and Turner (1993) found life events had a direct effect on attributional style and cognitive errors in a sample of elementary and middle school children. Similarly, negative life events predicted changes in self-perceived competence in ninth graders (Tram & Cole, 2000). In a sample of 5th and 6th graders, Rudolph et al. (2001) found recent stress contributed to deficits in perception of control and feelings of helplessness, which were directly associated with increased depressive symptoms. In a longitudinal study, negative life events were found to predict depressive attributional style one year later, even when controlling for prior cognitive style and maternal depression (Garber & Flynn, 2001). Reinemann and Teeter Ellison (2004) examined the relation between stressful life events and the cognitive triad in middle school children. Results suggested stressful life events were significantly associated with the children's cognitive triad, which partially mediated the relation between life stress and depressive symptoms. Greenberg, Sander, and Stark (2008) found negative life events had a direct effect on the cognitive triad and an indirect effect on depressive severity via the cognitive triad using data from the sample of early adolescent girls currently being studied. When conceptualizing the impact of negative life events on depression in children, these findings indicate negative life events likely have an indirect effect on depression via cognitive vulnerability of the child.

Familial Influences

Stark and colleagues (1999) propose a cognitive interpersonal pathway to depression in which core schema develop as a result of early learning experiences and communication within the family. Within the family milieu, children develop attachment behaviors, interpersonal skills, and expectations of interpersonal relationships. Thus, various aspects of the family environment may be important in a child's schema development, including parental depression, parent cognitions, parenting practices and family climate. Alloy and colleagues (2001) suggest children may develop these depressogenic cognitions through modeling parents' negative cognitive styles, direct learning from the parents inferential feedback (i.e. the causes and consequences of negative events), and indirect learning from negative parenting practices.

As previously discussed, parental depression is a strong predictor of childhood depression and has been postulated as a potential avenue through which children develop negative cognitions. Garber and Flynn (2001) found a significant relation between mothers' depression and low self-worth, depressive attributional style, and hopelessness in their children. These results indicate that exposure to a depressed mother may be one process by which negative cognitions develop in children. Furthermore, Garber and Flynn also examined other possible mechanisms associated with negative cognitions in children, including maternal cognitive style. Children's attributional style was found to mirror the mother's attributional style for child related events. Similarly, Alloy et al. (2001) found students' and parents' reports of high cognitive risk participants to have more stable, global attributional feedback for negative events in the child's life compared to participants' with low cognitive risk. Other research has specifically examined the

relation between youth depression, parents' cognitive triad, and youth cognitive triad (Stark et al., 1996). Results of Stark et al.'s study found the parents' view of the self, world and future was related to their children's view of the self, world and future, which in turn was related to depressive symptoms; however, findings were only significant for the mothers' cognitive triad. In addition, the children's cognitive triad fully mediated the relation between perceived parental messages about the self, world, and future and depressive symptoms. Results of this study suggest parental messages influence a child's depressogenic thinking, which ultimately leads to depression. These findings indicate children may directly learn maladaptive cognitions from their parents.

Negative parenting characteristics have also been associated with the development of childhood cognitive vulnerability. Lower levels of maternal acceptance (Garber & Flynn, 2001; Rudolph et al., 2001) and higher levels of rejection (Rudolph et al., 2001) have been associated with negative cognitions in adolescents. One longitudinal study examined 289 children from infancy to 5th grade and found negative parenting, characterized by high anger expression and negative inferential feedback, interacted with negative life events to predict child cognitive vulnerability (Mezulis et al., 2006). Results found the relation between maternal negative parenting and child cognitive style to be stronger than paternal negative parenting. Rose and colleagues (1994) found childhood negative parent child interactions were significantly associated with negative cognitions in a sample of psychiatric adults. Rose et al. suggested negative cognitions in the sample developed in response to a negative interaction style with parents, which were characterized by harsh, critical or overcontrolling interactions. For example, if a daughter is ridiculed for every mistake she makes, she may develop a negative belief that she can

never do anything right no matter how hard she tries. A study examining the relation between the family environment and negative thoughts in over 300 adolescents found a more cohesive family environment was associated with fewer negative thoughts (Aydin & Oztutuncu, 2001). Using data from the CVD project, Gibb and colleagues (2001) found further support for Beck's theory that schemas develop from negative childhood experiences. Participants were placed into a high risk or low risk group based on their cognitive vulnerability. Results of the study found cognitive risk fully mediated the relation between childhood emotional maltreatment and major depression. Childhood emotional maltreatment was defined as humiliation, rejection, extortion, and teasing in the family environment. Using data from the sample of early adolescent girls being currently studied, Greenberg et al. (2008) found family variables of social/recreational activities, conflict, and communication had direct effects on the girls' cognitive triad. In addition, there were indirect effects on depressive severity via the cognitive triad for family social/recreational activities and family conflict. Overall, Ingram (2003) suggests research supports the idea that cognitive variables mediate the relation between troublesome parent-child interactions and depression. As proposed by Alloy et al. (2001) children may also experience indirect learning of negative cognitions through negative parenting and an unsupportive family environment. Based upon the research, it seems clear that the family plays a role in the development of a child's cognitive vulnerability to depression.

Assessment of Depressogenic Cognitions

For research purposes, self-report questionnaires have typically been used to assess cognitive vulnerability in youth. Numerous questionnaires have been developed to

assess depressogenic cognition based upon Beck's cognitive theory of depression (Beck, 1967; Beck et al, 1976) and the attributional/hopelessness theory of depression (Abramson et al., 1978; Alloy et al., 1988). Considering this study is assessing depressogenic cognition based upon Beck's model of depression, only relevant cognitive measures to Beck's theory will be discussed.

The Children's Negative Cognitive Errors Questionnaire (CNEQ; Leitenberg, Yost & Carroll-Wilson, 1986) was designed to assess 4 types of cognitive errors outlined in Beck's theory (Beck, 1967), including: (1) catastrophizing, (2) overgeneralizing, (3) personalizing, (4) selective abstraction. The questionnaire has 24 items with hypothetical scenarios and a cognitive error. Respondents are to rate how likely they are to think like the item presented. This measure has demonstrated moderate test-retest reliability and acceptable internal consistency (Leitenberg et al., 1986). The Dysfunctional Attitude Scale (DAS; Weissman, 1979) is a measure based upon Beck's theory related to dysfunctional core beliefs and attitudes. The measure has two parallel forms assessing an individual's core beliefs on a 7-point scale. The DAS was designed to be used with adults but has been used with adolescent populations, aged 11-18, and has demonstrated good psychometric properties with this age group (Ingram, Nelson, Setidtmann, & Bistricky, 2007). The Negative Affect Self-Statement Questionnaire (NASSQ; Ronan, Kendall, & Rowe, 1994) corresponds to the Automatic Thought Questionnaire-Negative (ATQ-N; Hollon & Kendall, 1980) in adults. This measure evaluates the frequency of self-statements associated with negative affect in children and adolescents, aged 11 to 15. The measure provides anxiety specific, depression specific, and negative affect specific self-

statements. The NASSQ has been shown to demonstrate acceptable validity and reliability.

Lastly, the Cognitive Triad Inventory-Children (CTI-C; Kaslow, Stark, Printz, Livingston, & Tsai, 1992) was developed to assess Beck's cognitive triad in children. This 36-item questionnaire is considered a psychometrically validated instrument that can be completed in a relatively short amount of time (Kaslow et al., 1992). The measure has 12 item subscales that reflect the child's view of the self, world and future. Given that the cognitive triad is a central component of Beck's theory of depression (Beck, 1967), the current study utilizes the CTI-C to assess depressogenic cognitions.

Summary of Origins of Depressogenic Cognitions

Ingram (2003) suggests cognition is the "final pathway," holding together the vulnerability process to depression in children. As a result, understanding the origins of cognitive vulnerability to depression has important implications for the prevention and treatment of depression. Research is lending support to theorists suggesting negative schemas constituting an increased vulnerability to depression are primarily learned via childhood experiences. The literature presents multiple pathways associated with the development of depressogenic cognition.

Garber and Flynn (2001) suggest the experience of negative life events is likely one of most important factors underlying the development of cognitive vulnerability. Multiple studies have shown a direct relation between negative life events and depressogenic cognition (Cole & Turner, 1993; Garber & Flynn; Greenberg et al., 2008; Rienemann & Teeter Ellison, 2004; Rudolph et al., 2001; Tram & Cole, 2001). The findings from these studies suggest negative life events not only have a direct effect on

depression in childhood but are also a key factor in the developmental origins of maladaptive cognitions. In addition to negative life events, theorists have hypothesized depressive schemata are formed through early learning experiences within the family (Beck et al., 1979). Researchers have shown children develop depressogenic cognitions via their parents through modeling, direct learning from the parents' maladaptive cognitions, or indirect learning through the family environment. Research suggests exposure to a depressed mother may be one process by which negative cognitions develop in children (Garber & Flynn, 2001). Children's maladaptive cognitions, both attributional style and cognitive triad, have been significantly associated with parental cognitive style or triad (Alloy et al., 2001; Garber & Flynn, 2001; Stark et al., 1996). Lastly, the family environment has been another avenue through which children may develop maladaptive cognitions. Lower levels of maternal acceptance (Garber & Flynn, 2001; Rudolph et al., 2001) and higher levels of rejection (Rudolph et al., 2001) have been associated with negative cognitions in adolescents. Negative parenting, often characterized by harsh, critical, and rejecting environments, has been associated with maladaptive cognitions in several studies (Gibb et al., 2001; Mezulis et al., 2006; Rose et al., 1994). Overall, negative life events, parental depression, and a negative family environment are all possible contributors of depressogenic cognition in children.

Depression and Comorbidity

Comorbidity in psychology is recognized as the co-occurrence of two or more truly separate and independent psychiatric disorders (Caron & Rutter, 1991). The overall rates of psychiatric comorbidity in the general population are considered high and are associated with a more serious course of illness (Kessler et al., 1994). The observed co-

occurrence of child psychiatric disorders far exceeds chance alone (Caron & Rutter), and the presence of depression in children and adolescents increases the chance of having another disorder around 20-fold but can increase the likelihood up to 100-fold (Angold & Costello, 1993). Approximately 40% to 70% of depressed children and adolescents have a psychiatric comorbid disorder and 20% to 50% have two or more psychiatric comorbid disorders (Birmaher et al., 1996). A meta-analysis of comorbidity in community samples found depression in youth is most commonly associated with anxiety disorders, conduct disorder/oppositional defiant disorders, and attention deficit hyperactivity disorders (Angold et al., 1999). The likelihood of having a comorbid disorder with depression is considerably higher than the likelihood of depression being comorbid with another disorder (Angold & Costello). Rates of comorbidity are especially high in clinically referred samples. In a clinical sample of youth diagnosed with depression, Biederman and colleagues (1995) found 95% had at least one comorbid disorder and 80% had two or more comorbid disorders, which were not accounted for by symptom overlap. In addition, rates of comorbidity have been reported to be higher in children and adolescents than in adults (Rohde, Lewinsohn, & Seeley, 1991).

Research suggests “pure” depression is often less common than depression with a comorbid disorder (Avenevoli et al., 2001), and comorbidity should be considered the rule rather than the exception in childhood depression (Biederman et al., 1995). Comorbid diagnoses with depression generally have been found to precede the onset of depression (Biederman et al.; Birmaher et al., 1996). These findings suggest the presence of psychiatric comorbidity in youth with depression is considered high and there is a need

to better understand of how comorbidity may influence the etiological pathways of depression.

Implications of Comorbidity

The presence of a psychiatric comorbid disorder with depression is associated with a number of negative outcomes including: increased risk for recurrent depression, longer duration of an episode, more suicide attempts, greater functional impairment, and lower response to treatment (Birmaher et al., 1996). Research indicates adolescents with depression and a comorbid psychiatric disorder are more at risk for suicidal ideation and attempts (Lewinsohn, Rohde, & Seeley, 1995; Rohde et al., 1991). In fact, the presence of a second psychiatric disorder substantially increases the rate for suicide attempts in adolescents (Lewinsohn et al., 1995). The likelihood of receiving treatment increases as the number of comorbid disorders increases; however, specific disorders have not been found to affect treatment utilization. Academic problems, poor overall functioning, and conflict with parents are also outcomes impacted by comorbidity. The effects of comorbidity are similar for both boys and girls (Lewinsohn et al.). Research findings, therefore, indicate comorbidity is a common problem for depressed children and is associated with more significant functional impairment and a more detrimental course of depression.

Theoretical Perspectives of Comorbidity

The study of comorbidity has important implications in the field of psychological research. There are multiple indicators for the study of comorbidity including: nosology implications, implications for theories of etiology, clinical issues such as course and treatment of the disorder, and erroneous assumptions regarding a particular disorder

(Angold & Costello, 1993; Caron & Rutter, 1991; Lewinsohn et al., 1995; Klein & Riso, 1993). Comorbidity has been defined and presented in multiple ways in the literature; therefore, it is helpful to present some terminology relevant to this topic. Two important variables to differentiate when referring to comorbidity are the diagnostic grouping and temporal relation. Angold and colleagues (1999) define "homotypic comorbidity" as the comorbidity between disorders within one diagnostic grouping, such as DD and MDD. On the other hand, "heterotypic comorbidity" refers to comorbidity between disorders of different diagnostic groupings. This study focuses on the heterotypic comorbidity of depression and anxiety. Regarding temporal relations, "concurrent comorbidity" is when two disorders are present at the same time, whereas, "successive comorbidity" is when the presentation of disorders do not overlap in time (Angold et al.). This study assesses concurrent comorbidity.

Multiple explanations have been presented to account for the comorbidity among psychiatric disorders and are generally divided into four groups, including: (1) methodological artifacts; (2) artifacts of diagnostic criteria; (3) drawing boundaries in wrong places; (4) substantive causes (Angold et al., 1999; Klein & Riso, 1993). The first explanation proposes comorbidity is a simple artifact rather than a real phenomenon, which may be the result of sampling bias, halo effects, or collection strategies (Angold et al.). Studies using clinical populations are likely to overestimate rates of comorbidity because the likelihood of receiving treatment is higher in individuals with multiple disorders (Klein & Riso). Angold and colleagues, however, found that sampling bias does not account for the rates of comorbidity given the high rates of comorbidity in the general population and across all levels of symptom severity. Halo effects suggest comorbidity

may be the result of rater biases in information collecting and decision-making; however, the use of structured assessment in research and self-report measures with both parents and children rule out clinician bias as accounting for comorbidity rates. Angold and colleagues also suggest comorbidity is not the result of multiple informants because comorbidity is found in single informant situations and self-reports. In summary, research has demonstrated that comorbidity cannot be fully accounted for by methodological flaws or bias (Angold et al.)

Comorbidity is also commonly explained as being an artifact of diagnostic criteria (Angold et al., 1999; Caron & Rutter, 1991; Klein & Riso, 1993). These authors indicate symptoms may overlap between existing diagnostic criteria. For instance, sleep disturbance is a diagnostic criterion for both depressive and some anxiety disorders. The overlapping of symptoms, therefore, contributes to the high rates of comorbidity between the groups. Research, however, is beginning to show comorbidity is still present even when these overlapping symptoms are controlled. Seligman and Ollendick (1998) contend the empirical evidence does not support that symptom overlap between depression and anxiety accounts for the comorbidity between the two disorders; however, some symptom overlap may result in partial presence of the other disorder. Biederman and colleagues (1995) found comorbidity in a clinically referred sample was not due to symptom overlap. After controlling for comorbid symptomatology, the majority of participants maintained a diagnosis of depression. The results of these and other studies suggest comorbidity cannot solely be an artifact of overlapping symptoms (Angold et al.).

Another issue related to diagnostic criteria is problems with the nosology at the conceptual level. In other words, the boundaries drawn between disorders are inaccurate.

Klein and Riso (1993) suggest multiple ways the boundaries may be incorrect. For instance, a comorbid condition may be an atypical form of one disorder, distinct from the other disorder, or may represent atypical forms of both pure disorders. The comorbid condition may be a third disorder, independent from the pure forms. Lastly, the comorbid condition may be a different manifestation or phase of the same disorder. The extent to which diagnostic considerations contribute to comorbidity remains unclear; however, it is reasonable to assume based up on methodological and nosological considerations that comorbidity is a real phenomenon (Angold et al., 1999). Given that comorbidity is not simply a methodological artifact, researchers turn to substantive causes in order to explain why comorbidity is so common.

Substantive theories attempt to explain why two distinct disorders would co-occur together so frequently. There are two main substantive explanations of comorbidity based upon etiological relations (Klein & Riso, 1993). The first explanation suggests one disorder is a risk factor for the other disorder. For example, disorder X may cause or predispose an individual to disorder Y or vice versa. The second explanation suggests the two disorders develop due to overlapping etiological processes. Thus, each disorder has both shared and unique risk factors, which results in a high probability of co-occurrence. Common risk factors may likely account for rates of comorbidity given that most disorders are multifactorial in origin and many risk factors are not origin specific (Caron & Rutter, 1991). Even though disorders may not share the exact same risk factors, the risk factors may be associated or correlated with each other. Angold et al. (1999) indicate shared risk factors are an important component in the causation of comorbidity. Most research focuses on specific correlates of a disorder, but identifying nonspecific factors

that contribute to the development of multiple disorders is important (Klein & Riso). Given the high rates of comorbidity and the numerous theoretical perspectives accounting for the phenomenon, comorbidity among psychiatric disorders is likely to result from a combination of explanatory factors (Seligman & Ollendick, 1998).

Gender and Developmental Differences in Depressive Comorbidity

Gender and age are two variables that have been associated with the presentation and development of depression (Hankin et al., 1998). Gender differences, however, associated with the manifestation of comorbidity in depressed children and adolescents have been relatively unexplored in research (Kovacs et al., 2003). Some research suggests the prevalence rates for comorbid disorders have been found to differ by gender and development. Lewinsohn and colleagues (1995) found depression and anxiety were more prevalent in girls, whereas, disruptive disorders and substance abuse were more common in boys. Although girls are more likely to be depressed, depression with a comorbid disruptive disorder is most commonly found in males (Lewinshon et al., 1995). In childhood, the prevalence of comorbid conduct disorder and depression is higher for boys than for girls; however, sex differences for this comorbid condition are not found during adolescence (Loeber & Keenen, 1994; Mitchell et al., 1988). These findings, therefore, suggest prior to adolescence comorbid depression and disruptive disorders is found more frequently in males than in females. In addition, having multiple comorbid anxiety disorders during childhood and adolescence has been found to be almost exclusively female (Lewinsohn et al., 1997).

Kovacs and colleagues (2003) found differing developmental trajectories of comorbid psychiatric disorders by sex. For comorbid externalizing disorders with

depression, defined as attention deficit, conduct, and oppositional disorders, depressed girls had a peak risk during middle adolescence. On the contrary, boys with depression had consistently higher rates of externalizing problems and an increased risk for substance abuse in late adolescence. The risk of anxiety disorders was stable for both boys and girls across development. Keiley et al. (2003) found girls to have less covarying externalizing and internalizing symptoms than boys as reported by teachers.

While sex and developmental differences regarding comorbidity with depression is a relatively new area of research (Kovacs et al., 2003), findings in this area have important implications for this study given it is specific to girls with depression in childhood and early adolescence. The emerging research suggests comorbidity between depression and a disruptive disorder in childhood is less frequent for girls, whereas, the chance of having anxiety and depression is relatively high in girls across development. Given these varying sex and developmental differences in comorbidity, findings suggest etiological factors may differ for young men and women with different comorbid conditions (Lewinsohn et al., 1995). These trends suggest the need to focus research on comorbid disorders which are significant for girls with depression in early adolescence. The current study, therefore, focuses on comorbid anxiety disorders.

Summary of Depression and Comorbidity

Children with depression have a substantially increased chance of having one or more psychiatric comorbid disorders (Angold & Costello, 1993; Birmaher et al., 1996). Depression is most commonly associated with anxiety disorders, conduct disorder/oppositional defiant disorder, and attention deficit hyperactivity disorders (Angold et al., 1999). The presence of a psychiatric comorbid disorder with depression is

associated with a number of negative outcomes including: an increased risk for recurrent depression, increase in depressive duration, higher number of suicide attempts, greater functional impairment, and less response to treatment (Birmaher et al.). Emerging research suggests rates and manifestations of comorbidity in depression may vary by age and sex. Comorbidity between depression and disruptive disorders in childhood is less frequent for girls, whereas, the chance of having anxiety and depression is relatively high in girls across development. Given the age and sex differences in comorbidity, comorbid depression and anxiety presents as the most frequent comorbid condition for the sample in this study, which are early adolescent girls.

A number of theoretical models have been proposed to account for comorbidity among psychiatric disorders, including methodological artifacts, diagnostic artifacts, inaccurate boundaries, and substantive causes (Angold et al., 1999; Klein & Riso, 1993). Angold and colleagues ruled out most of the artifactual accounts of comorbidity and suggest comorbidity is a real phenomenon likely accounted for by substantive causes. Comorbidity, therefore, most likely results from shared etiological pathways of having common risk factors or the disorders directly influencing each other (Klein & Riso).

In summary, comorbidity should be considered the rule rather than the exception in childhood depression (Biederman et al., 1995). In fact, research suggests "pure" depression is less common than depression with a comorbid disorder (Avenevoli et al., 2001). This finding is especially true for the comorbidity of anxiety and depression given that comorbid anxiety and depression is more common than the "pure" forms of these disorders (Zahn-Waxler et al., 2000). Hankin (2006) indicates there is a methodological need to assess both depression and it co-occurring symptoms to investigate whether

hypothesized vulnerability factors predict the development of the disorder specifically or the comorbid symptoms presented. Without taking comorbid symptoms into account when studying depression, it presents challenges in evaluating whether the predictors of depression are associated with depression or its overlapping symptoms (Hankin, 2006). Despite the high rates of comorbidity, few studies have tested models of depression while measuring both anxiety and depressive symptoms to ensure the proposed risk factors are specific to the disorder being studied (Hankin et al., 2004); therefore, there is a need for more complex models accounting for comorbidity.

Depression and Anxiety

A significant and meaningful relation exists between depression and anxiety in children and adolescents (Brady & Kendall, 1992; Seligman & Ollendick, 1998). The rates of comorbidity for anxiety and depression can be as high as 70%, but typically range from 20% to 50% (Angold et al., 1999; Brady & Kendall), and are found both successively and concurrently in individuals (Clarke & Watson, 1991). In fact, comorbidity between anxiety and depression is more common than the "pure" forms of these disorders (Zahn-Waxler et al., 2000). In a review of depression and comorbidity, Angold and Costello (1993) found a high prevalence rate for anxiety disorders in every study, with the rate of anxiety disorders being between 2 and 26 times higher in children with depression than children without depression. In a study examining comorbidity with anxiety disorders, researchers found anxiety was most commonly comorbid with MDD, even when adjusting for other disorders (Lewinsohn et al., 1997). Given that depression and anxiety commonly co-occur, it is important to understand how anxiety may relate to the development of depression.

Clinical Presentation of Anxiety Disorders

Anxiety disorders are considered one of the most prevalent categories of psychiatric disorders in children and adolescents (American Academy of Child and Adolescent Psychiatry, 1997). Some anxiety in childhood is developmentally appropriate; however, a substantial number of children are suffering from anxiety levels that warrant an anxiety diagnosis (Muris, 2006). In addition, anxiety disorders are found to commonly co-occur with each other. The DSM-IV-TR recognizes approximately 14 anxiety disorders that may occur in children, adolescents, or adults (American Psychiatric Association, 2000). Given the large number of anxiety disorders presented in the DSM-IV-TR, this section will focus only on the disorders assessed in the diagnostic interview, K-SADS-IVR, as part of this study, including: Generalized Anxiety Disorder, Post-Traumatic Stress Disorder, Panic Disorder, Separation Anxiety Disorder, Specific Phobias, Social Phobia, and Obsessive-Compulsive Disorder. A diagnosis of Anxiety Disorder Not Otherwise Specified may be given if there are significant anxiety symptoms that fail to meet the criteria of any specific anxiety disorders.

According the American Psychiatric Association (2000), Generalized Anxiety Disorder (GAD; Appendix D) is characterized by excessive worry and anxiety occurring most days for at least six months, and the individual must have difficulty controlling their worries. In addition to excessive worry, children must have one additional symptom (3 is required for adults) that has been present for more days than not over the past 6 months. Associated symptoms include: restlessness, lack of energy or fatigue, difficulty concentrating, irritability, muscle tension, and sleep disturbance. Anxiety symptoms should result in clinically significant functional distress and other anxiety disorders

should be ruled out. In children and adolescents, worries are typically associated with their performance or competence at school or in sporting events, even when they are not being evaluated. Children may worry about catastrophic events as well. Youngsters with GAD may present as overly conforming, perfectionist, approval seeking, and may require excessive reassurance. There is some evidence to suggest GAD in children may be over diagnosed; therefore, careful evaluation is required. Prevalence for GAD over a one-year period is approximately 3% (American Psychiatric Association).

Post-Traumatic Stress Disorder (PTSD) is the development of symptomatology following exposure to a traumatic stressor, involving the experience, witnessing, or learning of an event capable of causing death, injury, or threat to the physical integrity of oneself or another person (American Psychiatric Association, 2000; Appendix E). A child's reaction to this event must be intense fear, helplessness, horror, or associated with disorganized or agitated behavior. In order to meet the criteria for PTSD, children must have specific symptoms from three broad categories including: re-experiencing, avoidance/numbing, and increased arousal. The criteria require at least one symptom of re-experiencing, three symptoms of avoidance/numbing, and two symptoms of increased arousal. The child must have these symptoms for more than one month and must present with significant impairment in important areas of functioning. A child may have acute, chronic, or delayed onset of the disorder. There may be some developmental differences in symptom manifestation, with older children exhibiting more adult-like PTSD symptoms and younger children demonstrating more reenactment of the trauma. Lifetime prevalence rates for PTSD in community samples range from 1% to 8%, but rates may be

much higher in communities exposed to traumatic events (American Psychiatric Association).

Panic Disorder is characterized by the presence of recurrent, unexpected Panic Attacks, which is followed by a minimum of 1 month of excessive concern about having another attack, worry about consequences of the attack, or significant change in behavior related to the attack (American Psychiatric Association, 2000; Appendix F). The criteria for a Panic Attack is a period of intense fear or discomfort in which at least four of the following symptoms suddenly onset and peak within 10 minutes: heart palpitations or accelerated heart rate; sweating; trembling or shaking; sensations of shortness of breath; feelings of choking; chest pain; nausea or abdominal discomfort; dizziness; being detached from reality or oneself; fear of losing control; fear of dying; numbness or tingling sensations; and chills or hot flashes. Panic Disorder is usually characterized with or without agoraphobia. Most studies find lifetime prevalence rates for Panic Disorder to be between 1% and 3.5% (American Psychiatric Association).

Separation Anxiety Disorder is a disorder specific to children and adolescents and is characterized by excessive anxiety related to the separation from the home or individuals the child is attached to beyond what is developmentally appropriate (American Psychiatric Association, 2000; Appendix G). Children must demonstrate three of the following symptoms: recurrent excessive distress when separated from major attachment figure, persistent worry about major attachment figure, persistent concern about being separated from major attachment figure, excessive fear to be alone without major attachment figure, persistent reluctance or refusal to go to sleep without being near major attachment figure, repeated nightmares related to themes of separation, or repeated

complaints of physical symptoms when separation from attachment figure is anticipated. Duration of the disorder must be at least 4 weeks and onset must occur before 18 years of age. Prevalence estimates appear to be approximately 4% in children and adolescents and generally decrease as children age (American Psychiatric Association).

Specific Phobia is the persistent fear that is excessive or unreasonable regarding the presence or anticipation of a specific object or situation (American Psychiatric Association, 2000; Appendix H). Exposure to the object or situation provokes an anxiety response, which may take the form of a Panic Attack. In children, the anxiety response may be crying, tantrums, freezing, or clinging. Adults may recognize the fear is unreasonable; however, children may not. The phobic stimulus is usually avoided or associated with intense distress and the avoidance or distress interferes with the individual's normal routine or functioning. Duration must be at least 6 months for children and adolescents. Specific types include: animal, natural environment, blood-injection-injury, situational, and other. Fears of animals and other objects in the natural environment are particularly common in childhood. Current prevalence in community samples range from 4% to 8.8% (American Psychiatric Association).

Social Phobia, also known as Social Anxiety Disorder, is characterized by a persistent fear of social or performance situation in which embarrassment may occur (American Psychiatric Association, 2000; Appendix I). In children, there needs to be evidence for age-appropriate social relationships and anxiety must occur in peer settings, not just with adults. Exposure to the feared social situation provokes anxiety, which may be crying, tantrums, freezing, or shrinking from social situations. Adolescents may perceive the fear as unreasonable, but this may be absent in children. The social situation

is usually avoided or experienced with intense dread. Avoidance or distress in the feared situation should interfere with the person's normal routine or functioning. For children under the age of 18, the duration must be for a minimum of 6 months. Community based sample prevalence rates range from 3% to 13% (American Psychiatric Association).

Obsessive-Compulsive Disorder (OCD) is characterized by recurrent obsessions or compulsions, which cause distress, consume more than one hour a day, or significantly impair a person's normal routine or usual social activities or relationships (American Psychiatric Association, 2000; Appendix J). Obsessions are persistent ideas, thoughts, impulses, or images, which are intrusive and inappropriate causing marked distress. An individual senses the content of the obsession is not in his/her control and a thought he/she would not expect to have. Most obsessions are about contamination, repeated doubts, need to have things in a particular order, aggressive or horrific impulses, and sexual imagery. Compulsions are repetitive behaviors (e.g. hand washing, checking) or mental acts (e.g., praying, counting), which are to prevent or reduce anxiety. Compulsions are often driven to prevent some dreaded event or situation. The most common compulsions involve washing and cleaning, counting, checking, requesting or demanding assurances, repeating actions, and ordering. Lifetime prevalence rates in children and adolescents in community studies is 1% to 2.3% (American Psychiatric Association).

Tripartite Model of Depression and Anxiety

As previously mentioned, anxiety disorders have been found to be commonly associated with depression with rates approximately 30% to 80% (Angold et al., 1999; Birmaher et al., 1996). Assessment measures also reflect the close relation between the

disorders. Correlations between self-report anxiety and depression measures range from .50 to .70 and tend to lack discriminant validity (Brady & Kendall, 1992). In addition to self-report measures, diagnostic interviews also have symptom overlap. These factors suggest nosological and methodological considerations cannot be ruled out when conceptualizing the relation between depression and anxiety. Given the overlap between these two disorders, many questions regarding the shared and unique factors of these two disorders have been raised. Various perspectives have been offered to account for the significant overlap between depression and anxiety. Two generally competing views of depression and anxiety have been suggested in the literature (Brady & Kendall). First, anxiety and depression are viewed as separate constructs that have considerable symptom overlap. The other view indicates depression and anxiety share a common factor related to an overall negative affect.

Clarke and Watson (1991) proposed a tripartite model of depression and anxiety, suggesting the disorders share a common factor but are distinguished by unique elements of the disorder. The tripartite division of symptoms suggests both depression and anxiety share general affective stress or negative affect. Unique aspects to depression and anxiety are suggested. Physiological hyperarousal is specific to anxiety, whereas, lack of positive affect is specific to depression. The nonspecific symptoms, which are associated with the shared negative affect, are thought to account for the strong association between anxiety and depression. Literature generally supports two distinct constructs of anxiety and depression though there is some evidence to support the tripartite model (Seligman & Ollendick, 1998).

Jacques and Mash (2004) tested the tripartite model of depression and anxiety in elementary and high school boys and girls. Results indicated self-reported symptoms of depression and anxiety were highly correlated, which is consistent with prior findings (Brady & Kendall, 1992). Consistent with the tripartite model, negative affect was highly correlated with both anxiety and depression, depression was correlated with low positive affect, and anxiety was correlated with physiological arousal in the total sample. The results, however, did not support the specificity of the dimensions as suggested by tripartite model. Physiological arousal was also correlated with depression, and low positive affect was correlated with anxiety. While the results showed good convergent validity, there was a lack of divergent validity of the tripartite constructs. Laurent and Stark (1993) also found support for the tripartite model examining cognitive distortion in children. The depressed group endorsed fewer positive items, whereas, there was no difference between the endorsement of negative items between the depressed and anxious groups. These findings were consistent with depression being associated with low positive affect. Some research suggests that the relation between anxiety and depression may vary across development (Cole, Truglio & Peeke, 1997). Cole and colleagues found younger children, 3rd graders, were less distinguishable on depression and anxiety measures, supporting a unified construct. On the other hand, older children, 6th graders, had more differentiation, which supported the emergence of the tripartite model.

Summary of Depression and Anxiety

In summary, depression and anxiety are strongly related in children and adolescents with significant diagnostic overlap. The rates of comorbidity for anxiety and depression can be as high as 70%, but typically range from 20% to 50% (Angold et al.,

1999; Brady & Kendall, 1992). Comorbid anxiety and depression is often more common than the "pure" forms of these disorders (Zahn-Waxler et al., 2000). Anxiety disorders generally associated with depression include: GAD, PTSD, Panic Disorder, Separation Anxiety Disorder, Specific Phobia, Social Phobia, OCD, and Anxiety Disorder NOS. Self-report measures of anxiety and depression often lack discriminant validity, and diagnostic symptoms tend to overlap on diagnostic interviews. The tripartite model, proposed by Clarke and Watson (1991), suggests depression and anxiety share a general factor, negative affect, but each have unique factors distinctive to the disorder. Despite the strong relation between depression and anxiety, studies rarely measure both anxiety and depressive symptoms to ensure the proposed risk factors are specific to the disorder being studied (Hankin et al., 2004). This study, therefore, aims to explore common risk factors associated with depression and anxiety as well as the influence anxiety may have on depression.

Common Etiological Factors to Depression and Anxiety

As previously reviewed, depression and anxiety commonly occur together; therefore, it is important to account for anxiety when studying depression. One of the substantive causes of comorbidity suggests two disorders share common risk factors or the risk factors are correlated (Angold et al., 1999; Klein & Riso, 1993). Given that both anxiety and depression are multifactorial in origin, it is possible the disorders share common risk factors. Common risk factors may even impact the onset of comorbid disorders that emerge at different times. Biederman et al. (1995) suggest comorbid disorders manifesting at different ages for developmental reasons may still share common risk factors. Negative life events and family variables have been shown to be etiological

pathways in the development of depression and the cognitive triad; therefore, this section reviews the literature regarding these variables as risk factors for anxiety.

Negative Life Events

As previously discussed, the association between negative life events and depression has been well documented in research (Franko et al., 2004; Nolen-Hoeksma et al., 1992; Pine et al., 2002; Tiet et al., 2001; Tram & Cole, 2000). Negative life events are significantly related to the onset of a number of psychiatric disorders in children and adolescents (Goodyear et al., 1987; Tiet et al.). Life events have been highlighted in a recent review as one of the etiological pathways to anxiety (Muris, 2006). Using both a between family and within family design, Boer and colleagues (2006) examined the relation between negative life events and anxiety disorders in a sample of 8 to 13 year olds. Results found anxious children experienced significantly more negative events, as reported by the parents, than the control children. This result was consistent for both lifetime negative events and those in the preceding 12 months. In addition, children with anxiety disorders also experienced more negative events than their siblings without an anxiety disorder in both the number of shared events and non-shared events. Shared events by siblings were rated as more negative by the parents for their child with an anxiety disorder, which could reflect objective differences, heightened awareness of their child's vulnerability, or reporter bias. Another study had similar results, finding children with an anxiety disorder reported significantly more negative life events the year prior to the anxiety onset compared to controls (Gothelf et al., 2004). In a longitudinal study of psychiatric disorders in the community, undesirable life events were significantly related to the onset of anxiety disorders (Cuffe et al., 2005). In addition, the experience of

negative life events may have a more negative impact for individuals with anxiety. Rappe, Litwin, and Barlow (1990) found the number of stressful life events did not differ between anxious and control groups; however, participants with an anxiety disorder reported a significantly more negative impact of life events than did the nonanxious group.

Adult literature on negative life events and psychiatric disorders, suggests specific categories of negative life events are associated with specific types of disorders. Specifically, research suggests events related to threat are associated with anxiety disorders and events related to loss are associated with depressive disorders (Sandin, Chorot, Santed & Valiente, 2004). Eley and Stevenson (2000) found some support for specific life events and the experience of depression and anxiety in children. The authors categorized life events as loss (i.e. the loss of attachment figure or loss of valued idea) or threat (i.e. risk of loss of attachment figure, trauma as a witness, physical jeopardy, or psychological challenge). Eley and Stevenson found reports of loss events in participants with depression significantly differed from the control group, whereas, threat events did not. Similarly, threat events in participants with anxiety disorders significantly differed from controls but loss events did not. The results, therefore, lend support that loss events are more likely to be associated with depression and threat events are more closely related to anxiety. These studies demonstrate that negative life events and life stressors likely play a role in the development of an anxiety disorder.

Parental Depression and Anxiety

The association between parental depression and offspring depression is well established (Beardslee et al., 1998; Weissman & Jenson, 2002; Pilowsky et al., 2006).

While there is established within-disorder association between parental and offspring depression, parental depression has been also linked to a number of cross-disorder associations. Children with a depressed parent are at a higher risk of having early depression, anxiety disorders, disruptive disorders, and alcohol dependence (Lieb et al., 2002; Pilowsky et al.; Weissman & Jenson, 2002). In a study examining depressed mothers in a primary care setting, mothers with depression compared to the control group were three times more likely to report their child as having serious emotional difficulties (Weissman et al., 2004). After controlling for other parental comorbid diagnoses, Weissman and colleagues found maternal MDD accounted for 55% of the variance of the child's emotional difficulties. In a recent study in 2006, Pilowsky and colleagues found high rates of psychopathology in children of 151 depressed mothers. Thirty-four percent of the children had a current diagnosis, including disruptive disorders (22%), anxiety disorders (16%), and depression (10%). A larger percentage of the children, 45%, met criteria for a lifetime diagnosis, including disruptive disorders (29%), anxiety (20%), and depression (19%). In addition, the children in the sample had high rates of comorbidity. Forty percent of the children with a depressive diagnosis had a concurrent anxiety disorder, which was independent of the child's age, gender, and severity of the mother's depression.

Parental depression has been specifically linked to anxiety symptomatology and disorders. A prospective longitudinal community study of 2,427 adolescents found parental depression was associated with anxiety disorders, even after adjusting for parental comorbidity (Lieb et al., 2002). The authors reported children with a depressed parent have a significant odds ratio (1.6) of developing anxiety when compared to

children with non-depressed parents. Children with a depressed parent and grandparent are at more risk for anxiety than depression, exemplifying the generational transmission of psychopathology (Warner, Weissman, Mufson, & Wickramarante, 1999; Weissman et al., 2005). Childhood anxiety is manifested more frequently in families with the highest familial loading for depression, which is important given that early anxiety is associated with an increased risk for later MDD (Warner et al., 1999). In addition, research findings indicate most anxiety disorders in children of mothers who are depressed have a preadolescent onset (Pilowsky et al., 2006; Weissman et al., 1997); therefore, the onset of anxiety among children of depressed parents may precede the onset of depression. Females of a depressed parent are a particularly high-risk group to develop early onset of anxiety before depression (Weisman & Jensen, 2002). These studies provide evidence that maternal depression is associated with an increased risk of having an anxiety disorder in childhood.

Maternal anxiety has also been found to significantly predict anxiety disorders in offspring (McClure et al., 2001). In a sample of 816 high risk adolescents, McClure and colleagues found children of mothers with an anxiety disorder were two times more likely to meet criteria for an anxiety disorder than peers whose mothers did not have an anxiety disorder. Adolescents, however, with the greatest risk of developing an anxiety disorder were those with mothers who had comorbid anxiety and depression. Participants whose mother presented with comorbid anxiety and depression were three times as more likely to have an anxiety disorder compared to the adolescents whose mother did not have a diagnosis. Similarly, Beidel and Turner (1997) examined anxiety disorders among children whose parents suffered from anxiety, depression, or both disorders. Their sample

consisted of 129 children, aged 7 to 12. Results indicated children of parents with an anxiety and/or depressive disorder have an increased risk for a broad spectrum of disorders, including anxiety disorders. The odds of a child having an anxiety disorder ranged from 3.76 to 7.38 higher if a parent had depression, anxiety, or mixed depression/anxiety. The results from Beidel and Turner's study suggested children whose parents experienced mixed depression and anxiety were at a higher risk for a broader range of disorders, including both anxiety and depression. Other studies have also found a significant relation between parents with both anxiety and depressive symptoms and internalizing symptoms in children (Bayer, Sanson & Hemphill, 2006; Grigoroiou-Serbanescu et al., 1991). Previous literature suggests both maternal depression and anxiety have been associated with symptoms of anxiety in their offspring. Furthermore, children of mothers who experience both anxiety and depression may be at a higher risk for developing an internalizing disorder than children whose mother has only depression or anxiety.

Family Environment

The family environment has broad implications on child and adolescent functioning and emotional development (Cuffe et al., 2005). Research investigating the relation between familial factors and anxiety has been sparse despite the high rates of comorbidity with depression (Vazsonyi & Belliston, 2006). Family factors, however, have been cited as a possible etiological pathway in the development of anxiety in children (Muris, 2006). When compared to normal controls, anxious children reported their family environments as being less supportive, more conflictual, less sociable, enmeshed, and more controlling (Stark et al., 1990). Thus, individuals with anxiety may

experience a more disturbed family environment than those without anxiety. Consistent with these findings, researchers found as the general functioning of the family deteriorated, levels of anxiety increased in a sample of young adults (Ballash, Pemble, Usui, Buckley, & Woodruff-Borden, 2006).

In addition to deficits in the general family environment, specific aspects of the family and anxiety have been explored. One study followed 303 families with an adolescent over 4 years examining the relation between parent-adolescent disagreements and internalizing symptoms (Rueter, Scaramella, Wallace, & Conger, 1999). Results found more parent-child conflict was associated with higher reports of anxiety at Time 1. Rueter et al. also found increases in parent-adolescent disagreements were associated with an increase in anxiety symptoms over the same time. The study, therefore, found persistent or increasing parent-child conflict is associated with an increase in anxiety and depressive symptoms. Family conflict was also positively related to both anxiety and depressive symptoms in adolescents in a cross-cultural study (Vazsonyi & Belliston, 2006).

Perceptions of parenting practices have also been associated with anxiety. In a review of literature, Rappe (1997) concluded both childhood and adult anxiety is associated with having parents that are rejecting and controlling. In an investigation with 117 school children, aged 9 to 12, researchers found positive associations between parental rejection, parental control, and anxiety symptoms (Gruner, Muris & Merckelback, 1999). These findings have been replicated in another study, which found parental overprotection and parental rejection were unique predictors of anxiety symptoms (Muris et al., 2006). These studies suggest family environments characterized

by parental rejection and overcontrolling behaviors are associated with anxiety symptoms in youngsters.

Summary of Common Etiological Factors to Depression and Anxiety

One of the proposed explanations for the association between comorbid disorders is common risk factors. Given that depression and anxiety often co-occur together, it is possible they share similar etiological pathways. This section reviewed the association between anxiety and common etiological factors associated with depression, including negative life events, parental depression and anxiety, and the family environment. Negative life events have been associated with anxiety symptoms and disorders in many studies (Boer et al., 2006; Cuffe et al., 2005; Gothelf et al., 2004; Phillips et al., 2005). Some research suggests life events associated with threat are more closely related to anxiety compared to other disorders (Eley & Stevenson, 1999), and individuals with anxiety disorders may interpret stress events more negatively (Rapee et al., 1990).

Family factors have also been cited as a possible contributor in the development of anxiety in children (Muris, 2006). Parental anxiety and depression, particularly comorbid anxiety and depression, has been associated with anxiety disorders in children (Beidel & Turner, 1997; McClure et al., 2001). In addition, most anxiety disorders in children of mothers who are depressed have a preadolescent onset, suggesting the onset of anxiety among children of depressed parents may precede the onset of depression (Pilowsky et al., 2006; Weissman et al., 1997). Aspects of the family environment and parent rearing behaviors have been associated with anxiety in children. Family environments characterized by low support, more conflict, being less sociable, enmeshment, and more controlling behaviors have been associated with anxious children

(Stark et al., 1990). Parental control and rejection have also been consistently related to childhood anxiety in research (Rappe, 1997). Research, therefore, suggests stressful events and familial factors are associated with both anxiety and depression in children and adolescents.

Influence of Anxiety on Depression

Another substantive explanation for the association between comorbid disorders is one disorder is a risk factor for the other disorder (Klein & Riso, 1993). Nottlemann and Jensen (1995) present comorbidity in the context of child development, suggesting there may be developmental factors in the co-occurrence of anxiety and depression. The authors note anxiety usually precedes depressive symptoms. Given this temporal relation between anxiety and depression, the substantive explanation of comorbidity suggests that anxiety may be a risk factor and influence the development of depressive symptoms. This section, therefore, reviews the literature regarding anxiety as a possible risk factor for depression.

Temporal Relation Between Anxiety and Depression

Temporally, psychiatric comorbid disorders with depression typically precede the onset of depression (Rohde, Clarke, Lewinsohn, Seeley, & Kaufman, 2001). Accordingly, onset of most subtypes of anxiety precedes the initial onset of major depression (Avenevoli et al., 2001; Costello et al., 2003; Lewinsohn et al., 1997). Childhood anxiety has been cited as a likely pathway to adolescent and adult depression and may be considered a risk factor to depression (Flannery-Schroeder, 2006; Seligman & Ollendick, 1998; Wittchen et al., 2003). Examining a developmental model for the etiology of depression in adult women, Kendler and colleagues (2002) found early onset

of an anxiety disorder was a potent and unique risk factor for past and present depressive episodes in the sample.

The influence of anxiety on depression has also been shown in children and adolescents. A longitudinal study of elementary age children found high levels of self and parent reported anxiety predicted increases of self and parent reported depression, while controlling for previous depression (Cole et al., 1998). Reports of depression, however, did not predict levels of anxiety. Results suggest the onset of depression is influenced by prior anxiety, but depression does not predict anxiety in children. Bittner and colleagues (2004) examined the relation between anxiety disorders and the development of subsequent depression in 3,021 adolescents and young adults, aged 14 to 24. Findings indicated the presence of any anxiety disorder and each of the anxiety disorders, including GAD, panic disorder, agoraphobia, social phobia and specific phobia, were associated with an increased risk of MDD. After adjusting for all other prior mental disorders, all anxiety disorders, with the exception of panic disorder, remained significantly related to the development of MDD. The authors found a dose-response relation between anxiety and MDD, suggesting two or more anxiety disorders had a significantly higher risk of first onset of MDD than those with just one anxiety disorder. The severity of impairment of the anxiety disorder emerged as the most important predictor of MDD. Specific phobia, however, was only found to be associated with the onset of MDD if it was complicated by a comorbid anxiety disorder. Some research, however, suggests the temporal relation between anxiety and depression may differ with dysthymia. Kovacs and colleagues (1989) found DD tended to precede the onset of anxiety disorders, whereas, anxiety disorders developed before MDD. The sample of

comorbid DD and anxiety in this sample, however, was small (*n*=9). Overall, these studies suggest that anxiety in childhood may serve as a risk factor for the development of major depression in children.

Influence of Anxiety on Depressive Severity

In addition to being associated with the onset of depression, anxiety has been found to influence the severity of depression. Children with both anxiety and depression tend to report more severe depressive symptoms than individuals with depression or anxiety alone (Brady & Kendall, 1992; Kendall, Kortlander, Chansky, & Brady, 1992; Stark, Humphrey, Laurent, Livingston, & Christopher, 1993). Comorbid depression and anxiety has been associated with higher depressive scores and poor functioning prior to treatment. Rohde and colleagues (2001) found adolescents with any comorbidity, especially anxiety, had significantly higher depression intake scores and poorer global functioning than adolescents with no history of comorbidity. In a sample of 95 children and adolescents with depression, those with comorbid depression and anxiety (*n*=43) rated their symptoms as more severe than the depression only group (Mitchell et al., 1988). As such, these studies suggest children with anxiety and depression experience a more severe depressive symptomatology than individuals with depression alone.

Possible Mechanisms of Anxiety's Influence on Depression

Researchers have postulated several possible reasons for the influence of anxiety on depression. Possible reasons include: impairment of the anxiety disorder leads to stressors or life course changes, a possible genetic link between the two disorders, and shared environment or psychosocial factors (Bittner et al., 2004). Bittner and colleagues found severity of anxiety was an important predictor of depression onset, suggesting

impairment from anxiety might result in stressful experiences, which contributes to depression. The development of anxiety is likely to be disruptive to interactions between persons and their environment, thus creating stressors or life course changes, which may increase their likelihood of becoming depressed (Rohde et al., 1991). Thus, one mechanism by which anxiety may contribute to the onset of depression is by creating more life stressors in the child's life.

Zahn-Waxler and colleagues (2000) suggest rumination may be a contributing factor in the influence of anxiety on depression. Nolen-Hoeksema and Girgus (1994) suggest girls tend to have a ruminative, self-focused style of coping with life stress. Ruminative coping is characterized by individuals focusing on and passively ruminating about the distress rather than taking action to distract themselves or change the situation. Rumination is thought to be an important cognitive vulnerability factor to depression and excessive worry is linked to anxiety; however, the two are thought to be similar at the conceptual level (Muris, Roelofs, Meesters, & Boomsma, 2004). Muris and colleagues examined the relation between rumination, worry, depressive symptoms, and anxiety in sample of over 300 adolescents. Results indicated rumination and worry are substantially correlated. In addition, the authors hypothesized worry would be more associated with anxiety and rumination would be more associated with depression. The findings, however, indicated both worry and rumination were more associated with anxiety than depression. As suggested by Zahn-Waxler, rumination related to anxiety may exacerbate an individual's problems, which may contribute to depression.

Given that cognition is considered the "final pathway" to depression (Ingram, 2003), it is possible that anxiety symptoms in children may contribute to the development

of depressogenic thinking. Internalizing disorders in general are associated with cognitive distortions, which can refer to the exaggeration of threat, underappraisal of personal abilities, or misperceptions of environmental demands (Kendall et al., 1992). Beck's cognitive model of psychopathology suggests disorders may be differentiated by the content of their thinking, referred to as the cognitive content-specificity hypothesis (Beck, 1976). According to Beck's theory, cognitions associated with anxiety are more related to threat, whereas, depressive cognitions are associated with loss.

Laurent and Stark (1993) found partial support for the applicability of Beck's cognitive content-specificity hypothesis to children. In their study of children, grades 4 to 7, the authors found depressed children endorsed more items on a cognitive measure than the anxious group. Both groups, however, reported statistically similar levels of negative cognitions. Self-reported anxious cognitions did not differentiate anxious from the depressed group. The comorbid anxious and depressed group endorsed more negative statements than the anxious only group. Jacob and Joseph (1997) examined the association between Beck's cognitive triad, as measured by the CTI-C, and symptoms of depression and anxiety in children and adolescents. Results indicated a more negative cognitive triad was associated with both symptoms of depression and anxiety in the sample; however, the CTI-C accounted for more of the variance on the measure of depression than the measure of anxiety. In general, distorted information processing is likely not specific to depression and may underlie anxious cognition as well (Kendall et al., 1992). Given most cognitive origins are believed to develop in childhood (Beck, 1972) and a child's self-schema is be less structuralized than adolescents or adults (Cole

& Turner, 1993), it is possible that anxiety symptoms may be associated with a more negative cognitive triad.

Summary of the Influence of Anxiety on Depression

A substantive explanation for comorbidity is one disorder is a risk factor for the other disorder (Klein & Riso, 1993). Nottlemann and Jensen (1995) suggest there may be developmental factors in the co-occurrence of anxiety and depression given its high rates of comorbidity in children. Anxiety has been found to precede the initial onset of major depression (Avenevoli et al., 2001; Costello et al., 2003; Lewinsohn et al., 1997) and is cited as a likely risk factor to depression (Flannery-Schroeder, 2006; Seligman & Ollendick, 1998; Wittchen et al., 2003). In addition to preceding depression, children with comorbid anxiety and depression tend to have a more severe depression than individuals with depression alone. This research suggests anxiety, therefore, may play a role in the development of depression. Based on the literature, it is still unclear how anxiety may influence the development of depression. Researchers believe it may be related to increased stressors due to functional impairment from the anxiety disorder, a possible genetic link between the two disorders, and shared environment or psychosocial factors (Bittner et al., 2004). Rumination has been highlighted as one cognitive mechanism of transmission given its relation to both anxiety and the development of depression. Research suggests it is also possible for anxiety to contribute to the development of a negative cognitive triad. This study, therefore, investigates the direct effects anxiety has on depressive severity and the cognitive triad.

Statement of the Problem

Depression during childhood is a common, recurrent, and disabling disorder (Kovacs, 2006; Kovacs et al., 1984) associated with significant functional impairment in school and relationships (Birmaher et al., 1996; Puig-Antich et al., 1993). The prevalence of depressive disorders steadily increases with age until early adolescence when the rate of depression dramatically increases (Lewinsohn et al., 1994). Prior to adolescence, the rate of depression for boys and girls is relatively the same; however, the prevalence rate for females doubles the rate for males around the onset of adolescence making girls particularly vulnerable to depression (Hankin et al., 1998). Given the increase in prevalence of depression around adolescence and the emerging gender differences, understanding the correlates of depression in early adolescent girls is an important area of research.

In trying to understand the etiology of depression, research has focused on multiple pathways associated with the development and onset of depression in children and adolescents (for a review, see Stark et al., 2000). A number of major theoretical approaches have been proposed regarding the etiology of depression including: biological models, cognitive models, behavioral/interpersonal models, and life stress models (Ciccetti & Toth, 1998; Hammen & Rudolph, 2003). Research suggests depressogenic cognitions play an essential role in the development of depression and may mediate the relation between risk factors and depression (Cole & Turner, 1993). Understanding the developmental origins of cognitions in children, therefore, is an important component of a developmental model of depression in early adolescents. Depression is a multifactorial disorder with diverse risk factors, which need to be

integrated into a comprehensive, transactional model of depression; however, few studies have combined these factors into an integrative model (Kendler et al., 2002). The present study integrates some of these risk factors into a comprehensive model of depression that is salient for early adolescent girls. In other words, this study examines a primarily interpersonal cognitive model of depression, exploring how various life stressors (i.e. parental depression and anxiety, family environment, negative life events) are correlated with cognitions and depression in early adolescent girls.

A limitation to current research examining models of depression is they typically do not account for comorbidity. The presence of depression in children and adolescents increases the chance of having another disorder around 20-fold, but can increase the likelihood up to 100-fold (Angold & Costello, 1993). Comorbidity is now being considered the rule rather than the exception in childhood depression (Biederman et al., 1995), and is associated with a number of negative outcomes including: increased risk for recurrent depression, longer duration of an episode, more suicide attempts, greater functional impairment, and lower response to treatment (Birmaher et al., 1996). Comorbid disorders commonly associated with depression in youth include conduct disorder/oppositional defiant disorder, anxiety disorders, and attention deficit hyperactivity disorders (Angold et al., 1999); although, developmental and sex differences have been found (Kovacs et al., 2003: Lewinsohn et al., 1997). In girls, the risk of comorbid disruptive disorders tends to be highest in middle adolescence; however, the risk of comorbid anxiety is prevalent across development (Kovacs et al.). Examining comorbid anxiety, therefore, appears to be important when proposing models of depression in early adolescent girls.

While a number of explanations for comorbidity have been presented, both artifactual and substantive, most explanations asserting comorbidity is an artifact can be ruled out (Angold et al., 1999). The presence of comorbidity most likely results from shared etiological pathways of having common risk factors or the disorders directly influencing each other (Klein & Riso. 1993). Depression and anxiety have a strong association in children with significant diagnostic overlap. In fact, anxiety and depression are thought to share a general factor, known as negative affect (Clarke & Watson, 1991). As suggested by one substantive explanation for comorbidity, anxiety and depression have been found to share similar risk factors associated with parental depression and anxiety, family environment, and life stressors. Additionally, anxiety has been found to precede depression and be a possible risk factor for the development of depression (Flannery-Schroeder, 2006).

Despite the high co-occurrence of depression and anxiety, few studies have tested models of depression while measuring both anxiety and depressive symptoms to ensure the proposed risk factors are specific to the disorder being studied (Hankin et al., 2004). Therefore, there is a methodological need to assess both depression and co-occurring anxiety to investigate whether hypothesized vulnerability factors predict the development of depression specifically or the comorbid symptoms presented (Hankin, 2006; Seligman & Ollendick, 1998). Without taking comorbid symptoms into account when studying depression, erroneous conclusions and theories may emerge (Keiley et al., 2003; Rutter & Sroufe, 2000).

The current study integrates correlates of depression while accounting for comorbid anxiety symptoms. Based upon substantive theories of comorbidity, this study

examines whether the proposed pathways of depression are correlated with the severity of depression and comorbid anxiety symptoms and whether the anxiety symptoms mediate the relation between the risk factors and severity of depression. Overall, the present study has two aims: (1) to replicate and build upon a cognitive-stress model of childhood depression, with depressogenic cognitions mediating the relation between stressors and depression, and (2) to present a model of childhood depression integrating comorbid anxiety symptoms as a possible mediator and risk factor for childhood depression.

Research Hypotheses

Hypothesis 1

Participants' report of depressogenic cognitions, as measured by the CTI-C, will directly affect the severity of her depressive symptoms (see path a in Figure 2). Specifically, lower scores on the total score of the CTI-C will be associated with higher scores on the composite depressive symptoms scale of the K-SADS-IVR.

Rationale. Previous research has shown children with depressogenic cognitions experience more depressive symptoms than children with a more positive sense of the self, world, and future (Epkins, 1996; Kendall et al., 1990; Stark et al., 1996). The relation between depressogenic cognition and depression also has been demonstrated in longitudinal, prospective studies. Both Alloy et al. (1999) and Lewinsohn et al. (2001) found individuals who had a higher cognitive vulnerability were more likely to become depressed than those who were considered low risk for cognitive vulnerability. This hypothesis will replicate previous research finding a more negative cognitive triad is associated with higher depressive symptom severity.

Hypothesis 2

Participants' anxiety symptoms, as measured by the K-SADS-IVR, will have a direct effect on her cognitive triad (see path b in Figure 2) and depressive symptom severity (see path c in Figure 2). Specifically, higher composite scores of anxious symptomatology on the K-SADS-IVR will be associated with a more depressogenic cognitive triad (i.e. lower scores on the total score of the CTI-C) and higher scores on the composite depressive symptoms scale of the K-SADS-IVR.

Rationale. Anxiety has been found to precede the initial onset of major depression (Avenevoli et al., 2001; Costello et al., 2003; Lewinsohn et al., 1997) and is cited as a likely risk factor to depression (Flannery-Schroeder, 2006; Seligman & Ollendick, 1998; Wittchen et al., 2003). A longitudinal study of elementary school children found high levels of self and parent reported anxiety predicted increases of self and parent reported depression, while controlling for previous depression (Cole et al., 1998). In addition, children with both anxiety and depression tend to report significantly greater depression than individuals with depression or anxiety alone (Brady & Kendall, 1992; Kendall et al., 1992; Stark et al., 1993). Thus, it is hypothesized that higher levels of anxiety will act as a vulnerability factor and be associated with more depressive symptoms. Anxiety and depression are both thought to have underlying cognitive dysfunction (Beck, 1976). Research has shown a more negative cognitive triad, as measured by the CTI-C, has been associated with anxiety symptoms but is more closely related to depression (Jacobs & Joseph, 1997; Laurent & Stark, 1993). Given that the negative schema is thought to develop in childhood, this hypothesis asserts that anxiety symptoms directly influence the cognitive triad in the girls.

Hypothesis 3

Mothers' reports of symptoms of depression and anxiety, as measured by the SCL-90R, will directly affect the severity of the daughters' anxiety symptoms (see path d in Figure 2) and cognitive triad (see path e in Figure 2). In addition, there will be an indirect association between mothers' reports of symptoms of depression and anxiety and the daughters' depressive symptoms via the daughters' anxiety symptoms (see paths d and c in Figure 2) and cognitive triad (See paths e and a in Figure 2). In other words, higher scores summated from the depression and anxiety symptom scales of the SCL-90R as reported by the mothers will be significantly associated with higher scores on the composite anxiety symptoms scale on the K-SADS-IVR and lower total scores on the daughters' CTI-C. Higher anxiety symptoms and a more negative cognitive triad will then be associated with a higher composite depressive symptoms scale of the K-SADS-IVR. The indirect path of effect, as hypothesized in this model, indicates maternal depression and anxiety influences both the daughters' anxiety symptoms and cognitive triad, which in turn influence their daughters' depressive symptomatology.

Rationale. Children with a depressed parent are at a high risk for a variety of psychological difficulties, including early depression, anxiety disorders, disruptive disorders, and alcohol dependence (Lieb et al., 2002; Pilowsky et al., 2006; Weissman & Jenson, 2002). Having a parent with major depression is considered one of the strongest predictive factors in childhood or adolescent depression (Beardslee et al., 1998). Research also links maternal depression and children's negative cognition, suggesting exposure to a depressed mother may be one process by which negative cognitions develop in children (Garber & Flynn, 2001). As hypothesized, parental depression and

anxiety is thought to influence the development of the cognitive triad. Furthermore, comorbid anxiety with depression in adults is common (Kessler et al., 1994). Research has suggested comorbid anxiety with maternal depression increases the risk of child psychopathology (Grigoroiu-Serbanescu et al., 1991). Additionally, parental depression and anxiety has been specifically associated with anxiety (Beidal & Turner et al., 1997; Lieb et al., 2002; Warner et al., 1999; Weissman et al., 2005). Research findings indicate most anxiety disorders in children of parents who are depressed have a preadolescent onset, which indicates the onset of anxiety among children of depressed parents may precede the onset of depression (Pilowsky et al., 2006). In addition, females of a depressed parent are a particularly high-risk group to develop early onset of anxiety before depression (Weisman & Jensen, 2002). Based on this research, it is hypothesized children of depressed and anxious mothers are at greater risk for developing anxiety, which in turn contributes to the development of depression. This model, therefore, examines the indirect influences of maternal depression and anxiety on daughters' depression via the daughters' anxiety symptoms and depressogenic cognitions.

Hypothesis 4

Participants' reports of the family environment, as measured by the SRMFF-CR, will directly affect her anxiety symptoms and cognitive triad. Specifically, more cohesive and less conflictual family relationships (see paths f and g in Figure 2), more communication within the family (see paths h and i in Figure 2), and higher levels of the family engagement in social recreational activities (see paths j and k in Figure 2) will be associated with a lower composite score of anxiety symptoms and a more positive cognitive triad (i.e. higher score on the total score of CTI-C). In this hypothesized model,

participants' anxiety symptoms and cognitive triad will mediate the relation between the family environment and depressive symptoms in the girls. Thus, the family environment will indirectly influence depressive symptoms via the participants' cognitive triad and anxiety symptoms.

Rationale. Research indicates multiple family factors are associated with depression in youth (Sander & McCarty, 2005). Research has found depressed children have families that are less cohesive, more controlled and conflictual, communicate less, express emotion more intensely, and engage in fewer social and recreational activities (Barerra & Garrison-Jones, 1992; Cole & McPherson, 1993; Jewell & Stark, 2003; Ostrander et al., 1998; Stark et al., 1990). The family environment has been another avenue through which children develop maladaptive cognitions. Lower levels of maternal acceptance (Garber & Flynn, 2001; Rudolph et al., 2001), higher levels of rejection (Rudolph et al.), and negative parenting (Gibb et al., 2001; Mezulis et al., 2006; Rose et al., 1994) have been associated with negative cognitions in adolescents. Previous research using data of the current sample found family conflict, communication, and social/recreational engagement directly affected the cognitive triad (Greenberg et al., 2008). Thus, this hypothesized model proposes the family environment factors being measured (i.e. family relationships, communication, and social/recreational engagement) will be associated with the girls' cognitive triad, which will directly affect her level of depression. In addition, family factors have also been highlighted in the development of anxiety (Muris, 2006). Family environments characterized by low support, more conflict, being less sociable, enmeshment, and more controlling behaviors have been associated with anxious children (Stark et al., 1990). Parental control and rejection have also been

consistently related to childhood anxiety in research (Rappe, 1997). As suggested in the hypothesized model, the family environment variables will be significantly related to anxiety, which then influences depressive severity. Thus, the indirect effects of the family environment on depressive severity are evaluated.

Hypothesis 5

Participants' report of negative life events, as measured by the LEC, will directly affect her anxiety symptoms (see path l in Figure 2) and her cognitive triad (see path m in Figure 2). Specifically, a higher number of negative life events, as determined by ratings of type of event and impact of event on the LEC, will be associated with a higher composite score of anxiety symptoms and a more negative cognitive triad (i.e. lower score on the total score of CTI-C). In this hypothesized model, participants' anxiety symptoms and cognitive triad will mediate the relation between negative life events and depressive symptoms in the girls. Thus, negative life events will indirectly influence depressive symptoms via the participants' cognitive triad (see paths m and a in Figure 2) and anxiety symptom severity (See paths l and c in Figure 2).

Rationale. Negative life events have been shown to predict depression in longitudinal studies using adolescent and adults (Franko et al., 2004; Pine et al., 2002; Tram & Cole, 2000). In a five-year longitudinal study, negative life events were found to predict depression in young children and continued to predict depression across five years (Nolen-Hoekesma et al., 1992). Garber and Flynn (2001) suggest the experience of negative life events is likely one of most important factors underlying the development of negative cognition. Multiple studies have found cognitive vulnerability mediated the relation between negative life events and depression in children (Cole & Turner, 1993;

Greenberg et al., 2008; Rienemann & Teeter Ellison, 2004; Tram & Cole, 2000). This study aims to replicate these findings examining the indirect effects of negative life events on depression via the cognitive triad. In addition, negative life events have been associated with anxiety symptoms and disorders in many studies (Boer et al., 2006; Cuffe et al., 2005; Gothelf et al., 2004; Phillips et al., 2005). This study examines the direct effects of negative life events on anxiety and the possibility of anxiety mediating the relation between negative life events and depression in early adolescent girls.

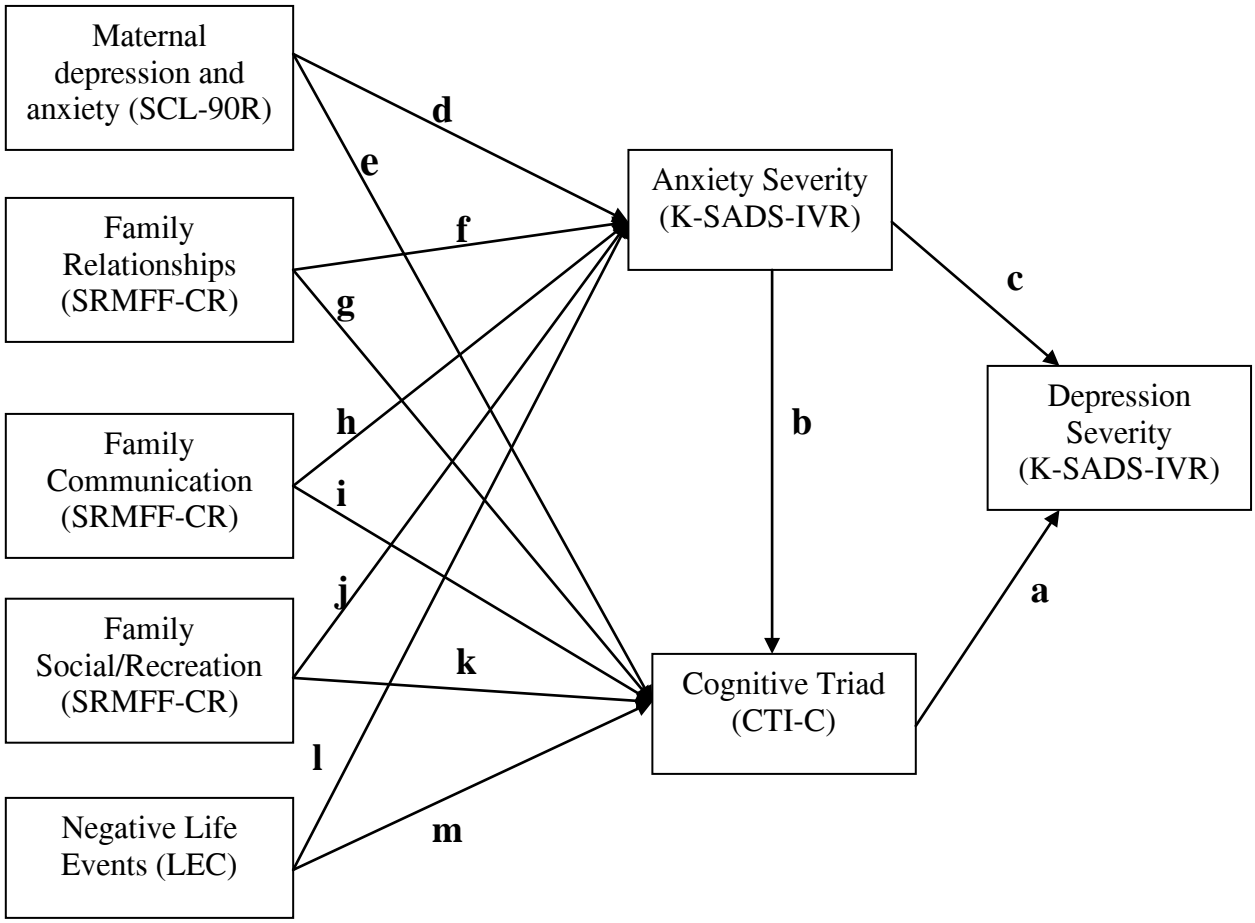


Figure 2. Proposed path analysis model

CHAPTER 3

Method

Participants

The sample includes 203 girls, aged 9 to 14 (*M*=10.79, *SD*=1.29), enrolled in grades 4 to 7 (*M*=5.48, *SD*=1.12), in two suburban central Texas school districts. One hundred fifty-four of the girls, along with a caregiver (*n*=99), are participants from a larger study evaluating the effectiveness of cognitive-behavioral therapy with and without a parent-training component for early adolescent girls with a depressive disorder. These participants have a primary diagnosis of MDD (*n*=114), MDD in partial remission (*n*=19), Dysthymic Disorder (*n*=16), Adjustment Disorder with Depressive Features (*n*=1) or Depressive Disorder NOS (*n*=4). Of the 154 girls, 58% of the participants received a psychiatric comorbid diagnosis, with 50% receiving at least one comorbid anxiety disorder diagnosis. Of the girls who had a psychiatric comorbid disorder, 86% had at least one comorbid anxiety disorder. Comorbid anxiety disorders in the sample include: GAD, PTSD, Separation Anxiety, Social Phobia, Specific Phobia, Panic Disorder and Anxiety Disorder NOS (see Table 1 for a summary of diagnoses). In addition, 49 child participants, along with a caregiver (*n*=47), from the control sample are included in order to increase the heterogeneity of variance in the sample. Of the 49 children in the control sample, 10 children received a psychiatric diagnosis, which included: MDD (*n*=2), DD-NOS (*n*=1), Specific Phobia (*n*=2), Oppositional Defiant Disorder (*n*=1), Attention Deficit Disorder (*n*=1), Panic Disorder (*n*=1) and GAD (*n*=1).

Two children in this sample received comorbid diagnoses. One child had comorbid Panic Disorder and Specific Phobia, whereas, the other child had comorbid MDD, GAD, OCD, and ADHD. Demographics of the total sample are presented in Table 2.

Participants were excluded from the larger study if they met one or more of the following criteria: (1) an additional psychological disorder that presents as primary due to its severity and impact on the child's life, (2) psychotic symptoms, (3) actively suicidal or homicidal, (4) currently being treated for depression through an outside therapist or pharmacological interventions, (5) have an IQ below 85 or a learning disability that would prevent them from validly completely research measures, or (f) have a severe medical disability that would prevent them from regularly attending meetings or completing activities. Control sample participants were excluded if they had an IQ below 85 or a learning disability that would prevent them from validly completing research measures.

Table 1

Summary of Participant Diagnoses (n=154)

Diagnosis	Frequency	Percent
Major Depressive Disorder	114	74.0
Dysthymia	27	17.5
Depressive Disorder NOS	4	2.6
Adjustment Disorder with Depressed Features	1	0.6
Major Depressive Disorder in Partial Remission	18	11.6
Generalized Anxiety Disorder	51	33.1
Specific Phobia	20	12.9
Separation Anxiety	11	7.1
Post Traumatic Stress Disorder	6	3.9
Social Phobia	6	3.9
Panic Disorder	1	0.6
Anxiety Disorder NOS	3	1.9
Eating Disorder	3	1.9
Oppositional Defiant Disorder	3	1.9
Attention Deficit Disorders	16	10.4

Table 2

Summary of Participant Demographic Variables (N=203)

Variable	Frequency	Percent
Age		
9	37	18.2
10	58	28.6
11	40	19.7
12	47	23.2
13	20	9.9
14	1	0.5
Grade		
4	49	24.1
5	58	28.6
6	46	22.7
7	50	24.6
Ethnicity		
Latina	64	31.5
White	89	43.8
African American	23	11.3
Asian	6	3.0
Biracial/Multiethnic	21	10.3
Parent Educational Status		
Less than high school	2	1.0
Some high school	7	3.4
Finished high school/GED	23	11.3
Some college/junior college	56	27.6
Finished 4 year college	34	16.7
Advanced degree	18	8.9
Unknown/Missing	62	30.5

Instrumentation

Measures of Depression

Children's Depression Inventory (CDI; Kovacs, 1992). The CDI is the most commonly used self-report measure of depression in children and adolescents and is designed for ages 7 to 17. The measure consists of 27 items assessing the presence and severity of cognitive, affective, and behavioral symptoms of depression over the preceding two weeks. Raters are provided with a three-alternative choice format, with each alternative representing a different level of severity. Statements are scored 0 to 2 in order of increasing severity. Items scores are then added up to a total depression score, ranging from 0 to 54. The general screening cut-point recommended by Kovacs (1992) is 20, and the median score for girls, aged 7 to12, is 9. For screening purposes, however, a cut-off score of 16 or above has been shown to have the highest predictive value (Timbremont, Braet, & Dreeson, 2004). In other words, the specificity and sensitivity of the CDI are maximized when the cut-off score is 16. Administration of the CDI is approximately 15 minutes or less and may be administered individually or in groups.

The CDI demonstrates good internal consistency, with reported reliability estimates ranging from .71 to .86 (Kovacs, 1992). While split-half and alpha reliability measures tend to be consistently adequate, test-retest reliability has ranged from .38 to .87 in different samples of children (Kovacs; Saylor, Finch, Spirito & Bennett, 1984). The wide range of test-retest reliability coefficients may reflect the mood dependent nature of the measure, given that it is designed to measure state rather than a trait depressive mood (Kovacs). The CDI has shown good discriminant validity between

clinical samples and school children without emotional distress (Carey, Faulstich, Gresham, Ruggiero & Enyart, 1987; Saylor et al., 1984). While some past research has shown the CDI is unable to statistically discriminate between depression and other disorders in clinical samples (Carey et al.; Saylor et al.), Timbremont and colleagues (2004), found the CDI to have good predictive validity and was able to successfully discriminate depressive disorders from anxiety and disruptive disorders. Overall, the validity of the CDI has been well established using a variety of techniques (Kovacs). While the CDI cannot be used by itself as a psychiatric diagnosis (Sitarenios & Kovacs, 1999), there is strong support for the use of the CDI as a screening tool for depression (Timbremont et al.). The CDI was given in the screening process of this study. Cronbach's alpha for the screening sample was .90.

Beck Depression Inventory for Youth (BDI-Y; Beck, Beck & Jolly, 2001). The BDI-Y is a measure assessing the presence and severity of depressive symptoms in children and adolescents, aged 7 to 14. The measure was developed as part of a group of questionnaires designed to assess youth's emotional and social functioning. Administration time is approximately 5 to 10 minutes and may be given individually or in groups. The measure includes 20 items assessing negative thoughts, feelings of sadness, and physiological symptoms of depression. Each statement is on a 4-point scale of *never*, *sometimes*, *often,* and *always*. Scores for each item are summated for a total score, with higher scores reflecting greater depressive severity. Scores range from 0 to 60. Based on the age group, 7 to 10 years old, scores of 35 or higher are extremely elevated, scores 25-34 are moderately elevated, scores 20-24 are mildly elevated, and scores 20 and below are considered average. For the 11 to 14 year old group, scores of 29 or higher are

extremely elevated, scores 21-28 are moderately elevated, scores 17-20 are mildly elevated, and scores 16 and below are considered average (Beck et al., 2001).

Internal consistency of the BDI-Y has been found to be high, with coefficient alphas of .91 for females aged 7 to 10, .90 for males aged 7 to 10, .91 for females aged 11 to 14, and .92 for males aged 11 to 14. Test-retest reliabilities ranged from .79 to .92 over a retest interval of seven days (Beck et al., 2001). The BDI-Y has also been found to have high convergent validity with the CDI total score (r=.72). Additionally, children with a mood disorder scored significantly higher on the BDI-Y than children from other clinical groups (Beck et al.). These reliability and validity estimates have been replicated on school-based sample of girls (N=859), aged 9 to 13 (Stapleton, Sander, & Stark, 2007). Reliability and validity estimates were found to be similar across race and ethnic groups, but were found to be slightly lower for the 9-year-old age group. Overall, Stapleton and colleagues found support for the use of the BDI-Y as a screening tool for depression. The BDI-Y was given as part of the screening process in this study. Cronbach's alpha for the screening sample was found to be high (α=.93).

Diagnostic and Statistical Manual Brief Symptom Interview for Depression (DSM Interview; Stark & Sander, 2002). This is a semi-structured interview created for the purposes of screening and monitoring depressive symptoms within the context of a large ongoing depression study. The interview is based upon the DSM-IV criteria for depressive disorders and briefly assesses the presence of symptoms. A symptom is considered "present" if the child reports it as a problem for most days within the past two weeks and is considered clinically impairing. The DSM interview was used within the screening process of this study. Cronbach's alpha for the DSM Interview for this study

was acceptable (α=.86). The number of symptoms endorsed on the DSM-IV Interview was summated for a total score. The total score had a positive significant correlation the BDI-Y (r=.49, p<.001), and the CDI (r=.51, p<.001), demonstrating convergent validity.

Measures of Depression and Anxiety

Schedule for Affective Disorders and Schizophrenia for School Age Children (K-SADS-IVR; Ambrosini & Dixon, 2000). The K-SADS-IVR is a semi-structured clinical interview designed for use with both a parent and child by a mental health clinician. The K-SADS is the most widely used semi-structured interview for children and adolescents (Klein et al., 2005). The interview is appropriate to use with children from ages 6 to 18 with normal intelligence. The interview assesses the presence, absence, and severity of symptoms according to the DSM-IV criteria in six major areas: major depression, mania, eating disorders, anxiety disorders, behavioral disorders, substance abuse, and psychotic disorders. Specific anxiety disorders assessed include: GAD, PTSD, Panic Disorder, Separation Anxiety, Specific Phobias, Social Phobia, and OCD. In the larger study, all participants completed the depression interview section and screening questions were provided to assess possible co-occurring disorders. If the participant endorsed the screening question, the entire section of that disorder was administered. On the other hand, if the participant did not endorse the screener, the interviewer proceeded to other sections of the interview. Administration time is considered to be lengthy, approximately 1.5 hours for each parent and child interview. Administration time, however, varies depending on range and severity of psychopathology (Kaufman et al., 1997). The K-SADS-IVR is not a completely inclusive interview and does not assess Axis II diagnoses,

pervasive developmental disorders or learning disabilities (Ambrosini, 2000). For this study, the depression and anxiety sections were analyzed.

The K-SADS-IVR has been modified from its previous version in order to be compatible with the current DSM-IV diagnoses. According to Ambrosini (2000), ratings from the participant and a primary caregiver are obtained separately and the interviewer provides a consensus summary rating taking into account all sources of information. The interviewee is asked about symptoms during two different time periods, the past 12 months (present episode) and the past 7 days (last week). Each symptom is given a severity rating from its most severe point during the present episode and from its most severe point during the last week. Severity ratings typically range from 0 to 4 and from 0 to 6. The ordinal values represent symptoms based upon increasing levels of severity and frequency (e.g. 1=not at all, 2= slight/occasional, 3=mild/sometimes, 4=moderate/often, 5=severe/most of the time, 6=extreme/almost all the time). Symptoms are indicated to be clinically significant if they receive a three or greater on the scales. The K-SADS-IVR also provides measures of global assessment, clinical global severity and improvement, and dates of onset and offset for each disorder.

Considering the K-SADS-IVR is a relatively recent version of the K-SADS, there is limited information regarding the reliability and internal consistency of this version. Previous studies using this sample, however, found high inter-rater agreement (Graves, 2006; Greenberg et al., 2008). Both Graves and Greenberg et al. reported the correlation between raters on total depressive symptoms severity rating of last week to be .91 for 20% of the sample. In addition, Ambrosini (2000) found inter-rater reliability to be high for the diagnosis of Major Depression, Dysthymic Disorder, Generalized Anxiety

Disorder, Separation Anxiety Disorder, and Oppositional Defiant Disorder in a small sample. Previous versions of the K-SADS have adequate measures of reliability, including inter-rater, internal consistency and test-retest reliability (Ambrosinsi). Chambers and colleagues (1985) found the K-SADS to have acceptable reliability for depressive disorders, which were a little lower but generally similar to studies using the adult version of the interview. In an evaluation of an earlier version, K-SADS-PL, Kaufman and colleagues (1997) found support for the concurrent validity of diagnoses. Inter-rater agreement in diagnosing was high, ranging from 93% to 100%. Additionally, test-rest reliability was high for major depression, bipolar, generalized anxiety, conduct disorder, and oppositional defiant, ranging from .77 to 1.00. Test-retest estimates were considered good for PTSD and ADHD, ranging from .63 to .67. Overall, versions of the K-SADS are reported to a reliable diagnostic tool to use with children and adolescents (Ambrosini; Kaufman et al.).

The present study used a composite score of depression that is a summation of the last week summary ratings for depressive symptoms. Based upon taxometric procedures, depression has been best supported as a dimensional construct, indicating the importance of using varying levels of depression severity instead of presence or absence of a depression diagnosis (Ruscio & Ruscio, 2000). Using a continuous measure of depression, therefore, results in a more powerful research investigation. The total depression scale score was computed based upon the 17 item criteria used by Ambrosini and colleagues. The social withdrawal item was not used because the K-SADS-IVR does not include that item. Additionally, a self-esteem item not included in the K-SADS-IVR depression section was added to the depression scale considering low self-esteem is a

central feature of Dysthymia. This item was adapted from the negative self-image item from the Overanxious Disorder section in the previous version of the K-SADS. Finally, the diurnal mood variation (morning only) and the diurnal variation of fatigue (morning only) were removed from the scale and both anhedonia (loss of interest) and anhedonia (loss of pleasure) were included. These adjustments were made to make the scale more consistent with the specific symptoms used to diagnose depression in children. Similarly, anxiety severity is based upon a composite score across all anxiety disorder symptoms assessed. If a child screened out of a particular anxiety disorder section, a rating of 1 was given for each symptom, indicating the symptom is not present. The current study used continuous, composite scores for measuring both depression and anxiety severity.

The K-SADS-IVR composite scores utilized in this study were found to have acceptable internal reliability in the current sample (see Table 3). Inter-rater reliability was computed on 20% of the 203 interviews (n=42) for the depression summary ratings and anxiety summary ratings. Both Pearson correlations between the original and reliability interviewers' total depression score (r=.93, p< .001) and total anxiety score (r=.94, p<.001) from the last week summary ratings were high and significantly correlated.

Table 3

Summary of Cronbach's α for K-SADS-IVR Depression and Anxiety Scales (N=203)

K-SADS-IVR Scale	α
Depression	.88
Generalized Anxiety Disorder	.92
Post-Traumatic Stress Disorder	.97
Panic Disorder	.89
Separation Anxiety Disorder	.91
Specific Phobia	.82
Social Phobia	.74
Obsessive Compulsive Disorder	.76
Anxiety Composite Total	.92

Symptom Checklist 90-R (SCL-90R; Derogatis, 1994). The SCL-90R is a questionnaire designed to screen for a broad range of psychological problems and symptoms of psychopathology in community, medical, and psychiatric samples (Derogatis, 2000). It is a self-report measure, assessing severity of psychological symptoms in 9 dimensions including: Somatization, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, and Psychoticism. The measure consists of 90 items rated on a 5-point scale (0=not at all, 1=a little bit, 2=moderately, 3=quite a bit, 4=extremely) of symptom distress over the past 7 days. The measure is designed for use with adults and adolescents over the age of 13 with a sixth grade reading level. According to Derogatis, the internal consistency reliabilities range from .77 to .90 for the various scales. Test-retest reliabilities over a 1-week period ranged from .80 to .90. In addition, acceptable interrater reliability has been demonstrated for matching clinical scales. Derogatis indicates over 1,000 studies have been published using the SCL-90R, which demonstrates the high utilization of this instrument.

The current study utilized the SCL-90R to assess maternal depression and anxiety. Both mothers and fathers of the participants were invited to complete the SCL-90R; however, only mother data was used given that a substantially higher number of mothers (n=146) completed the measure compared to fathers (n=63). Two scales, the depression dimension and the anxiety dimension, were used in the current study as well as six additional items from the additional items index. The additional items included were: (1) poor appetite, (2) trouble falling asleep, (3) thoughts of death or dying, (4) awakening in the early morning, (5) sleep that is restless or disturbed, (6) feelings of guilt. These items are separate items on the SCL-90R because they load onto several dimensions and are considered to be clinically significant (Derogotis, 1994). The above items were included in this study in order to be more consistent with the specific symptoms used to diagnose depression and anxiety. A summation of the depression and anxiety dimensions as well as the above additional items was used to determine severity of maternal depression and anxiety. Internal consistency reliabilities for the current sample were acceptable and are as follows: Depression (α=.88), Anxiety (α=.79), and Additional Items (α=.76). The internal consistency reliability for the total summated score used in this study was high (α=.92).

Measure of Negative Life Events

Life Events Checklist (LEC; Johnson & McCutcheon, 1980). The Life Events Checklist assesses major life events over the past 12 months. The LEC is a self-report questionnaire including 46 life event items as well as additional space for the participant to include additional life events if desired. The measure consists of both positive and negative life events from 10 categories of life experiences, including: family health,

family member changes, family moves, money, crises, unexpected news, parents' marital relationship, parent-child relationship, general, and family resources.

Participants are asked to indicate whether each item has "happened to them." If an item occurred, the participant then must indicate whether it was "good" or "bad," and how much effect it had their life (e.g. no effect, some effect, medium effect, big effect). A score of 0, 1, 2, or 3 is assigned relative to the impact the participant indicated of an event. The measure provides information regarding the number of positive and negative events in the past year as well as the severity of the events. Summing the number of positive events and summing the number of negative events, giving each event a weight of one, provides a unit rating. A mean score for the quality of bad events is computed by summing all the item ratings for "bad" events and then dividing that number by the total number of items marked "bad." To capture the number and impact of negative events for participants, this study used the mean score for quality of bad events. The LEC has demonstrated acceptable levels of reliability and validity (Johnson & McCutcheon, 1980). Brand and Johnson (1982) found the test-retest reliability to be adequate over a 2-week interval, especially since an individual may have experienced some of the events during the 2-week interval. The unit rating test-retest correlations were .69 for positive events and .72 for negative events. Impact rating test-retest correlations were .71 for positive events and .66 for negative events. The internal consistency reliability for this sample was acceptable (α=.77).

Measure of Cognitions

Cognitive Triad for Children (CTI-C; Kaslow et al., 1992). This measure is a downward version of the Cognitive Triad Inventory (CTI; Beckham, Leber, Watkins,

Boyer, & Cook, 1986) used with adults. The scale was adapted to make it more developmentally appropriate for children. The CTI-C is a 36- item questionnaire consisting of three scales: view of the self, view of the world, and view of the future. In addition, a total score can be computed by summing the scores for each scale. Each scale consists of 12 items that state a specific thought about the self, world or future. Half of the items are worded in a positive direction and half of the items are worded in a negative direction. Following each item, respondents indicate endorsement of the item by circling *yes*, *maybe,* or *no*. Responses are scored 0, 1, or 2 and are summed after reverse scoring for negative items. Higher summed scores represent a more adaptive cognition.

The CTI-C has demonstrated acceptable internal consistency reliability and convergent and discriminant validity (Kaslow et al., 1992). Kaslow and colleagues found internal consistency estimates were .83 for the self subscale, .69 for the world subscale, .85 for the future subscale, and .92 for the total scale. These findings have been replicated in both children and adolescents. A confirmatory factor analysis of a sample of 122 school-aged children found the internal consistency total score of the CTI-C to be .82 (Zauszniewki, Panitrat, & Youngblut, 1999). In a community sample of 967 adolescents, Greening and colleagues (2005) found the CTI-C to be internally consistent (Cronbach's α= .90), have acceptable test-retest reliability (r=.70), and demonstrate concurrent validity with the CASQ-R (r=.53). Research, however, has failed to extract the three factors from the scale that are consistent with Beck's three-factor model of negative cognitions related to the self, world, and future (Greening et al.; Zausniewski et al.). In addition, research has shown the association between the CTI-C and depressive symptoms in adolescents (Jacobs & Joseph, 1997; Kaslow et al., 1992). This study uses

the CTI-C total score as a measure of cognitive vulnerability of participants. Internal consistency for the CTI-C total score in the current study was high (α=.94).

Measure of Family Environment

Self-Report Measure of Family Functioning-Child Revised (SRMFF-CR; Stark, 2002). The SRMFF-CR is a 40-item self-report measure of the family environment and functioning. The current measure is an adaptation from Bloom's Self-Report Measure of Family Functioning (SRMFF; Bloom, 1985), which is a 75 item questionnaire for adolescents and adults derived from a number of family functioning measures including the Family Environment Scale (Moos & Moos, 1981), the Family Concept Q Sort (Van der Veen, 1965), the Family Adaptability and Cohesion Evaluation Scales (Olson et al., 1985), and the Family Assessment Measure (Skinner et al., 1983). A children's version (SRMFF-C; Stark et al., 1990) was modified from the original measure to make it more developmentally appropriate by simplifying the language, removing double negatives, and simplifying the descriptive anchors to "Never", "Sometimes," and "Always." The current version, SRMFF-CR (Stark, 2002) was modified from the SRMFF-C to eliminate unreliable subscales and items with low factor loadings. In addition, a few items were reworded to be more child-friendly and another subscale, Communication, was added based upon expressiveness and democratic style subscales of the SRMFF-C. The SRMFF-CR consists of 6 subscales based upon the 40 items, which include: Communication, Conflict, Social/Recreational Orientation, Cohesion, Laissez-Faire Style, and Authoritarian Style. Participants are presented with a five-point scale (1=never true, 2=little true, 3=somewhat true, 4=true, 5=very true) to rate each item. The current study used the SRMFF-CR as a measure of family environment.

For the purposes of theoretical relevance, the study used the Communication, Conflict, Social/Recreational Orientation, and Cohesion scales only. Cohesion and Conflict scales were summated and function as a Family Relationship dimension in this study. The following internal consistency reliabilities have been reported for the above scales: Conflict= .74, Communication= .87, Social/Recreation Orientation=.85, Cohesion= .73 (Greenberg et al., 2008). Internal consistency reliabilities for the current sample were found to be acceptable for all scales: Conflict (α=.75), Communication (α=.87), Cohesion (α=.79), and Social/Recreation Orientation (α=.84). Additionally, internal consistency for the Relationship scale (Conflict + Cohesion) used in the present study was found to be acceptable (α=.83).

Procedure

Ethical Considerations

This study complied with the ethical issues and standards of research set forth by the American Psychological Association and the University of Texas at Austin. The principal investigator of the larger study has obtained approval from the Departmental Review Committee within the Department of Educational Psychology and the Institutional Review Board. Prior to the data analysis of this study, the current study was also approved by the Departmental Review Committee within the Department of Educational Psychology and the Institutional Review Board (IRB Protocol #2007-12-0025). The superintendents of the involved school districts have also approved the larger study.

Depressed Sample

A multi-gate screening, identification, and assessment process was used to identify the sample of 154 girls who were experiencing a depressive disorder (see Figure 3). Multiple elementary and middle schools in the two school districts were invited to participate. Fourteen elementary schools and 6 middle schools participated over the 5-year investigation. Following school approval, all girls in the appropriate grades (4-7) were invited to participate in the screening process. Parent permission letters were sent home with 7,737 girls (Appendix K). All girls whose parents provided permission (*n*= 3,436) were invited to participate in the screening. Participants had an assent letter read aloud to them as they read along with it. If the girls agreed to participate in the screening, they were asked to sign the child assent form (Appendix L). Youth in one school district completed the CDI at the time of screening and youth in the other school district completed the CDI and BDI-Y in large groups. The total number of girls screened with the CDI was 3,396. Directions for the measures were read aloud by a graduate student and then participants completed the questionnaires independently.

The first year of the larger study followed a slightly different procedure than the following years for the second gate of the identification and assessment process. In the initial year of the study, participants who scored at least a 16 on the CDI (*n*=127) were administered a second CDI one week later as the second gate of screening. Those who scored above the cut-off on the second administration of the CDI were invited to participate in the K-SADS-IVR (*n*=87). In order to reduce the time lapse between screening and treatment and the number of false positives, the larger investigation

changed to using the DSM Interview instead of the second CDI for the remainder of the investigation.

Starting the second year of the project, if the participant scored at least the cut-off on either measure, CDI=16 and BDI-Y=25, they completed an individual DSM Interview with a trained graduate student the same day. Participants were immediately interviewed if they endorsed suicidal ideation on the screening measures. Forty-eight girls (1.4%) endorsed the suicidal ideation item, "I want to kill myself" on the CDI. For girls whose score on the measure(s) was below the cut-off, a letter was sent home to the parents thanking them for participation (*n*=2,670; Appendix M). A total of 726 girls were interviewed using the DSM Interview. The DSM interviewer assessed whether the participant was experiencing a depressive disorder based on the symptoms she reported experiencing. If the interviewer believed the participant might meet diagnostic criteria for depression, he/she recommended the child for an interview with the K-SADS-IVR. A total of 505 girls were invited to participate in the K-SADS-IVR. If depression was ruled out based on the DSM Interview, a feedback letter was sent home to the parent(s) (*n*= 221; Appendix N). For girls invited to participate in the diagnostic interview, the DSM interviewer called the primary caregiver who gave consent for the screening to provide feedback regarding their child's responses to the questionnaire(s) and the DSM interview. In addition, the interviewer established rapport with the parent, addressed safety concerns, explained the project, and alerted the parent to the second consent letter.

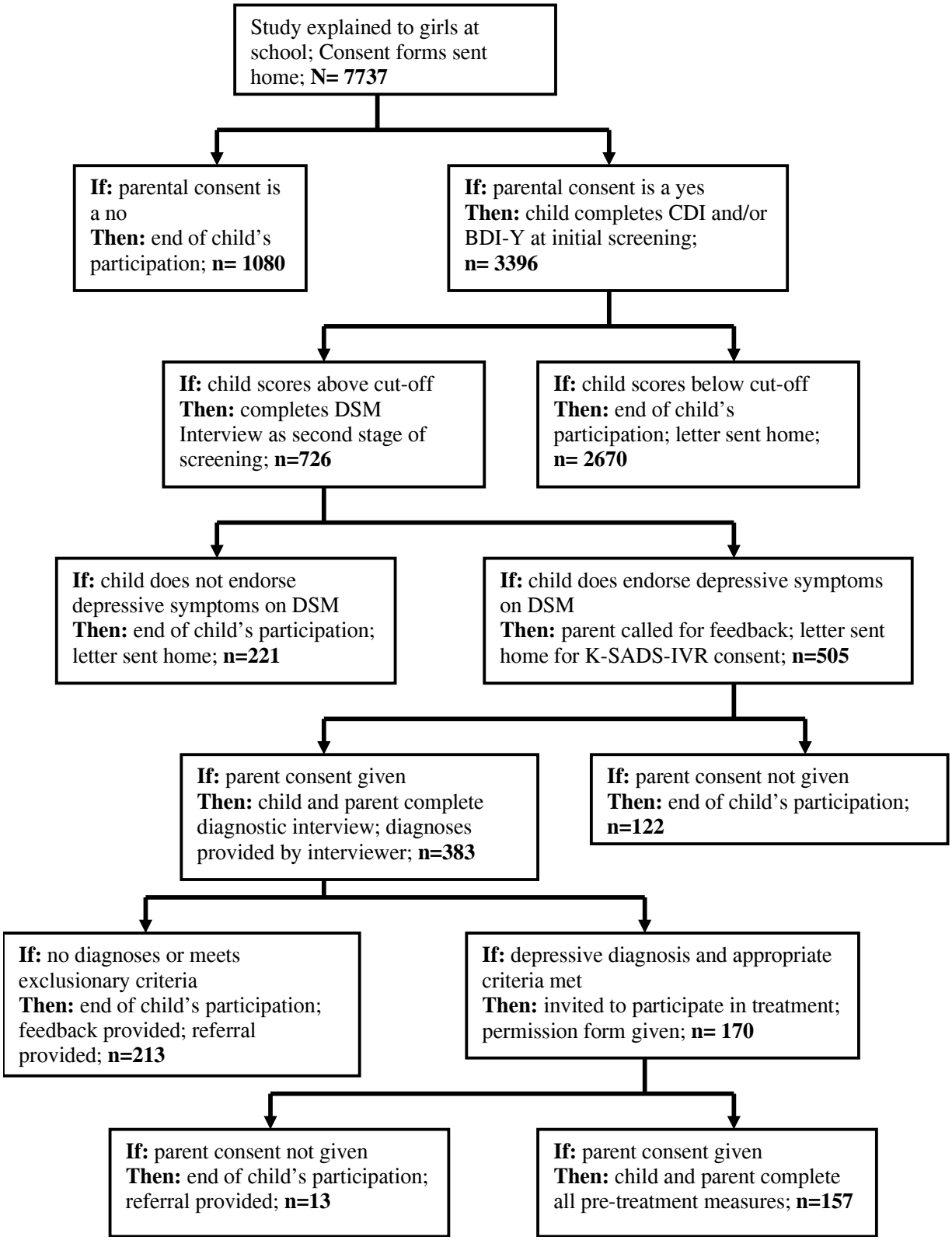
A second consent form including a description of the diagnostic interview was sent home for the parents to read and sign to complete the third gate in the screening and assessment process (Appendix O). Once consent was obtained, participants were

interviewed at their school and the caregivers were interviewed over the telephone, at home, or at school depending on their preference. Parental permission for the diagnostic interview was obtained for 383 girls. Parental permission was not granted or consent letters were not returned for 122 girls. Participant interviews were audio taped and 20% of the interviews were randomly picked for computation of inter-rater reliability. When there were differences in symptom ratings or diagnoses, the interviewers met and worked out consensus ratings and diagnoses. This was uncommon and only occurred once in the sample.

Based on the diagnostic interview, participants were invited to participate in treatment if they had a primary depressive disorder and met all other study requirements. Of the 383 interviews, 170 were recommended for participation in treatment and 213 did not meet criteria or were excluded. One hundred fifty-seven girls participated in the study.

Therapists in the larger study met with the girls and their caregiver(s) to go over the treatment consent forms and explain the treatment process (Appendix P). Once treatment consent was obtained, participants completed pre-treatment measures, including the LEC, CTI-C, and SRMFF-CR. The caregivers completed pre-treatment measures as well, including the SCL-90R. The girls and their caregivers completed these questionnaires in small groups at the girls' school. The measures were presented in counter-balanced order across groups to reduce ordering effects. All the above assessment procedures were completed by trained and supervised graduate students.

Figure 3. Flowchart of Multiple Gate Screening Process



Control Sample

The participants from the control sample were recruited from elementary and middle schools in the same school districts as the depressed sample. Graduate students went to classrooms to describe the procedure and sent home consent letters with the students (Appendix Q). Each participating family received $50 as compensation for their participation. Once consent was obtained, graduate students met with individual families to complete the measures. Daughters completed the CTI-C, CDI, SRMFF-CR, and K-SADS-IVR, and the caregiver completed the K-SADS-IVR and SCL-90R. Parent and child measures and interviews were completed independently. All interviews were audio taped for reliability and supervision purposes. These measures were completed at the family's home. Participation time varied but was approximately two hours.

Training of Measures Administrators

All individuals who administered the measures were doctoral students with at least one year of experience on the larger research study. They were trained and supervised by the project coordinator and/or the principal investigator. Instructions for administering and scoring each measure were provided. When administering measures to children, a minimum of one administrator was trained in suicidal risk assessment.

Training of Interviewers

The training of interviewers for the administration of the K-SADS-IVR was extensive. All diagnostic interviewers were doctoral students who completed a minimum of one year of training in the doctoral program in School Psychology. An advanced doctoral student under the supervision of the principal investigator and who had expertise in the area of child psychopathology and administering semi-structured interviews,

trained interviewers for approximately six months. As part of the training, interviewers were required to listen to a minimum of six previously audio taped interviews and practice rating symptoms. Additionally, interviewers in training practiced the K-SADS-IVR with volunteers and the trainer during a weekly training meeting. Interviewers in training observed at least one senior interviewer conducting the K-SADS-IVR in person. General interviewing skills, both micro and macro, were also addressed in training. Overall, interviewers had approximately 50 hours of training prior to conducting independent interviews.

Interviewers in training needed to demonstrate competence in the K-SADS-IVR ratings prior to conducting independent interviews. Interviewers were considered reliable raters when they were able to listen to an audio taped K-SADS-IVR and correctly identify the presence, absence, and severity of depressive symptoms. If they inaccurately identified the presence or absence of one or more symptoms, they were given more training until they reached this level of accuracy. Once the trainer believed these requirements were met, interviewers conducted their initial interview under live supervision of a senior interviewer. All interviewers received weekly group supervision as well as individual supervision as needed. The lead interviewer also reviewed tapes and provided feedback for all interviewers. Beginning interviewers' tapes were reviewed more frequently. Inter-rater reliabilities for the last week summary ratings for the total depression score (r=.93, p<.001) and total anxiety score (r=.94, p<.001) were high in this sample.

CHAPTER 4

Statistical Analyses

The current investigation examined the direct effects of anxiety symptoms and the cognitive triad on the severity of depression in early adolescent girls as well as the direct effects of negative life events, maternal symptoms of depression and anxiety, and family environment variables on the cognitive triad and severity of anxiety. Utilizing path analytic techniques, the present study tested the indirect effects of family functioning, maternal symptoms of depression and anxiety, and negative life events on depressive symptom severity. Descriptive statistics are outlined first, followed by preliminary analyses. The results for each of the hypotheses are presented and an exploratory analysis is discussed.

Descriptive Statistics

Means, standard deviations, sample sizes, Cronbach's alphas, and correlation coefficients were calculated for the main variables in the analysis and presented in Table 4. All analyses use the total sample (N=203), which is a combination of the control sample and depressed sample discussed in the previous chapter.

Table 4

Means, Standard Deviations, Sample Sizes, Cronbach's Alphas, and Pearson Product Correlations among Variables in the Path Analysis Model

Variable	SCL-90R	Family-Rel	Family-Com	Family – S/R	CTI - C	LEC	Dep Total	Anx Total
SCL-90R	1.00							
Family-Rel	-.13	1.00						
Family-Com	-.12	.60**	1.00					
Family –S/R	-.11	.54**	.63**	1.00				
CTI-C	-.10	.46**	.48**	.48**	1.00			
LEC	.20*	-.23**	-.17*	-.08	-.22**	1.00		
Dep Total	.17*	-.22**	-.24**	-.30**	-.46**	.09	1.00	
Anx Total	.04	-.05	-.12	-.15*	-.19**	.07	.53**	1.00
M	18.93	38.00	14.98	21.14	51.88	2.75	33.83	1.90
SD	14.94	9.72	7.74	8.06	13.87	.92	10.76	.06
N	146	203	203	203	203	202	203	203
α	.92	.83	.87	.84	.94	.77	.88	.92

Note. **Represents significance at the .001 level. *Represents significance at the .05 level. SCL-90R= Maternal Depression and Anxiety; Family Rel= SRMFF-CR Family Relationship Scale; Family Com= SRMMF-CR Communication Scale; Family-S/R= SRMFF-CR Family Social/Recreational Scale; CTI-C= Cognitive Triad Inventory for Children Total Score; LEC= Life Events Checklist Mean Score; Dep Total= K-SADS-IV Depression Scale Total; Anx Total= K-SADS-IV Anxiety Scales Converted Log Score.

Preliminary Analyses

Demographic Variables and Total Depression Score

A series of analyses were conducted to determine whether the total K-SADS-IVR depression score was related to age, race/ethnicity, or grade. The correlation between age and total K-SADS-IVR depression score was not statistically significant (r=-.04, p=.54). Analysis of variance (ANOVA) results suggested the total depression scores were not

significantly different by racial/ethnic groups, $F(4, 198)= 1.94$, $p=.11$, or grade, $F(4, 198)= 1.59$, $p=.38$.

Violation of Normality

Past research has primarily used diagnoses, a dichotomous variable, from the K-SADS-IVR interview rather than an interval scaled score. The present study, however, uses scale scores as measures of anxiety and depression. The K-SADS-IVR depression scale scores used in the study are assumed normal, with estimates of skewness (.19) and kurtosis (-.58) being acceptable (see Figure 4). On the other hand, the total anxiety scale scores had high estimates of skewness (2.30) and kurtosis (8.81) (see Figure 5; West, Finch, & Curran, 1995). A log transformation, therefore, was utilized to help normalize the data. Based on log transformation data, estimates of skewness (1.50) and kurtosis (3.02) for the total anxiety scale scores were acceptable (Kline, 2005). The log transformed scores for total anxiety symptoms were used in the main analyses of this study.

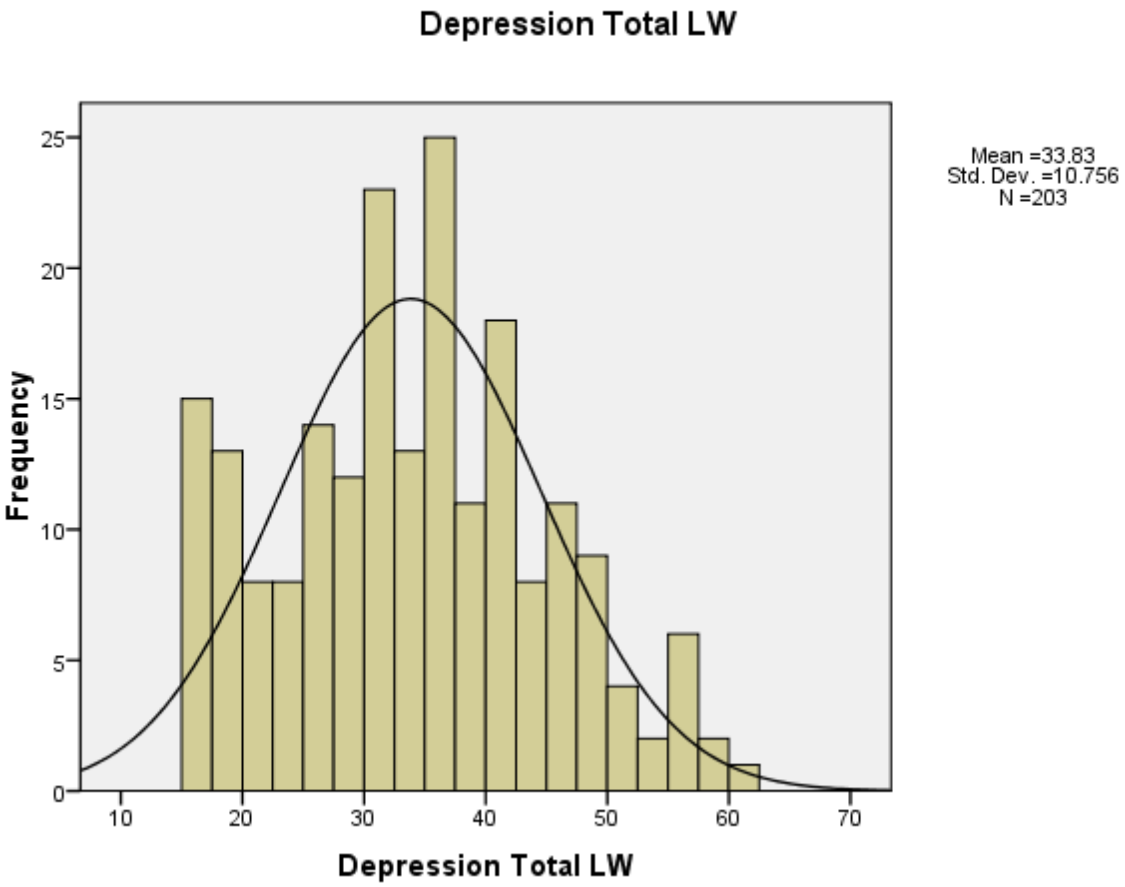


Figure 4. Histogram of total depression scale scores on the K-SADS-IVR

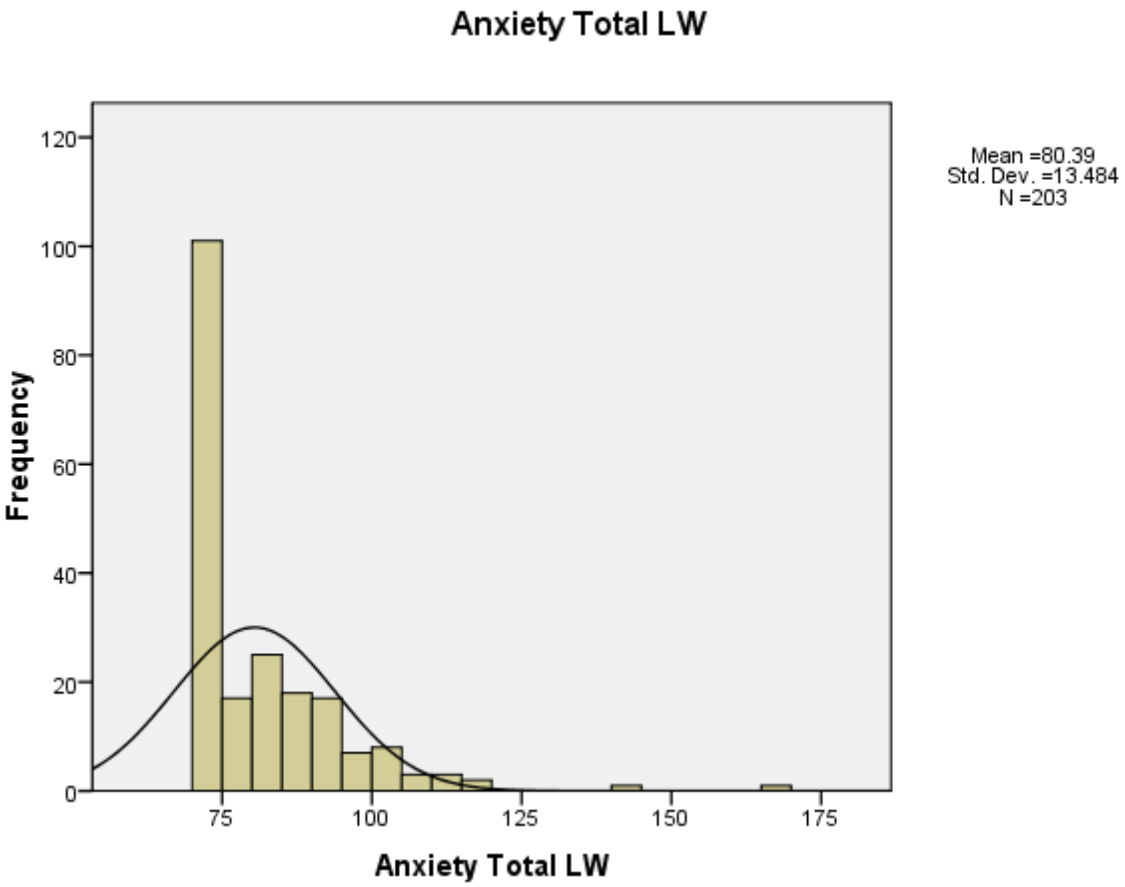


Figure 5. Histogram of total anxiety scale scores on the K-SADS-IVR

Missing Data

Maternal data were not completed for a significant number of participants (*n*=57) despite extensive efforts by research staff to collect this data. In addition, only 4% of the maternal data is missing for the control group, whereas, 36% of the maternal data is missing for the depressed sample. The discrepancy in completion rates is likely attributable to differences in data collection as discussed in the previous chapter.

Analyses were conducted to determine if there were significant mean differences between participants whose mother completed the data and participants whose mother did not complete data on the variables in the model. Only the depression group was included in this analysis to account for differences in data collection. A series of independent samples *t*-tests were conducted to determine whether there were significant differences in the cognitive triad, reports of family environment, negative life events, anxiety scores, and depressive scores between the two groups. Results are presented in Table 5. None of the results were statistically significant suggesting there were no significant differences between participants' reports of cognitive vulnerability, family environment, negative life events, anxiety symptoms and depressive symptoms for girls whose mother completed the SCL-90R and girls whose mother did not in the depressed sample.

Full information maximum likelihood estimation was used to deal with missing data. Maximum likelihood based procedures are recommended for dealing with missing data at random and is considered superior to other methods (Keith, 2006; Schafer & Graham, 2002).

Table 5

T-Tests of CTI-C Total Score, K-SADS-IVR Depression Total, K-SADS-IVR Anxiety Total, LEC Total and SRMFF-CR Subscales by Parent Participation for the Depressed Sample (n=154)

Variables	*t*	*p*
CTI-C Total	-.31	.76
Anxiety Total	.63	.53
Depression Total	.71	.48
LEC- Negative Life Events	-.73	.90
SRMFF- Relationships	-.49	.78
SRMFF-Communication	-1.14	.49
SRMFF-Social/Recreational	-.73	.89

Main Analyses

All analyses were performed using SPSS version 16.0 (SPSS, Inc., Chicago IL) and Amos 16.0 (Analysis of Moment Structures; Arbuckle, 2007). Amos uses a graphic approach allowing researchers to draw the path diagram and analyze it using maximum likelihood estimation (Keith, 2006). The current analysis examined the direct effects of anxiety symptoms and the cognitive triad on severity of depression in early adolescent girls as well as direct effects of negative life events, maternal symptoms of anxiety and depression, and family environment variables on the cognitive triad and severity of anxiety. Additionally, the analysis examined the indirect effects of negative life events, maternal symptoms of anxiety and depression, and family environment variables on depressive symptom severity. See Figure 6 for the hypothesized path analysis model.

The hypothesized model is an over-identified, recursive, path model. As shown in Figure 6, observed variables are typically represented by rectangles. Paths are drawn

using arrows from each variable to any other variable that it may affect directly. Exogenous variables in the model do not have an arrow pointing to them. As shown in Figure 6, exogenous variables in the proposed model include negative life events, maternal symptoms of depression and anxiety, and family environment variables. The curved and double-headed arrows between the exogenous variables, as shown in Figure 6, represent covariances. Variables affected by other factors in the model are called endogenous variables and have arrows pointing toward them. In the hypothesized model (see Figure 6), the endogenous variables include the girls' cognitive triad, anxiety symptoms, and depressive symptoms. Error variances, or disturbances, are variables relating to the endogenous variables that represent all other influences from outside the model as shown in Figure 6.

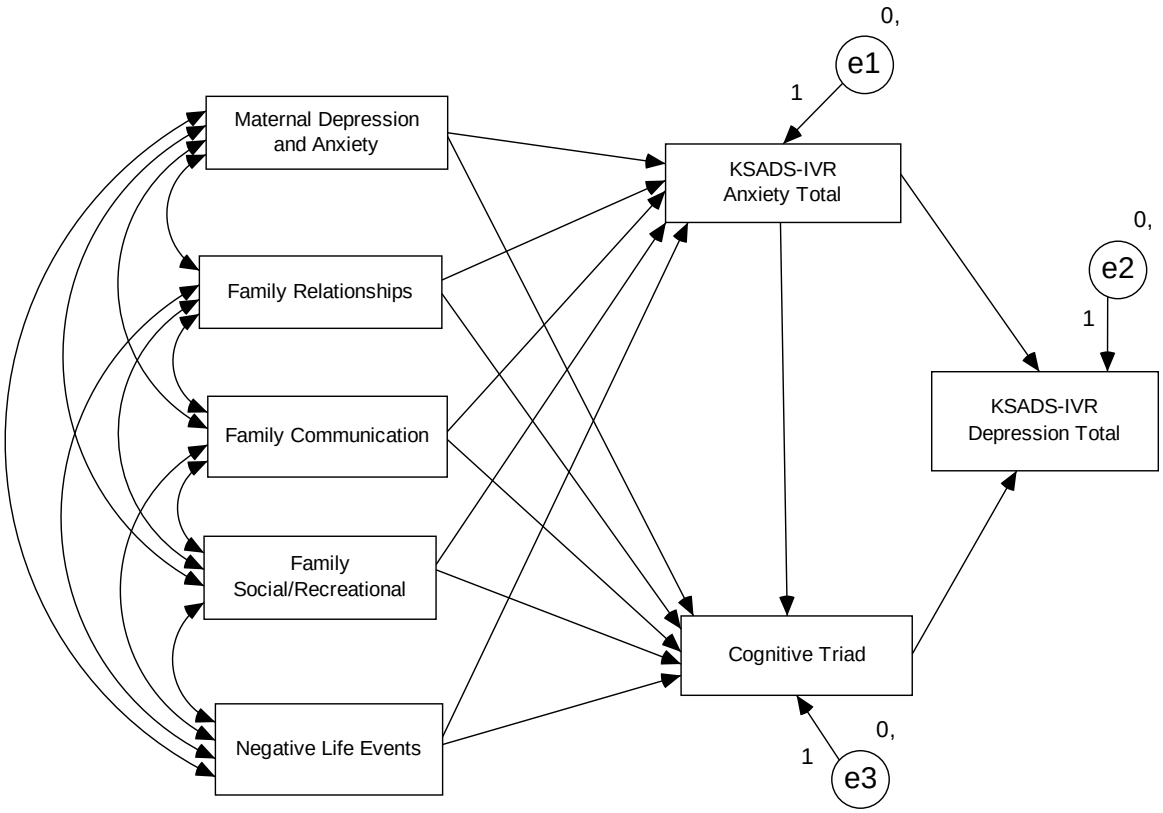


Figure 6. Hypothesized Path Model

Currently, there is no consensus in the literature regarding the appropriate sample size to use in structural equation modeling. In general, the larger the sample size, the better (Keith, 2006). According to Kline (2005), sample sizes over 200 in SEM are considered large. The sample for this study is 203, which may be considered medium to large by Kline's standards. The complexity of the model, however, is important to consider when determining the sample size needed to achieve adequate power. In other words, the more complex the model is (i.e. a large number of parameters), the more subjects are needed to obtain adequate power. Kline suggests the minimum requirement

is a ratio of 5:1 for the number of subjects to parameters in a research design. Given the number of parameters in the hypothesized path analysis, the sample size for this study is adequate based upon the minimum ratio requirement.

In sum, path analysis was used to evaluate the direct effects of anxiety symptoms and cognitive triad on the severity of depression in early adolescent girls, the direct effects of negative life events, maternal symptoms of depression and anxiety, and family environment variables on the cognitive triad and severity of anxiety, and the indirect effects of family functioning, maternal symptoms of depression and anxiety, and negative life events on depressive symptom severity. Bootstrapping, which takes repeated, random samples from the existing sample, was used to determine the significance of the indirect effects after missing maternal data was imputed through regression imputation (Arbuckle, 2007). MacKinnon, Fairchild, and Fritz (2007) recommend bootstrapping for testing mediation effects and consider it to be more accurate than traditional mediation analysis because this method does not require as many assumptions as other tests.

Path Analytic Model Fit

Model fit was evaluated using several measures of fit. The chi-square (χ^2), in addition to degrees of freedom and probability, were examined. In SEM, a small χ^2 and one that is not statistically significant is preferred and suggests the model and data are consistent with each other. Given that χ^2 is sensitive to sample size (Keith, 2006), additional measures of fit, the comparative fit index (CFI), the Tucker-Lewis index (TLI), and the Root Mean Square of Approximation (RMSEA) were examined. The TLI and CFI fit indices compare the fit of the existing model with that of the null, or independence model (Keith). According to Hu and Bentler (1999), values approaching

1.0 for the CFI and TLI are a better fit, with values above .95 representing a good fit and values above .90 representing an adequate fit of the model to the data. The RMSEA assesses how well the model reproduces the sample data without comparison to a reference model (Hu & Bentler). Keith (2006) suggests RMSEA values below .05 indicate a good fit, values below .08 represent a reasonable fit, and values above .10 represent a poor fit.

The indices of fit for the hypothesized model indicate an excellent fit of the model to the data with χ^2 [4, *N*=203]=4.850, *p*=.434, CFI=1.00, TLI=1.00, and a RMSEA of .00. See Figure 7 for path-analytic model with standardized coefficients.

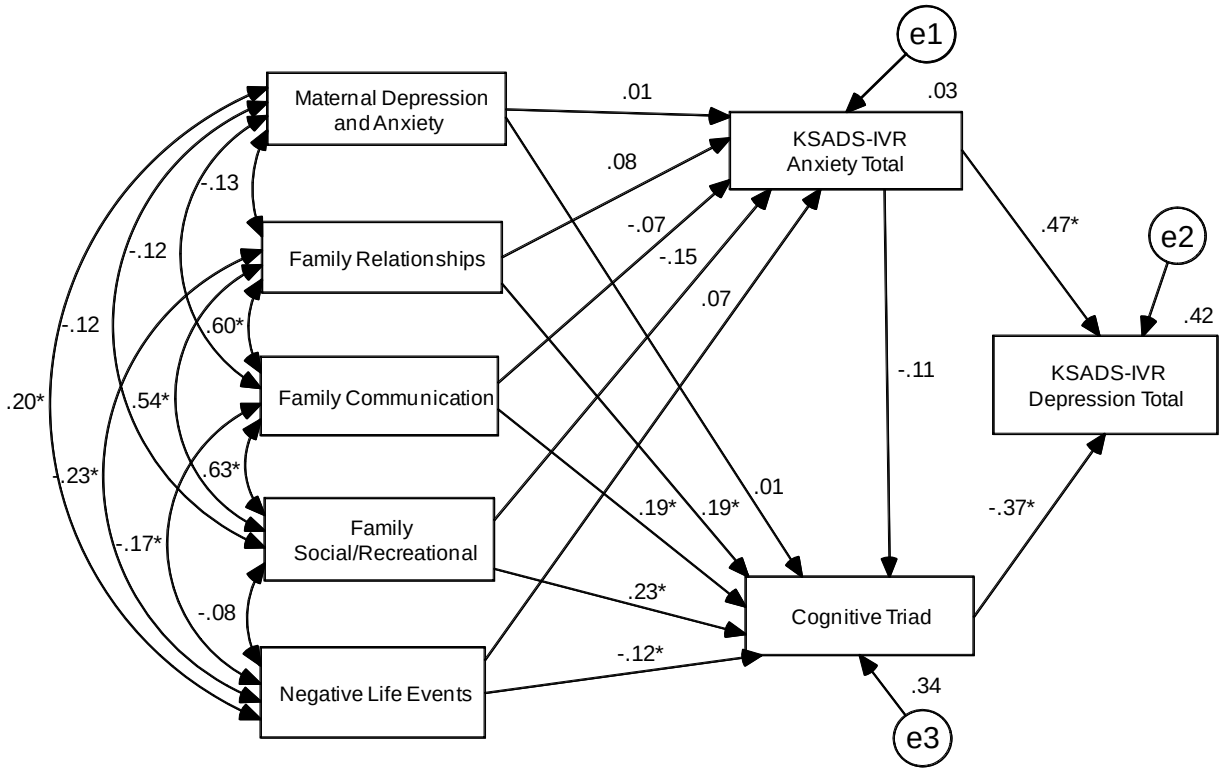


Figure 7. Path-Analytic Model with Standardized Coefficients . * Represents significant pathways

Results for Hypotheses 1 to 5, testing the path analytic model shown in Figure 7, are presented below.

Hypothesis 1

Hypothesis 1 predicted participants' report of depressogenic cognitions, as measured by the CTI-C, would directly affect the severity of her depressive symptoms. Specifically, lower scores on the total score of the CTI-C would be associated with higher scores on the composite depressive symptoms scale of the K-SADS-IVR.

Results indicate a statistically significant negative direct effect from participants' cognitive triad to the severity of depressive symptoms (β=-.37, *b*=-.29, *p*<.001).

Hypothesis 2

Hypothesis 2 predicted participants' anxiety symptoms, as measured by the K-SADS-IVR, would have a direct effect on her cognitive triad and depressive symptom severity. Specifically, a higher composite score of anxious symptomatology on the K-SADS-IVR would be associated with a more depressogenic cognitive triad (i.e. lower scores on the total score of the CTI-C) and higher scores on the composite depressive symptoms scale of the K-SADS-IVR.

Results indicate a statistically significant positive direct effect from participants' anxiety symptoms to depressive symptoms (β=.47, *b*=77.76, *p*<.001). There was not a statistically significant direct effect from participants' anxiety symptoms to her cognitive triad (β=-.11, *b*=-23.40, *p*=.06). The indirect effect from the anxiety symptoms to participants' depressive symptoms was not significant (β=.04, *b*=6.69, *p*=.10).

Hypothesis 3

Hypothesis 3 predicted mothers' reports of symptoms of depression and anxiety, as measured by the SCL-90R, would directly affect the severity of the daughters' anxiety symptoms and cognitive triad. In addition, there would be an indirect association between mothers' reports of symptoms of depression and anxiety and the daughters' depressive symptoms via the daughters' anxiety symptoms and cognitive triad. In other words, higher scores summated from the depression and anxiety symptom scales of the SCL-90R as reported by the mothers would be significantly associated with higher scores on the composite anxiety symptoms scale on the K-SADS-IVR and lower total scores on the daughters' CTI-C. Higher anxiety symptoms and a more negative cognitive triad would

then be associated with a higher composite depressive symptoms scale of the K-SADS-IVR.

Results indicate there was not a significant direct effect from maternal depressive and anxiety symptoms to daughter's cognitive triad (β=.01, *b*=.01, *p*=.84) or to anxiety symptoms (β=.01, *b*=.00, *p*=.91). The indirect effect from maternal depression and anxiety to participants' depressive symptoms was not significant (β=.00, *b*=.00, *p*=.96).

Hypothesis 4

Hypothesis 4 predicted participants' reports of the family environment, as measured by the SRMFF-CR, would directly affect her anxiety symptoms and cognitive triad. Specifically, more cohesive and less conflictual family relationships, more communication within the family, and higher levels of family engagement in social recreational activities would be associated with a lower composite score of anxiety symptoms and a more positive cognitive triad (i.e. higher score on the total score of CTI-C). In the hypothesized model, family environment variables were predicted to have significant indirect effects on depressive symptoms in the girls. Thus, the family environment would indirectly influence depressive symptoms via the participants' cognitive triad and anxiety symptoms.

Results indicate there were significant positive direct effects from family social/recreational activities (β=.23, *b*=.40, *p*<.01), family communication (β=.19, *b*=.34, *p*<.05), and family relationships (β=.19, *b*=.27, *p*=.01) to the cognitive triad. There were no significant direct effects from family social/recreational activities (β=-.15, *b*=-.001, *p*=.11), family communication β=-.07, *b*=-.001, *p*=.50), and family relationships (β=.08, *b*=.001, *p*=.36) to anxiety symptoms. Results indicate the indirect effect from family

social/recreational activities to depression was significant (β=-.16, *b*=-.22, *p*=.02). The indirect effects from family communication (β=-.10 *b*=-.14, *p*=.11) and family relationships (β=-.03, *b*=-.03, *p*=.66) to depression were not significant.

Hypothesis 5

Hypothesis 5 predicted participants' report of negative life events, as measured by the LEC, would directly affect her anxiety symptoms and her cognitive triad. Specifically, a higher number of negative life events, as determined by ratings of type of event and impact of event, would be associated with a higher composite score of anxiety symptoms and a more negative cognitive triad (i.e. lower score on the total score of CTI-C). In the hypothesized model, participants' negative life events were predicted to have a significant indirect effect on depressive symptoms. Thus, negative life events would indirectly influence depressive symptoms via the participants' cognitive triad and anxiety symptom severity.

Results indicate there was a significant negative direct effect for negative life events on the cognitive triad (β=-.12, *b*=-1.77, *p*=.05) but there was not a significant direct effect on anxiety symptoms (β=.07, *b*=.01, *p*=.34). Results indicate a significant indirect effect from negative life events to depression (β=.08, *b*=.92, *p*=.02).

Figure 8 presents the path analytic model with only significant pathways. Table 6 presents a summary of the direct and indirect effects.

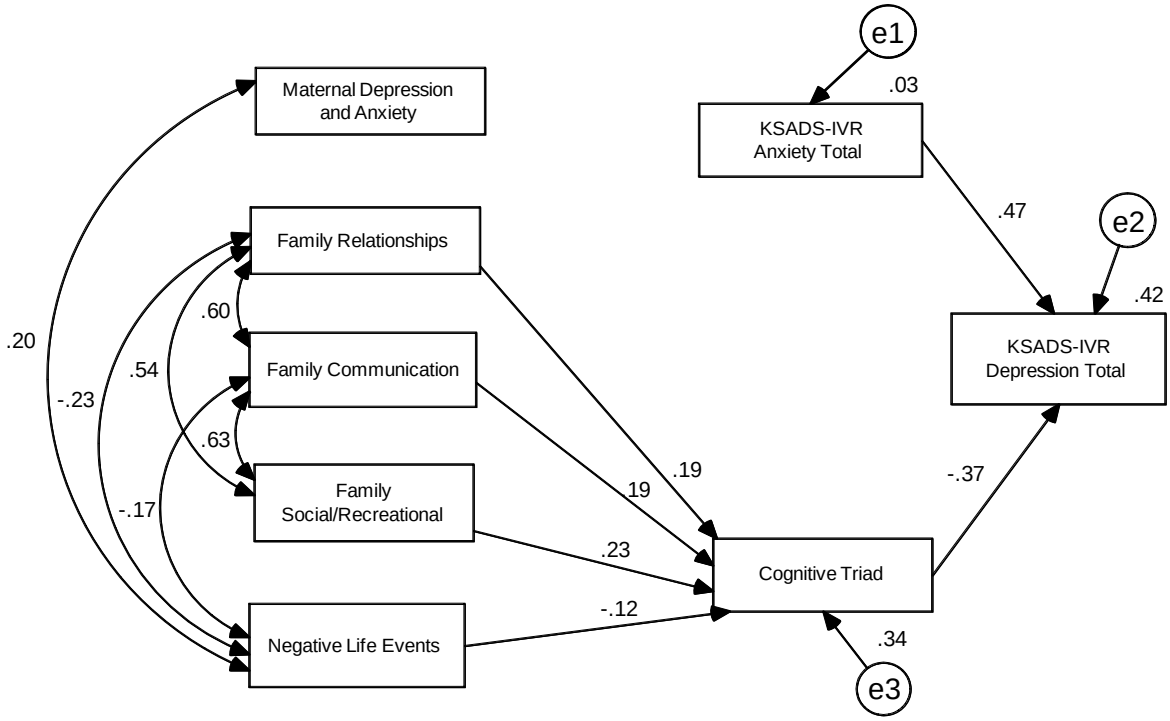


Figure 8. Final path analytic model with statistically significant pathways only

Table 6

Summary of Standardized (β) and Unstandardized (b) Estimates and Statistical Significance (p) for Hypotheses 1-5 shown in the Path-Analytic Model in Figure 7

Variable	β	*b*	*p*
CTI-C: Cognitive Triad			
Direct Effect on Depression	**-.37**	**-.29**	**<.001**
K-SADS-IVR: Anxiety			
Direct Effect on Depression	**.47**	**77.76**	**<.001**
Direct Effect on Cognitive Triad	-.11	-23.40	.06
Indirect Effect on Depression	.04	6.69	.10
SCL-90R: Maternal Anxiety and Depression			
Direct Effect on Cognitive Triad	.01	.01	.84
Direct Effect on Anxiety	.01	.00	.91
Indirect Effect on Depression	.00	.00	.96
SRMFF-CR: Family Social/Recreational			
Direct Effect on Cognitive Triad	**.23**	**.40**	**.003**
Direct Effect on Anxiety	-.15	-.00	.11
Indirect Effect on Depression	**-.16**	**-.22**	**.02**
SRMFF-CR: Family Communication			
Direct Effect on Cognitive Triad	**.19**	**.34**	**.02**
Direct Effect on Anxiety	-.07	-.00	.50
Indirect Effect on Depression	-.10	-.14	.11
SRMFF-CR: Family Relationships			
Direct Effect on Cognitive Triad	**.19**	**.27**	**.01**
Direct Effect on Anxiety	.08	.00	.36
Indirect Effect on Depression	-.03	-.03	.66
LEC: Negative Life Events			
Direct Effect on Cognitive Triad	**-.12**	**-1.77**	**.05**
Direct Effect on Anxiety	.07	.01	.33
Indirect Effect on Depression	**.08**	**.92**	**.02**

Note. Significant pathways are bolded.

Exploratory Analysis: Anxiety as a Moderator for the CTI-C to Depression Path

The following analysis was included to build upon the previous results and provide additional information regarding how anxiety may influence the participants' cognitive triad and severity of depressive symptoms. The discussion will review the findings of this exploratory analysis, how it relates to the original hypothesized model, and the relevance to previous research in the field.

The original analysis found anxiety to have a significant direct effect on depressive severity but that anxiety was not significantly related to life stressors. The original analysis, however, did not examine whether anxiety may serve as a possible moderator. Beck's cognitive model of psychopathology suggests disorders may be differentiated by the content of their thinking, referred to as the cognitive content-specificity hypothesis (Beck, 1976). According to Beck's theory, cognitions associated with anxiety are more related to threat, whereas, depressive cognitions are associated with loss. Previous research has found partial support for this hypothesis with depressed children in grades 4 to 7 endorsing more depressive cognitions than children with anxiety (Laurent & Stark, 1993). Based upon the cognitive content-specificity hypothesis, it is possible the relation between the cognitive triad and depressive severity may be stronger for children without anxiety symptoms than children with anxiety symptoms. In order to examine whether anxiety moderates the effect of the cognitive triad on depressive severity, multi-sample analyses were utilized by analyzing a model of depression separately for children with minimal to no anxiety symptoms and children with anxiety symptoms and then comparing the effect of the CTI-C on depression for the two groups.

Anxiety severity was removed from the path model in the original analysis because it is the variable on which the sample is divided into subsamples.

The sample was divided into two groups based upon severity of anxiety symptoms. The histogram for anxiety severity (see Figure 5) seemed to indicate evidence for two distinct groups, one group with no or minimal anxiety symptoms (<75) and one group with anxiety symptoms ($\geq$75). Additionally, a cut-off score of 75 also divided the sample into two equal groups. The first group reported either no anxiety symptoms (70) or minimal anxiety symptoms (71-74) (n=101). The second group, those with anxiety symptoms, had a score of 75 or above (n=102). Means, standard deviations, sample sizes, Cronbach's alphas and correlation coefficients were calculated for the main variables in the analysis by group and presented in Table 7.

Table 7

Means, Standard Deviations, Sample Sizes, Cronbach's Alphas, and Pearson Product Correlations among Variables for the Exploratory Analysis

Variable	SCL-90R	Family-Rel	Family-Com	Family – S/R	CTI -C	LEC	Dep Total
SCL-90R	1.00 (1.00)						
Family-Rel	.04 (-.24*)	1.00 (1.00)					
Family-Com	.05 (-.21)	.54** (.64**)	1.00 (1.00)				
Family –S/R	-.14 (-.05)	.56** (.52**)	.63** (.60**)	1.00 (1.00)			
CTI-C	.09 (-.27*)	.41** (.50**)	.47** (.45**)	.47** (.44**)	1.00 (1.00)		
LEC	.04 (.30**)	-.17 (-.26**)	-.19 (-.15)	-.06 (-.07)	-.23* (-.19)	1.00 (1.00)	
Dep Total	-.19 (.38**)	-.06 (-.32**)	-.12 (-.22*)	-.22* (-.25*)	-.24* (-.55**)	.12 (.03)	1.00 (1.00)
M	20.70 (17.48)	37.05 (38.96)	13.52 (16.45)	19.41 (22.89)	48.35 (55.45)	2.81 (2.68)	38.99 (28.62)
SD	14.62 (15.14)	9.27 (10.12)	7.49 (7.75)	7.77 (8.00)	13.98 (12.87)	.80 (1.04)	9.22 (9.66)
N	66 (80)	102 (101)	102 (101)	102 (101)	102 (101)	102 (101)	102 (101)
α	.90 (.92)	.80 (.86)	.86 (.87)	.79 (.83)	.94 (.94)	.76 (.77)	.79 (.87)

Note. **Represents significance at the .001 level. *Represents significance at the .05 level. No Anxiety Group coefficients in parentheses.

To determine if there was a significant difference in the direct effect of the cognitive triad to depression, multi-sample analyses were conducted using the chi-square difference test to assess whether the path was significantly different between groups in the sample. In Step 1, the initial model was unconstrained, where paths were allowed to differ between the two groups. For Step 2, the first model was then compared to a second model in which all paths but the path from cognitive triad to depression were constrained. In Step 3, all paths in the model were constrained, including the path from the cognitive triad to depression. The model in Step 3 was compared to the model in Step 2. Results of the chi-square difference test are shown in Table 8.

Table 8

Chi-Square Test of Significance for Exploratory Analysis

Step	Model	χ^2	*df*	$\Delta\chi^2$	Δ *df*	*p*	RMSEA	CFI
1	Initial Multi-sample Model	20.043	10				.07	.97
2	Constrained Paths: Family Variables, Maternal Depression & Anxiety, Negative Life Events to Cognitive Triad	24.041	15	3.998	5	.55	.06	.97
3	Constrained Paths: All paths from Step 2 plus Cognitive Triad to Depression	31.711	16	7.67	1	<.01	.07	.95

The results from Table 8 suggest the initial model may be considered a reasonable fit of the data according to the CFI. Given the CFI supports the fit of the model to the data, the model in Step 2 is compared to the initial model. When the path from cognitive triad to depression was unconstrained and the other pathways were constrained across the two groups, the change in chi square was not significant. Thus, the pathways from family variables, maternal depression and anxiety, and negative life events to the cognitive triad did not significantly differ between the two groups. In Step 3, when all pathways are constrained including the path from the cognitive triad to depression, there was a statistically significant decrement in model fit as demonstrated by the change in chi-square. This finding suggests that anxiety moderated the relation between the cognitive triad and depression, with a stronger effect for the group with little to no anxiety symptoms than the group with anxiety symptoms (no anxiety: β=-.55, b=-.41, p<.001; anxiety: β=-.24, b=-.16, p=.01). In other words, the path from the cognitive triad to depression was significantly different for the two groups.

CHAPTER 5

Discussion

Overview of Findings and Integration with Previous Research

Overall, results of this study replicated and built upon previous literature examining specific risk factors of childhood depression. The findings of this study support a cognitive-stress model of childhood depression, specifically finding family environment variables and negative life events were significant pathways to the cognitive triad. Furthermore, a more depressogenic cognitive triad was significantly associated with higher depressive severity. Additionally, both family social/recreational activities and negative life events had a significant indirect effect on depression. Maternal symptoms of depression and anxiety were not found to have significant direct effects in the model. Results also support previous literature, finding a strong relation between anxiety and depression in girls. On the other hand, maternal depression and anxiety, family environment variables, and negative life events were not significantly associated with anxiety symptoms. Lastly, exploratory results found anxiety severity was a moderator for the relation between depressogenic cognitions and depression. The results of this study have significant implications for understanding correlates of depression in early adolescent girls. This study supports a cognitive-stress model of depression, emphasizing the relation between the cognitive triad and depression as well as highlighting the direct effects family environment and negative life events have on depressogenic cognitions. Furthermore, the findings of this study stress the importance of including comorbid

anxiety in models of depression in children given its strong relation to depression. Each specific finding and how it relates to previous literature are presented in more detail in this section.

Cognitive Triad to Depression

As hypothesized, the results indicated a strong relation between the cognitive triad and severity of depressive symptoms. In other words, girls who reported more negative beliefs about the self, world, and future presented with more severe depressive symptoms as assessed on a diagnostic interview. This finding is consistent with previous literature examining Beck's theory of depression. Depressogenic cognitions have been found to be associated with an increase in depressive symptoms in multiple studies (e.g. Abela & D'Allesandro, 2002; Hankin et al., 2004; Joiner et al., 1999; Kendall et al., 1990; McDermut et al., 1997; Stark et al., 1996). Depressogenic cognitions have been shown to be related to depressive symptoms in various populations, including adults (Jolly et al., 1994; Lewinsohn et al., 1988), adolescents (Garber et al., 1993; Jolly, 1993), and children (Epkins, 1996; Kendall et al., 1990; Stark et al., 1996). Furthermore, cognitive vulnerability has been found to be a specific risk factor for developing depression in longitudinal studies (Alloy et al., 1999; Lewinsohn et al., 2001). One advantage of this study is the majority of previous studies assessing the relation between cognitive vulnerability and depression used self-report measures of depression; however, this study assessed depression using a diagnostic interview. Another advantage of this study is the relation between cognitive vulnerability and depression was specific to one gender and age group, demonstrating the importance of cognitive vulnerability in relation to depression for early adolescent girls. The strong relation between the cognitive triad and

depression severity found in this study supports previous literature and provides further evidence that the cognitive triad may be an important correlate of depression in early adolescent girls.

Pathways to the Cognitive Triad

Beck and colleagues (1979) hypothesize depressive schemata are formed through early learning experiences, particularly within the family; therefore, this study examined the direct effects of family environment variables on the cognitive triad. The results of this study supported the hypothesis that family environment variables would have a significant direct effect on the cognitive triad. Specifically, participants' reports of more family social/recreational activities, more communication, and more cohesive and less conflictual relationships were significantly associated with a more positive view of the self, world, and future. These results suggest the family environment may play a role in the development of a girl's sense of the self, world, and future. Thus, family environments characterized by more cohesive and less conflictual relationships, more communication, and higher engagement in social/recreational activities are associated with girls have a more positive beliefs about the self, world, and future. Additionally, the family environment variables used in this study were found to be significantly correlated with each other (communication and social/recreational activities: r=.63, p<.001; relationships and social/recreational activities: r=.54, p<.001; relationships and communication, r=.60, p<.001). These significant correlations suggest the family variables in the model are closely related. Thus, it is important to consider how these various aspects of the family environment may be related to each other and how each variable may influence the cognitive triad.

The social/recreational activities subscale assesses the child's perception of her family's engagement in fun activities and sociability with others outside the family. It is likely that families who engage in more fun activities with each other may have better family relationships, characterized by more cohesion and less conflict. In other words, early adolescent girls who spend quality time with their families, such as "playing games together," may perceive their family as being more supportive and available. Furthermore, it is not unreasonable to suggest that families who are engaged with each other in social/recreational activities may have better communication. It is possible families who spend more time together have more open communication in the family and are more likely to engage other family members in decision making processes. Additionally, families who are more social outside of the family may have better communication skills in general. In the path model, the social/recreational activities variable had the largest direct effect on the cognitive triad of all the family variables (β=.23, b=-.40, p<.01), suggesting the importance of family social/recreational activities in relation to the cognitive triad. Thus, a girl's perception of her family's engagement in social/recreational activities is likely internalized into how she feels about herself, the world, and the future. If families regularly engage in fun activities together, the child may perceive the time spent with her family as evidence of her lovability and worth. On the other hand, if families rarely engage in fun activities, the child may perceive herself as not being worthy of her parents time or as evidence that she is unlovable. This subscale also taps into the family's sociability with other people. Socialization with others may contribute to whether a child perceives the world as being positive or negative. For instance, if a child is isolated from other people besides her family, she may begin to

internalize the world as being “mean” or “unfriendly.” In the larger intervention study, increasing family engagement in fun activities was found to be an effective coping strategy for girls with depression. Perhaps girls whose families are more active and social internalize a sense of positivity and hopefulness regarding the future.

The family social/recreational activities subscale was the only family environment variable to have a significant indirect, or mediated, effect on depressive severity. Additionally, the family social/recreational activities subscale had the largest direct effect on the cognitive triad of all the family variables. These results provide evidence for the unique and substantial influence a family’s engagement in social/recreational activities may have on the development of depression for early adolescent girls. In essence, the more social and recreational activities a family participates in, as perceived by the child, the more positive the child will view herself, world, and future, which will be associated with less depressive symptoms.

Family communication demonstrated a direct effect on the cognitive triad and significant positive correlations with family relationships and social/recreational activities. As previously mentioned, family engagement in recreational activities could facilitate more communication within the family. Additionally, more family communication was associated with more positive family relationships. Families who discuss problems and voice opinions may also tend to get along better and perceive their family as being supportive. It is possible that girls who perceive their family communication to be high may also perceive their family as being closer and having a sense of togetherness. Family communication also directly affects the child’s beliefs about the self, world, and future. If a child views her family communication to be low,

the child may internalize this isolation and lack of communication as evidence of her unlovability and worthlessness. Similarly, a child may perceive herself as being helpless if she believes she has no voice in her family. A child may also generalize little communication at home to her perception of the world. For instance, a lack of open communication may be internalized as the world being full of burdens or problems.

Lastly, the family relationship subscale was also found to have a significant effect on the cognitive triad. This finding suggests that girls who perceive less conflict and more cohesion in their family relationships had more positive beliefs about the self, world, and future. Perhaps girls who experience more conflict within their family feel unsupported and isolated in their family. As a result, these girls integrate beliefs of unlovability and worthlessness into their sense of self. Furthermore, high levels of conflict and low levels of support may lead girls to interpret the world as being negative and the future as being hopeless.

The finding that the family environment directly affects participants' cognitive triad supports Beck et al.'s (1979) theory that cognitions and schemas are formed through early learning experiences, especially within the family. Furthermore, the results are consistent with other studies findings that family variables, such as low maternal acceptance (Garber & Flynn, 2001; Rudolph et al., 2001), rejecting family environments, (Rudolph et al., 2001), and negative parenting (Gibb et al., 2001; Mezulis et al., 2006; Rose et al., 1994), are associated with maladaptive cognitions in children and adolescents. This body of evidence suggests children may internalize aspects of their family environment through which they develop complimentary beliefs about the self, world, and future.

Previous research also supports that family environments are associated with depression. Research has found depressed children have families that are less cohesive, more controlled and conflictual, communicate less, express emotion more intensely, and engage in fewer social and recreational activities (Barerra & Garrison-Jones, 1992; Cole & McPherson, 1993; Jewell & Stark, 2003; Ostrander et al., 1998; Stark et al., 1990). Low familial support and high family conflict have been consistently associated with depression in the literature (Lewinsohn, Roberts, Seeley, et al., 1994; Sheeber et al., 1997; Stice et al., 2004; Windle, 1992). While this study did not test the direct effects of these family variables to depression, the family social/recreational activities subscale was found to have a significant indirect, or mediated, effect on depression.

The hypothesis that negative life events would have a significant direct effect on the cognitive triad was supported in this study. An increase in negative life events, as measured by the number and impact of negative events, was associated with more negative beliefs about the self, world, and future. The experience of negative life events, therefore, may influence how a child develops her perception of the self, world, and future. The measure of negative life events in this study assesses a variety of life experiences. Based upon clinical experiences with the current sample, girls experiencing depression sometimes blamed themselves for negative events and report the event as being "their fault." These girls, therefore, may develop complimentary beliefs about the self, such as they are bad or worthless. Girls who experience more negative life events and perceive these events as having a strong effect on them may internalize the world as hostile and unfair. Similarly, it is possible that such negative events contribute to a sense of helplessness and unpredictability about the future. Children who experience adverse

life events may internalize that things will never get better for them and they will continue to suffer in the future. Negative life events also had a significant indirect effect on depressive severity further emphasizing the importance of negative life events as an etiological pathway to depression. This finding replicates past research and suggests negative life events negatively impact a child's sense of the self, world, and future. The child's cognitive triad, in turn, influences the depressive severity.

It also seems important to consider the significant correlation between family relationships and negative life events (r=-.23, p<.001). The negative relation suggests more negative life events are associated with less positive family relationships. Upon examining the frequency of reported negative life events, the most commonly reported negative life events were "trouble with brother or sister" (n=106), "serious illness or injury to family member" (n=92), "major arguments between you and mom or dad" (n=85), and "trouble with classmates" (n=85). Three of these negative life events seemed to be directly related to family conflict or family relationships. While the LEC measures a variety of life events, it seems that the most frequently reported negative life events were related the family, providing further support for the importance of the family in relation to the cognitive triad. Similarly, negative life events was the only variable in the model significantly associated with maternal symptoms of depression and anxiety (r=.20, p<.05). This finding raises important questions regarding the relation between these two variables. The association between negative life events and depression in adolescents and adults has been consistently established in the literature (Franko et al., 2004; Pine et al., 2002; Tram & Cole, 2000). Perhaps the negative life events reported by participants in the study were directly related to the mothers' symptoms of depression and anxiety. It is

also possible that symptoms of maternal depression and anxiety may serve as a type of negative life event for the participants.

The finding that negative life events were significantly associated with the cognitive triad is consistent with prior research. Garber and Flynn (2001) suggest the experience of negative life events is likely one of the most important factors underlying the development of cognition. Multiple studies have shown a direct relation between negative life events and depressogenic cognition (Cole & Turner, 1993; Garber & Flynn; Greenberg et al., 2008; Rienemann & Teeter Ellison, 2004; Rudolph et al., 2001; Tram & Cole, 2000). Findings in previous research suggest negative life events may be a key factor in the developmental origins of maladaptive cognitions. The finding that negative life events had a significant indirect, or mediated, effect on depression was also consistent with past research, which found that cognitive vulnerability mediated the relation between negative life events and depression in children (Cole & Turner, 1993; Rienemann & Teeter Ellison, 2004; Tram & Cole, 2000).

Results did not support the hypothesis that symptoms of maternal anxiety and depression would affect the participants' cognitive triad. This finding is inconsistent with the literature supporting the relation between maternal depression and childhood depression (Beardslee et al., 1998; Hammen & Brennon, 2003; Lieb et al., 2002; Warner et al., 1992), maternal depression and anxiety and childhood depression (Grigoroiu-Serbkescu et al., 1991), as well as maternal depression and maladaptive cognitions in children (Garber & Flynn, 2001; Goodman, Admanson, Riniti, & Cole, 1994; Jaenicke et al., 1987). While this result may initially seem surprising in light of the previous literature, there are several plausible explanations to account for this insignificance.

One possibility for the insignificance between maternal depression and anxiety and the cognitive triad is the mothers may have underreported their symptoms. Overall, the mothers of the depressed sample did report more symptoms (*M*= 20.12, *SD*=14.63) compared to the mothers in the control group (*M*= 16.43, *SD*= 15.43); however, this difference was not statistically significant. Prior to completing the SCL-90R, the mothers in the depressed sample received feedback regarding their daughter's experience with depression. This feedback may have influenced the mothers to underreport symptoms when filling out the SCL-90R. It is possible mothers may have been defensive, self protective, or embarrassed and wanted to portray themselves more positively on the measure. Additionally, some mothers in the larger intervention study reported that they were taking antidepressant medications and/or receiving psychosocial intervention, which may have affected the presence of symptoms in the past week. Unfortunately, information related to maternal treatment of depression was not systematically collected in the larger study; therefore, it is impossible to know how this may have affected the results.

Another plausible explanation for why maternal depression and anxiety was not significantly associated with the cognitive triad is the nature of the assessment of maternal psychopathology. Several studies have found a significant relation between maternal depression and negative cognitions in children (Garber & Flynn, 2001; Goodman et al., 1994; Jaenicke et al., 1987). All of these studies used a positive history of MDD episodes, or lifetime rates of depression, as a measure of maternal depression. The current study, however, only assessed symptoms of depression and anxiety for the past seven days. It is possible that using a history of depression and anxiety based upon a

diagnostic interview rather than current self-reported symptoms may produce different results. Similarly, most studies examining the relation between maternal depression and childhood depression use "top down" sampling rather than "bottom up" sampling used in this sample (Swartz et al., 2005), and are based upon lifetime rates of psychopathology (Grigoroiu-Serbkescu et al., 1991). Differences in methodology raise important questions regarding the insignificant relation of maternal psychopathology and maladaptive cognitions in children in this study.

Lastly, another possible explanation for why maternal depression and anxiety was not significantly associated with the cognitive triad is that this relation was better accounted for by other variables in the model. The current study was not assessing the genetic transmission of depression or anxiety, but rather exploring the environmental influence maternal symptoms would have on a child's sense of the self, world, and future. Garber and Flynn (2001) found other familial factors, such as maternal cognitive style and parenting style, uniquely contributed to children's cognitions beyond maternal depression. Perhaps having a mother with depression and/or anxiety symptoms may result in a more negative family environment, such as less social/recreational activities, less communication, and less positive family relationships. As suggested by Goodman and Gotlib (1999), mechanisms of depression transmission may include exposure to negative maternal affect, cognitions and behaviors, and the environmental context of the child's life. These mechanisms could potentially be accounted for by the family environment subscales in the model.

The hypothesis that there would be a significant direct effect from anxiety to the cognitive triad was not supported. It is important to note that while the path was not

statistically significant, it was approaching significance (β=-.11, *b*=-23.40, *p*=.06). The negative path coefficient indicates that higher anxiety symptoms were associated with a more negative cognitive triad. While not statistically significant, these results seem to be consistent with the previous literature. Both Kaslow et al. (1992) and Jacobs and Joseph (1997) found the CTI-C to be associated with both anxiety and depression in children; however, the CTI-C was found to be more closely related to depressive symptoms than anxiety symptoms. Based on their findings with the CTI-C, Jacob and Joseph concluded, "cognitive schema accounted for more of the variance on the measure of depression than the measure of anxiety suggesting that although the negative cognitive triad may be present in both symptomatology groupings it is more prominent in depression than anxiety" (p. 770). Supporting this statement, results from the current study found a stronger relation between depressive symptoms and the cognitive triad than between the cognitive triad and anxiety symptoms.

These results also raise important questions regarding the relation between cognitions and internalizing symptoms. Research suggests both depression and anxiety disorders are associated with cognitive distortions (Kendall et al., 1992). Beck's cognitive model of psychopathology suggests disorders may be differentiated by the content of thinking, referred to as the cognitive content-specificity hypothesis (Beck, 1976). The results of this study seem to support the cognitive content-specificity hypothesis given that the CTI-C, which measures depressogenic cognitions, was significantly associated with depressive symptoms but not with anxious symptoms. Furthermore, the model suggests anxiety symptoms may be directly affecting the cognitive triad; however, it is just as feasible that the participants' beliefs about the self, world, and future could be

contributing to anxious symptomatology; therefore, it is important to raise questions about the directionality of this relation.

Overall, the results of this study provide further information regarding the relation between the cognitive triad and depression and the pathways to cognitive vulnerability. Past research suggests a mediational model of depression may be more applicable to children given that cognitive schemas are less structuralized in childhood (Cole & Turner, 1993; Reinemann & Ellison, 2004; Tram & Cole, 2000). While this study did not directly compare moderational and meditational models of depression, the findings of this study offer some support for the perspective of a meditational model in childhood. This study found fewer family social/recreational activities, more conflictual and less cohesive family relationships, less family communication, and more negative life events were significantly associated with a more depressogenic cognition. A more depressogenic cognition, in turn, was associated with more depressive symptoms. Additionally, significant indirect, or mediated, effects were found for negative life events and family social/recreational activities to depressive severity.

Relation between Anxiety and Depression

As expected, results supported the hypothesis that there would be a significant positive relation between anxiety symptoms and depression; therefore, participants with more anxiety symptoms also experienced higher depressive symptoms. Additionally, the standardized regression weight ($\beta = .47$) implies that participants' anxiety symptoms had a strong effect on her depressive symptoms. For every one standard deviation the participants' anxiety symptoms score went up, there was a .47 standard deviation increase in the participants' depressive symptoms score.

This finding is consistent with previous research examining the relation between anxiety and depression. Previous literature suggests there is a significant and meaningful relation between depression and anxiety in children and adolescents (Brady & Kendall, 1992; Seligman & Ollendick, 1998). Multiple studies have found children with both anxiety and depression tend to report more severe depressive symptoms than those with depression or anxiety only (Brady & Kendall, 1992; Kendall et al., 1992; Mitchell et al., 1988; Rohde et al., 2001; Stark et al., 1993). Diagnostically, there was a high rate of concurrent comorbid anxiety, approximately 50%, in the sample of depressed girls in this study. This rate is consistent with previous studies finding rates of comorbid anxiety and depression typically ranging from 20% to 50% (Angold et al., 1999; Brady & Kendall, 1992). Additionally, many girls in the study presented with subclinical symptoms of anxiety but did not meet criteria for a diagnosis.

As proposed in this model, anxiety has been cited as a likely risk factor to depression in the literature (Flannery-Schroeder, 2006; Seligman & Ollendick, 1998; Wittchen et al., 2003). The results of this study indicate anxiety symptoms had a direct effect on depressive severity. Previous literature supports the proposal that anxiety may be a risk factor for depression. Nottlemann and Jenson (1995) suggest there is a developmental progression in co-occurring anxiety and depression, and anxiety has been found to precede the initial onset of major depression (Avenevoli et al., 2001; Costello et al., 2003; Lewinsohn et al., 1997). Additionally, longitudinal studies have found higher levels of anxiety in children predicted an increase in depressive symptoms (Bittner et al., 2004; Cole et al., 1998). Clark and Watson (1991) suggest the relation between anxiety and depression is accounted for by a shared negative affect, known as the tripartite

model. Research has shown that negative affect and general anxious emotion are very similar (Stark & Laurent, 2001), which supports the idea that anxiety may represent a risk factor for depression (Albano, Chorpita & Barlow, 2003). This research suggests anxiety may play a role in the development of depression, but the mechanisms for how anxiety may influence the development of depression remain unclear. It is possible having an anxiety disorder creates more life stress for children, which contributes to the development of depression. There also may be similar genetic factors or shared cognitive distortions which contribute to the onset of depression. It is important to note, however, that nosological and methodological considerations cannot be ruled out when conceptualizing the relation between depression and anxiety (Brady & Kendall, 1992). As such, the findings of this model insinuate anxiety symptoms have a direct effect on depression symptoms; however, other explanations for the relation cannot be ruled out.

Overall, this study replicated previous literature finding a significant relation between reported symptoms of anxiety and depression. Despite the strong relation between depression and anxiety, studies rarely measure both anxious and depressive symptoms in models of depression and anxiety (Hankin et al., 2004). Given the strong relation found in this study and research suggesting anxiety is a risk factor for depression, including anxiety symptoms in models of depression in future research is important.

Pathways to Anxiety

The hypotheses that maternal depression and anxiety, negative life events, and family environment variables would significantly affect severity of anxiety were not supported in this study. These results are inconsistent with previous literature that has found anxiety to be associated with negative life events (Boer et al., 2006; Cuffe et al.,

2005; Gothelf et al., 2004; Phillips et al., 2005), parental anxiety and depression (Beidel & Turner, 1997; McClure et al., 2001) and the family environment (Muris, 2006; Stark et al., 1990). In light of this previous literature, the results of this study may seem surprising; however, there are several plausible explanations for the lack of significance between these variables and anxiety. Considering none of pathways to anxiety were significant, potential explanations are discussed as a whole.

One explanation is that maternal depression and anxiety, negative life events, and the family environment, as measured in this study, are not significantly related to anxiety symptoms. It is possible the specific aspects of these variables assessed in this study resulted in a lack of significance. For instance, some research suggests negative events related to threat are associated with anxiety disorders and negative events related to loss are associated with depressive disorders (Sandin et al., 2004). Perhaps the measure of negative life events in this study was more reflective of loss events rather than those associated with threat. Similarly, parental control is an aspect of the family environment that has been consistently related to childhood anxiety in research (Rappe, 1997). The family environment variables, however, in this study did not seem to reflect specific aspects of parental control. As previously discussed, the measure of maternal depression and anxiety has frequently been based upon history of diagnosis rather than current symptoms as used in this study. It is possible, therefore, that anxiety disorders are not specifically related to the variables in this model but to similar risk factors correlated with these variables.

The most plausible explanation suggests the relation between these stressors and anxiety is through cognitive vulnerability, similar to depression. The current study found

negative life events and the family environment were significantly related to the cognitive triad and the cognitive triad was then significantly related to depression. It is possible that the variables of stress in this model may significantly contribute to maladaptive anxious cognition, which then is significantly related to anxiety. This explanation is supported by a model of the development of anxiety proposed by Chorpita (2001). Chorpita suggests a sense of "diminished control" acts as a mediator between stressful events and anxiety in children. In other words, a child experiences a life event which contributes to her general sense of limited control, which then affects her level of anxiety. Additionally, this model has been supported by research. Chorpita, Brown, and Barlow (1998) examined the relation between the family environment, locus of control, and severity of anxiety in a mixed sample of children with anxiety and controls. The authors found the measures of control mediated the relation between the family environment and children's anxiety symptoms. The model in this study, however, did not include a measure of anxious cognition. Rather than being directly related to anxiety symptoms, it is possible the stressors in this study are related to the participants' locus of control, and then the child's sense of control is related to anxiety. Unfortunately, this explanation cannot be tested because a measure of locus of control is not available.

This study examined specific direct pathways to anxiety, which were not found to be significant. The hypotheses that these pathways would be significant were based upon the substantive theory of comorbidity, which suggests comorbid disorders may share similar etiological pathways. These results raise questions regarding the application of this theory and whether anxiety and depression share similar risk factors. On the other hand, this study found negative life events and the family environment had significant

direct effects on depressive cognitive vulnerability. It is possible these same variables may have significant direct effects on anxious cognitive vulnerability. Future research is needed to address these questions.

Anxiety as a Moderator between the Cognitive Triad and Depression

One of the overall aims of this study was to investigate the role of anxiety in an model of depression in early adolescent girls. The main analyses in the study tested whether anxiety had a direct effect on depression and whether anxiety mediated the relation between life stressors and depression. The results suggest anxiety and depression are strongly related, with anxiety having a significant direct effect on depression. None of the life stressors, however, had a direct effect on anxiety symptoms. An exploratory analysis was utilized to further understand how anxiety may influence etiological models of depression, specifically examining whether anxiety is a moderator for the relation between the cognitive triad and severity of depression. This hypothesis was based upon the Beck's cognitive content-specificity theory (Beck, 1976) suggesting disorders may be differentiated by the content of their thinking. While this exploratory analysis was not directly testing this theory, it explored whether the relation between depressogenic cognitions and depression may be affected by the presence of anxiety symptoms.

Results from the exploratory analysis supported the hypothesis that the relation between the cognitive triad and depression was stronger for girls without anxiety symptoms than for girls with anxiety symptoms. A multi-sample model comparison revealed that when the paths from maternal anxiety and depression, family environment variables, and negative life events to the cognitive triad were constrained across groups, there was not a significant change in chi-square. As expected, this finding suggests there

was not a significant difference in the effect of these life stressors on the cognitive triad for girls with anxiety and girls without anxiety. When the path from the cognitive triad to depression, however, was set equal across groups there was a significant change in the chi-square. This result suggests the relation between the cognitive triad and depression is significantly different for girls with anxiety and girls without anxiety. There was a stronger relation between the cognitive triad and depression (p<.001) for girls with minimal to no anxiety than for girls with anxiety symptoms (p=.01). It is important to note that the path from the cognitive triad to depression was significant across groups, suggesting the relation between the cognitive triad and depression is important for both groups; however, the effect was stronger for girls without anxiety symptoms.

The results of the exploratory analysis add to the current literature and debate regarding the relation between anxiety, depression, and cognition. Perhaps, the current finding is best related to previous literature regarding the cognitive content-specificity hypothesis. A meta-analysis of Beck's cognitive content-specificity theory (Beck, 1976) found partial support for the hypothesis, with depressive symptomatology sharing more variance with depressed cognitive content than with anxious cognitive content (Beck & Perkins, 2001). Furthermore, previous research has found partial support for Beck's hypothesis with depressed children in grades 4 to 7 endorsing more depressive cognitions than children with anxiety (Laurent & Stark, 1993). This research suggests depression symptomatology is closely related to depressogenic cognitions, as found with this study. For children with anxiety symptoms, the relation between the cognitive triad and depression was still significant but not as strong as the other group without anxiety. Perhaps, the girls with anxiety also have anxious cognitions that are contributing to

depressive symptoms. In fact, Beck and Perkins (2001) found anxious cognitions shared equal variance between depressive symptomatology and anxious symptomatology. The current study, however, was not able to account for anxious cognitions. Future studies examining the relation between anxiety, depression, and cognition are needed.

Limitations

A number of limitations should be considered when interpreting the results of this study. First, a significant limitation of this study is the results are based upon cross-sectional data, suggesting causal inferences should not be made. While the path model was based upon theory and previous research, it is impossible to truly know the path direction based upon this data. For instance, even though the model suggests only the cognitive triad influences depression, it is impossible to rule out that depression does have direct effects on the cognitive triad as well. Additionally, these variables have been proposed to have reciprocal influences on each other. An ideal study examining risk factors for depression would be longitudinal, assessing these cognitive interpersonal pathways over time. Given the majority of participants in this sample were already depressed when the data was collected, it is important to consider how this may have impacted the participants' responses on the self-report measures. For example, the girls in this study may have interpreted their family environment as being overly negative. One study found adolescents who were experiencing depression reported poorer relationships with their mothers compared to other groups; however, observational data did not support this difference (Pavlidis & McCauley, 2001). Given the strong relation between cognitive distortion and depression, it is possible the girls perceived their family environments to

be more negative than actuality. The correlations, therefore, in this model may be inflated due to the nature of this data collection.

Another limitation, which is also an advantage of the study, is the study is specific to early adolescent girls. Results from the study, therefore, do not account for potential gender differences and care should be given not to generalize these results to boys or other developmental ages. Having a gender-specific model of depression for girls this age, however, is particularly useful given the prevalence and gender differences found with depression.

A third limitation is the measure of negative life events in this study, the LEC, only assesses life stressors in the past 12 months. Research, however, has found negative events have long-term effects on the onset of psychiatric disorders in children and adolescents (Goodyear et al., 1987). For example, negative life events were found to predict depression across a 5 year span (Nolen-Hoekesma et al., 1992). It is possible adverse life events occurring earlier than the 12 months assessed in this study may have had a significant effect on the development of the cognitive triad and depression.

As previously mentioned, a significant number of mothers did not complete the SCL-90R in the depressed sample. While there did not appear to be a significant differences in reports of family functioning, negative life events, the cognitive triad, or symptoms of depression or anxiety between the two groups, it is unclear whether more parental data would have affected the results. Additionally, parental data in regards to psychopathology is limited to the SCL-90R. No information on history of depression or anxiety was collected. Parents were also not asked if they were currently being treated for depression or anxiety through therapy or medication. Having this information would be

helpful to further assess the relation between maternal depression and anxiety and their daughters' symptoms of depression and anxiety.

One of the limitations in research examining comorbid depression and anxiety is the overlapping of symptoms. This study used a semi-structured interview including symptoms of both anxiety and depression. In order to maintain construct validity, all diagnostic symptoms, included overlapping symptoms, were calculated into the total depression score and total anxiety score. While research has ruled out that overlapping symptoms accounts for the relation between anxiety and depression (Seligman & Ollendick, 1998), it is possible the overlapping symptoms may have inflated the relation between the two disorders in this study.

Additionally, a limitation of the current model is it did not account for all correlates of depression. One of the advantages of this study is that it represents a more comprehensive model of depression in early adolescent girls; however, the model did not include interpersonal peer factors or genetic vulnerability associated with depression. Both genetics (Sullivan et al., 2000) and deficits in social competency (Cole et al., 1996) have also been linked to depressive symptomatology; therefore, it is important to consider how these correlates would fit into the current model.

One advantage to the model of depression in the current study is it accounts for co-occurring anxiety symptoms. The current model, however, does not account for other comorbid disorders. While less prevalent in the sample (n=25), other comorbid diagnoses included attention deficit disorders, eating disorders, and oppositional defiant disorder. Future research is needed to explore the relation between these disorders and depression.

Another limitation of the study is that it did not include a measure of anxious cognitions, such as cognitions about vulnerability and lack of control over potentially dangerous events. Additional research is needed to better understand whether depression and anxiety share similar risk factors. Future research should examine whether the family environment and negative life events are related to anxious cognitions, as they were found to relate to depressogenic cognitions in this study.

Lastly, one of the assumptions of path analysis is that the exogenous variables are completely reliable and valid measures. Unfortunately, the exogenous variables in this study are not completely reliable and valid measures as found in most social science research. Latent variable structural equation modeling is often used to remove the effects of unreliability and invalidity from the estimation of the effect of one variable on another (Keith, 2006). A latent SEM model, however, could not be utilized in this study because the sample size was not large enough. Future studies, however, with larger sample sizes may wish to use latent variable SEM modeling to address this limitation.

Implications

The results of this study have important implications for the intervention and prevention of depression in early adolescent girls. Consistent with previous literature, this study found a significant relation between participants' cognitive triad and depressive severity. This finding suggests a more depressogenic cognitive triad is associated with higher depressive symptoms. Intervening at the cognitive level to restructure these negative cognitive schemas, therefore, is important with this population. Cognitive behavioral therapy, a treatment which targets these maladaptive beliefs, would be

particularly useful in helping girls develop a more positive sense of the self, world, and future.

Additionally, this study also found the family environment to have a significant effect on the cognitive triad, suggesting the importance of including the family environment in conceptualization of childhood depression. Promoting changes in the family environment, therefore, would also be important when intervening with this population. Utilizing family therapy or parent training to promote better family communication and improve family relationships may help girls this age internalize more healthy perspectives of the self, world, and future. Family engagement in social/recreational activities was found to have the strongest effect on the cognitive triad, and a significant indirect effect on depression. These results suggest this aspect of the family environment is particularly important when intervening with girls who are depressed. Encouraging the family to engage in fun activities, such as playing games or participating in hobbies, would likely help girls develop a more positive cognitive triad. Engagement in these fun activities could be especially powerful, as coping skills are utilized to help minimize symptoms of depression in therapy. These findings also have important implications in the prevention of depression. In childhood and early adolescence, cognitive schemas are developing; therefore, creating a healthy, supportive family environment is important. Increasing cohesion, decreasing conflict, encouraging open communication and engaging in social/recreational activities is likely to help a child perceive the self, world and future more positively, which may then decrease her vulnerability to depression in the future.

Negative life events had a significant direct effect on the cognitive triad and a significant indirect effect on depression, highlighting how adverse events may impact children. Addressing beliefs related to these events in therapy may help children develop more healthy cognitions related to the events. Additionally, therapy may help girls effectively cope with negative events that are out of their control.

This study also has important implications regarding the relation between depression and anxiety in early adolescent girls. This study found a significant relation between anxiety symptoms and depressive symptoms, suggesting these disorders are closely related. This result suggests it is important to consider comorbid anxiety symptoms in future models of childhood depression in girls. Exploratory results suggest anxiety symptoms may moderate the relation between the cognitive triad and depression. This preliminary result suggests the relation between the cognitive triad and depression is stronger for girls without anxiety symptoms, which would have implications for the treatment of girls with and without anxiety symptoms. Additional research is needed to better understand how these two disorders are related and whether anxiety is a risk factor for depression.

Lastly, the pathways in the model that were not significant raise questions and suggest the need for more research. This study did not find a significant relation between maternal depression and anxiety and the cognitive triad, which has been found in previous studies. Additionally, the pathways from negative life events, family environment, and maternal depression and anxiety to anxiety symptoms were not significant. It is possible these variables may significantly impact anxious cognition

rather than anxiety symptoms. More research, however, is needed to address these findings.

Conclusions

The current study sought to investigate the role of anxiety symptoms in a cognitive-stress meditational model of depression in early adolescent girls. Overall, results of this study replicated and built upon previous literature examining specific risk factors of childhood depression. An important finding was that a more depressogenic cognitive triad was significantly associated with higher depressive severity. This finding supports Beck's theory of cognitive vulnerability to depression and highlights the importance of cognition in the conceptualization and intervention of depression in early adolescent girls.

This study also examined the pathways of cognitive vulnerability given that Beck and colleagues (1979) propose depressive schemata are formed through early learning experiences. Results supported this theory, finding family environment variables were significantly associated with the cognitive triad. Specifically, more cohesive and less conflictual family relationships, more communication, and higher involvement in social/recreational activities were associated with a more positive cognitive triad. Family engagement in social/recreational activities had the strongest effect on the cognitive triad and was the only family variable to have a significant indirect effect on depressive severity, suggesting the importance of family social/recreational activities in relation to depression. Negative life events also had a significant direct effect on the cognitive triad and a significant indirect effect on depression. Overall, these results suggest the family environment and negative life events are potentially important factors in the development

of depressogenic cognitions and ultimately depression, which have useful treatment and prevention implications.

Results did not find the cognitive triad to be significantly related to maternal depression and anxiety or anxiety symptoms. The relation between maternal symptoms of depression and anxiety and the cognitive triad is a bit surprising given the previous literature in the field; therefore, more research is needed to better understand this finding. While the relation between anxiety symptoms and the cognitive triad was not statistically significant, it was approaching significance (p=.06). The results of this study are consistent with previous literature suggesting that the CTI-C is more closely related to depression than to anxiety.

This study found a significant relation between anxiety and depression in girls. This relation was strong and suggested that higher anxiety symptoms were associated with higher depressive symptoms. This finding is consistent with previous literature and raises important questions regarding anxiety as a risk factor for depression. Further research is needed to explore this theory. Additionally, most previous research examining models of depression do not include co-occurring symptoms. The results of this study, however, suggest it is important to include co-occurring anxiety symptoms in future models of depression.

While anxiety symptoms were significantly associated with depression, family environment variables, negative life events, and maternal depression and anxiety were not significantly associated with anxiety symptoms. This finding raises questions about whether depression and anxiety share similar risk factors; however, the current study found these same variables were related to depressogenic cognition. This study did not

explore whether these variables may be related to anxious cognition. It is possible these two disorders share risk factors, but they are both significantly related to cognition rather than directly to the disorder's symptomatology. Further research is needed in this area.

Lastly, exploratory results found anxiety severity was a moderator for the relation between depressogenic cognitions and depression. Specifically, the results suggested the relation between the cognitive triad and depression was stronger for girls without anxiety than for girls with anxiety symptoms. These results are preliminary but have potential implications for treatment. Additional research is needed to replicate this finding.

This investigation has continued to highlight the importance of depressogenic cognitions as a risk factor for depression in children. Additionally, the study has demonstrated how family environment and negative life events are significantly related to depressogenic cognitions. Lastly, results of the current study demonstrate the significance of co-occurring anxiety symptoms in relation to depression severity. Hopefully, this investigation will prompt further discussion and research regarding the correlates of depression in early adolescent girls as well as the specific relation between depression and anxiety in childhood.

APPENDICES

Appendix A

DSM-IV Criteria for Major Depressive Disorder and Major Depressive Episode

DSM-IV Criteria for Major Depressive Disorder

A. Presence of one or more Major Depressive Episodes (to be considered separate episodes, there must be an interval of two consecutive months in which criteria are not met for a Major Depressive Episode).
B. Major Depressive Episode is not better accounted for by Schizoaffective Disorder and is not superimposed on Schizophrenia, Schizophreniform Disorder, Delusional Disorder, or Psychotic Disorder Not Otherwise Specified.
C. There has never been a Manic Episode, Mixed Episode, or Hypomanic Episode.

DSM-IV Criteria for Major Depressive Episode

A. Five (or more) of the following symptoms must be present during the same two-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood, or (2) loss of interest or pleasure.
    1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad or empty) or observation made by others (e.g., appears tearful). **Note: in children and adolescents, can be irritable mood**.
    2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation made by others).
    3. Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. **Note: in children, consider failure to make expected weight gains**.
    4. Insomnia or hypersomnia nearly every day.
    5. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
    6. Fatigue or loss of energy nearly every day.
    7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
    8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
    9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.
B. The symptoms do not meet criteria for a Mixed Episode.
C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hypothyroidism).
E. The symptoms are not better accounted for by Bereavement, i.e., after the loss of a loved one, the symptoms persist for longer than two months or are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.

Appendix B

DSM-IV Criteria for Dysthymic Disorder

A. Depressed mood for most of the day, for more days than not, as indicated either by subjective account of observation by others, for at least two years. **Note: In children and adolescents, mood can be irritable and duration must be at least one year.**
B. Presence, while depressed, of two (or more) of the following:
    1. Poor appetite or overeating
    2. Insomnia or hypersomnia
    3. Low energy or fatigue
    4. Low self-esteem
    5. Poor concentration or difficulty making decisions
    6. Feelings of hopelessness
C. During the two-year period (one year for children or adolescents) of the disturbance, the person has never been without the symptoms in Criteria A and B for more than two months at a time.
D. No Major Depressive Episode has been present during the first two years of the disturbance.
E. There has never been a Manic Episode, a Mixed Episode, or a Hypomanic Episode, and criteria have never been met for Cyclothymic Disorder.
F. The disturbance does not occur exclusively during the course of a chronic Psychotic Disorder, such as Schizophrenia or Delusional Disorder.
G. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hypothyroidism).
H. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Appendix C

DSM-IV Criteria for Depressive Disorder Not Otherwise Specified

A. A mood disturbance, defined as follows:
    1. At least two (but less than five) of the following symptoms have been present during the same two-week period and represent a change from previous functioning; at least one of the symptoms is either (a) or (b):
        a. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad or empty) or observation made by others (e.g., appears tearful). **Note: in children and adolescents, can be irritable mood**.
        b. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation made by others).
        c. Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. **Note: in children, consider failure to make expected weight gains**.
        d. Insomnia or hypersomnia nearly every day.
        e. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
        f. Fatigue or loss of energy nearly every day.
        g. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
        h. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
        i. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.
    2. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
    3. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hypothyroidism).
    4. The symptoms are not better accounted for by Bereavement.
B. There has never been a Major Depressive Episode, and criteria are not met for Dysthymic Disorder.
C. There has never been a Manic Episode, a Mixed Episode, or a Hypomanic Episode, and criteria are not met for Cyclothymic Disorder.
D. The mood disturbance does not occur exclusively during Schizophrenia, Schizophreniform Disorder, Schizoaffective Disorder, Delusional Disorder, or Psychotic Disorder Not Otherwise Specified.

Appendix D

DSM-IV Criteria for Generalized Anxiety Disorder

A. Excessive anxiety and worry (apprehensive expectation), occurring more days than not for at least 6 months, about a number of events or activities (such as work or school performance).

B. The person finds it difficult to control the worry.

C. The anxiety and worry are associated with three (or more) of the following six symptoms (with at least some symptoms present for more days than not for the past 6 months). **Note: Only one item is required in children**.

(1) restlessness or feeling keyed up or on edge
(2) being easily fatigued
(3) difficulty concentrating or mind going blank
(4) irritability
(5) muscle tension
(6) sleep disturbance (difficulty falling or staying asleep, or restless unsatisfying sleep)

D. The focus of the anxiety and worry is not confined to features of an Axis I disorder, e.g., the anxiety or worry is not about having a Panic Attack (as in Panic Disorder), being embarrassed in public (as in Social Phobia), being contaminated (as in Obsessive-Compulsive Disorder), being away from home or close relatives (as in Separation Anxiety Disorder), gaining weight (as in Anorexia Nervosa), having multiple physical complaints (as in Somatization Disorder), or having a serious illness (as in Hypochondriasis), and the anxiety and worry do not occur exclusively during Posttraumatic Stress Disorder.

E. The anxiety, worry, or physical symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

F. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hyperthyroidism) and does not occur exclusively during a Mood Disorder, a Psychotic Disorder, or a Pervasive Developmental Disorder.

Appendix E

DSM-IV Criteria for Posttraumatic Stress Disorder

A. The person has been exposed to a traumatic event in which both of the following were present:

1. the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others
2. the person's response involved intense fear, helplessness, or horror. Note: In children, this may be expressed instead by disorganized or agitated behavior

B. The traumatic event is persistently reexperienced in one (or more) of the following ways:

1. recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. **Note: In young children, repetitive play may occur in which themes or aspects of the trauma are expressed**.
2. recurrent distressing dreams of the event. **Note: In children, there may be frightening dreams without recognizable content**.
3. acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated). **Note: In young children, trauma-specific reenactment may occur.**
4. intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.
5. physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:

1. efforts to avoid thoughts, feelings, or conversations associated with the trauma
2. efforts to avoid activities, places, or people that arouse recollections of the trauma
3. inability to recall an important aspect of the trauma
4. markedly diminished interest or participation in significant activities
5. feeling of detachment or estrangement from others
6. restricted range of affect (e.g., unable to have loving feelings)
7. sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:

(1) difficulty falling or staying asleep
(2) irritability or outbursts of anger
(3) difficulty concentrating
(4) hypervigilance
(5) exaggerated startle response

E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than 1 month.

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if:

Acute: if duration of symptoms is less than 3 months
Chronic: if duration of symptoms is 3 months or more

Specify if:

With Delayed Onset: if onset of symptoms is at least 6 months after the stressor

Appendix F

DSM-IV Criteria for Panic Attacks and Panic Disorder with and without Agoraphobia

DSM-IV Criteria for Panic Attacks

A discrete period of intense fear or discomfort, in which four (or more) of the following symptoms developed abruptly and reached a peak within 10 minutes:

1. palpitations, pounding heart, or accelerated heart rate
2. sweating
3. trembling or shaking
4. sensations of shortness of breath or smothering
5. feeling of choking
6. chest pain or discomfort
7. nausea or abdominal distress
8. feeling dizzy, unsteady, lightheaded, or faint
9. derealization (feelings of unreality) or depersonalization (being detached from oneself
10. fear of losing control or going crazy
11. fear of dying
12. paresthesias (numbness or tingling sensations)
13. chills or hot flushes

DSM-IV Criteria for Panic Disorder with or without Agoraphobia

A. Both (1) and (2):

1. recurrent unexpected Panic Attacks
2. at least one of the attacks has been followed by 1 month (or more) of one (or more) of the following:
   a. persistent concern about having additional attacks
   b. worry about the implications of the attack or its consequences (e.g., losing control, having a heart attack, "going crazy")
   c. a significant change in behavior related to the attacks

B. Presence or Absence of Agoraphobia.

C. The Panic Attacks are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hyperthyroidism).

D. The Panic Attacks are not better accounted for by another mental disorder, such as Social Phobia (e.g., occurring on exposure to feared social situations), Specific Phobia (e.g., on exposure to a specific phobic situation), Obsessive-Compulsive Disorder (e.g., on exposure to dirt in someone with an obsession about contamination), Posttraumatic Stress Disorder (e.g., in response to stimuli associated with a severe stressor), or Separation Anxiety Disorder (e.g., in response to being away from home or close relatives).

Appendix G

DSM-IV Criteria for Separation Anxiety

A. Developmentally inappropriate and excessive anxiety concerning separation from home or from those to whom the individual is attached, as evidenced by three (or more) of the following:

(1) recurrent excessive distress when separation from home or major attachment figures occurs or is anticipated
(2) persistent and excessive worry about losing, or about possible harm befalling, major attachment figures
(3) persistent and excessive worry that an untoward event will lead to separation from a major attachment figure (e.g., getting lost or being kidnapped)
(4) persistent reluctance or refusal to go to school or elsewhere because of fear of separation
(5) persistently and excessively fearful or reluctant to be alone or without major attachment figures at home or without significant adults in other settings
(6) persistent reluctance or refusal to go to sleep without being near a major attachment figure or to sleep away from home
(7) repeated nightmares involving the theme of separation
(8) repeated complaints of physical symptoms (such as headaches, stomachaches, nausea, or vomiting) when separation from major attachment figures occurs or is anticipated

B. The duration of the disturbance is at least 4 weeks.

C. The onset is before age 18 years.

D. The disturbance causes clinically significant distress or impairment in social, academic (occupational), or other important areas of functioning.

E. The disturbance does not occur exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia, or other Psychotic Disorder and, in adolescents and adults, is not better accounted for by Panic Disorder With Agoraphobia.

Specify if:
Early Onset: if onset occurs before age 6 years

Appendix H

DSM-IV Criteria for Specific Phobia

A. Marked and persistent fear that is excessive or unreasonable, cued by the presence or anticipation of a specific object or situation (e.g., flying, heights, animals, receiving an injection, seeing blood).

B. Exposure to the phobic stimulus almost invariably provokes an immediate anxiety response, which may take the form of a situationally bound or situationally predisposed Panic Attack. **Note: In children, the anxiety may be expressed by crying, tantrums, freezing, or clinging**.

C. The person recognizes that the fear is excessive or unreasonable. Note: In children, this feature may be absent.

D. The phobic situation(s) is avoided or else is endured with intense anxiety or distress.

E. The avoidance, anxious anticipation, or distress in the feared situation(s) interferes significantly with the person's normal routine, occupational (or academic) functioning, or social activities or relationships, or there is marked distress about having the phobia.

F. In individuals under age 18 years, the duration is at least 6 months.

G. The anxiety, Panic Attacks, or phobic avoidance associated with the specific object or situation are not better accounted for by another mental disorder, such as Obsessive-Compulsive Disorder (e.g., fear of dirt in someone with an obsession about contamination), Posttraumatic Stress Disorder (e.g., avoidance of stimuli associated with a severe stressor), Separation Anxiety Disorder (e.g., avoidance of school), Social Phobia (e.g., avoidance of social situations because of fear of embarrassment), Panic Disorder with Agoraphobia, or Agoraphobia Without History of Panic Disorder.

Specify type:
Animal Type
Natural Environment Type (e.g., heights, storms, water)
Blood-Injection-Injury Type
Situational Type (e.g., airplanes, elevators, enclosed places)
Other Type (e.g., phobic avoidance of situations that may lead to choking, vomiting, or contracting an illness; in children, avoidance of loud sounds or costumed characters)

Appendix I

DSM-IV Criteria for Social Phobia (Social Anxiety Disorder)

A. A marked and persistent fear of one or more social or performance situations in which the person is exposed to unfamiliar people or to possible scrutiny by others. The individual fears that he or she will act in a way (or show anxiety symptoms) that will be humiliating or embarrassing.
Note: In children, there must be evidence of the capacity for age-appropriate social relationships with familiar people and the anxiety must occur in peer settings, not just in interactions with adults.

B. Exposure to the feared social situation almost invariably provokes anxiety, which may take the form of a situationally bound or situationally predisposed Panic Attack.
Note: In children, the anxiety may be expressed by crying, tantrums, freezing, or shrinking from social situations with unfamiliar people.

C. The person recognizes that the fear is excessive or unreasonable. Note: In children, this feature may be absent.

D. The feared social or performance situations are avoided or else are endured with intense anxiety or distress.

E. The avoidance, anxious anticipation, or distress in the feared social or performance situation(s) interferes significantly with the person's normal routine, occupational (academic) functioning, or social activities or relationships, or there is marked distress about having the phobia.

F. In individuals under age 18 years, the duration is at least 6 months.

G. The fear or avoidance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition and is not better accounted for by another mental disorder (e.g., Panic Disorder With or Without Agoraphobia, Separation Anxiety Disorder, Body Dysmorphic Disorder, a Pervasive Developmental Disorder, or Schizoid Personality Disorder).

H. If a general medical condition or another mental disorder is present, the fear in Criterion A is unrelated to it, e.g., the fear is not of Stuttering, trembling in Parkinson's disease, or exhibiting abnormal eating behavior in Anorexia Nervosa or Bulimia Nervosa.

Appendix J

DSM-IV Criteria for Obsessive Compulsive Disorder

A. Either obsessions or compulsions:

Obsessions as defined by (1), (2), (3), and (4):

(1) recurrent and persistent thoughts, impulses, or images that are experienced, at some time during the disturbance, as intrusive and inappropriate and that cause marked anxiety or distress
(2) the thoughts, impulses, or images are not simply excessive worries about real-life problems
(3) the person attempts to ignore or suppress such thoughts, impulses, or images, or to neutralize them with some other thought or action
(4) the person recognizes that the obsessional thoughts, impulses, or images are a product of his or her own mind (not imposed from without as in thought insertion)

Compulsions as defined by (1) and (2):

(1) repetitive behaviors (e.g., hand washing, ordering, checking) or mental acts (e.g., praying, counting, repeating words silently) that the person feels driven to perform in response to an obsession, or according to rules that must be applied rigidly
(2) the behaviors or mental acts are aimed at preventing or reducing distress or preventing some dreaded event or situation; however, these behaviors or mental acts either are not connected in a realistic way with what they are designed to neutralize or prevent or are clearly excessive

B. At some point during the course of the disorder, the person has recognized that the obsessions or compulsions are excessive or unreasonable. Note: This does not apply to children.

C. The obsessions or compulsions cause marked distress, are time consuming (take more than 1 hour a day), or significantly interfere with the person's normal routine, occupational (or academic) functioning, or usual social activities or relationships.

D. If another Axis I disorder is present, the content of the obsessions or compulsions is not restricted to it (e.g., preoccupation with food in the presence of an Eating Disorders; hair pulling in the presence of Trichotillomania; concern with appearance in the presence of Body Dysmorphic Disorder; preoccupation with drugs in the presence of a Substance Use Disorder; preoccupation with having a serious illness in the presence of Hypochondriasis; preoccupation with sexual urges or fantasies in the presence of a Paraphilia; or guilty ruminations in the presence of Major Depressive Disorder).

E. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

Appendix K

Initial Screening Consent Letter

(SCHOOL LETTERHEAD HERE)

Dear Parent,

SCHOOL is teaming up with Kevin Stark, Ph.D. from the University of Texas to evaluate a coping skills training program for girls called ACTION. The ACTION program is designed to teach girls how to manage their emotions and stress, solve problems, and think more positively about themselves. While we believe that all students could benefit from this program, currently, only girls who are experiencing high levels of distress will be able to participate. We are asking for permission from all parents of girls in GRADES for their daughters to participate in a screening that will help identify girls who are experiencing distress. Girls who participate in the screening will fill out a questionnaire that takes approximately 10 minutes to complete. Doctoral psychology students with appropriate training will supervise the completion of the questionnaires. At this time we do not anticipate any discomfort in completing the ACTION questionnaire.

Girls who report having more than a typical number symptoms of distress will be interviewed about specific symptoms of depression to determine if they are experiencing high levels of distress. The brief symptom interview will be conducted by trained graduate students or project staff under the supervision of Dr. Stark. If a girl in the study is reporting distress on the questionnaire or brief symptom interview, the parents will be contacted by phone to ensure the girl's well-being. ACTION staff or the school counselor may discuss your child's further participation in this research project at that time. For all girls who complete the questionnaire or interview and do not show significant symptoms of distress, parents will receive a letter stating those findings.

The purpose of the project is to determine whether the ACTION coping skills program is more effective than no counseling, and whether parent participation makes the program more effective. In addition, we are trying to learn whether adding follow-up meetings prevents future distress. The benefits to participants include possible participation in the ACTION program and helping advance our understanding of how to best help young girls manage emotions and stress, solve problems and feel better about themselves.

Participation in the project will not cost you anything and there will not be any financial compensation for participation. There are not any risks of harm from completing the questionnaire. There are no anticipated risks from completing the brief symptom interview. In fact, the procedure is designed to quickly identify and assist children who

are in distress. All materials and forms will be stored in locked file cabinets in a secure office at UT to protect confidentiality.

If a child reports that she is at risk of hurting herself or others, her parents would be immediately informed and she would immediately talk with her school counselor. In addition, she would be evaluated by one of the consulting psychiatrists at no cost to the family.

If you choose to participate, you or your daughter may stop participation at any time. Participation in the study is entirely voluntary. You are free to say that you do not want to participate by returning this form indicating on the back of this page that you do not want to participate. You can refuse to participate without penalty or loss of benefits to which you and your daughter are otherwise entitled. It will not affect your relationship with your child's school or the University of Texas.

Researchers are required by Texas state law and professional ethics codes to report to Child Protective Services (or other appropriate regulatory agency) all instances of alleged child abuse and neglect. Please note that if your child completes the screening questionnaire or interview and is believed to be at risk for emotional, psychological or possible physical harm or neglect, then the investigator will report this information to the attending physician, Child Protective Services, and any other necessary regulatory agencies. Please note when a child reports neglect or being harmed, participants cannot stop the referral of their child's case to the authorities and any subsequent actions taken.

If you have any questions about the study, you can call Kevin Stark, Ph.D. at (512) 471-0267, your school counselor, or principal.

If you have questions about your rights as a participant, please contact Lisa Leiden, Ph.D., Chair, The University of Texas at Austin Institutional Review Board for the Protection of Human Subjects, (512) 471-8871.

Sincerely,

_____

Researcher's Signature

_____

Principal's Signature

_____

Date

PLEASE KEEP THIS LETTER FOR YOUR RECORDS

PARENT/GUARDIAN SCREENING PROCEDURE CONSENT

Please check the appropriate box indicating that **YES** you have read this letter and are giving permission for your daughter to participate in the ACTION project at your child's school by completing the screening questionnaire and brief symptom interview, or **NO**, you have read this letter and you do not want your daughter to complete the questionnaire or interview. Regardless of your decision, please sign this form and return it to your child's teacher.

PLEASE RETURN THIS FORM TO YOUR CHILD'S SCHOOL WITH YOUR PREFERENCE NOTED BELOW:

_____**YES** I **give my permission** for my daughter to participate by completing the screening questionnaire and brief symptom interview.

_____**NO** I **do not give my permission** for my daughter to participate by completing the screening questionnaire or brief symptom interview

_____ _____

Parent's Signature Date

_____

Child's Name (please print)

We will provide feedback for all participants. Please provide information below if your child will be participating.

Parent/adult guardian name(s): _____

Mailing address: _____

City/ZIP:_____

Parent phone number(s) in case we need to reach you with a concern about your child:

Home_____cell_____work_____

Revised JLH 8/29/05

Appendix L

Child Assent Form

I agree to complete a questionnaire about my thoughts, feelings, and behaviors. This questionnaire has been explained to my parent or guardian and he or she has given permission for me to participate. I may decide at any time that I do not wish to participate and that it will be stopped if I say so. My specific responses will not be shared with anyone. However, general information about how I am doing and feeling may be shared with my parent.

When I sign my name to this page I am indicating that I read this page and that I am agreeing to participate.

_____ _____

Your Signature Date

_____

Please print your Name

Date of Birth _____

Month Day Year

ETHNICITY

(1) Do you consider yourself to be Hispanic or Latino? Please put an X in the box that best describes you.

☐ **Hispanic or Latino**

☐ **Not Hispanic or Latino**

RACE

(2) What race do you consider yourself to be? You may be more than one race. Please put an X in the box for <u>each</u> race that you believe you are. For example, if you believe you are White, you put an X in that box. If you believe you are White and Black, then you put an X in <u>both</u> the White <u>and</u> in the Black or African American box.

☐ ***American Indian or Alaska Native***

☐ ***Asian***

☐ ***Black or African American***

☐ ***Native Hawaiian or Other Pacific Islander***

☐ ***White***

☐ Check here if you do not wish to provide some or all of the above information

Appendix M

Letter to Parents if Screening Score is Below the Cutoff

DEPARTMENT OF EDUCATIONAL PSYCHOLOGY
THE UNIVERSITY OF TEXAS AT AUSTIN

School Psychology Program •George I. Sanchez Building 504 •Austin, Texas 78712-1296
(512) 471-4470 •FAX (512) 471-1288 •Campus Code D5800 •http://edpsych.edb.utexas.edu

Dear Parent,

We would like to thank you for giving your daughter permission to complete the screening measures as part of the collaborative project between UT and your child's school district. The screening measures have been scored and you will be glad to know that your daughter did not report experiencing a significant level of distress or other signs of stress. If you question these results or would like additional information, please feel free to call Kevin Stark, Ph.D. at 471-0267, or contact your school counselor.

We will be conducting the screener on a periodic basis throughout the district. Since life circumstances can change, and adolescence can be a difficult time in a girl's life, we hope that you will allow your daughter to participate again in the future.

Sincerely,

_____

Kevin D. Stark, Ph.D.

Appendix N

Letter to Parents if Screening Score is Higher than Cutoff but DSM Interview Okay

DEPARTMENT OF EDUCATIONAL PSYCHOLOGY
THE UNIVERSITY OF TEXAS AT AUSTIN

School Psychology Program •George I. Sanchez Building 504 •Austin, Texas 78712-1296
(512) 471-4470 •FAX (512) 471-1288 •Campus Code D5800 •http://edpsych.edb.utexas.edu

Dear Parent,

We would like to thank you for giving your daughter permission to complete the screening questionnaires and the interview as part of the collaborative research project between UT and your child's school district. On the screening questionnaires your daughter reported experiencing some stress. Due to concern that she may be in distress, our research staff conducted an individual brief interview to assess her stress level. However, on the individual brief interview she indicated that she was not experiencing a significant level of distress. Typically, this is an indication that her reaction to stress is within normal range or that she had some misunderstanding of the items on the first questionnaire. If you question these results or would like additional information, please feel free to call Kevin Stark, Ph.D., licensed psychologist and principal investigator (512-471-0267), your school counselor, or Janay B. Sander, Ph.D., project coordinator (512-471-0218).

Sincerely,

_____

Kevin D. Stark, Ph.D.
(512) 471-0267

Appendix O

K-SADS-IVR Consent Form

DEPARTMENT OF EDUCATIONAL PSYCHOLOGY

THE UNIVERSITY OF TEXAS AT AUSTIN

School Psychology Program •George I. Sanchez Building 504 •Austin, Texas 78712-1296

(512) 471-4470 •FAX (512) 471-1288 •Campus Code D5800 •http://edpsych.edb.utexas.edu

Dear Parent,

Per our contact with you regarding your daughter's responses to the screening questionnaire and brief symptom interview, we are requesting permission for you and your daughter to complete a more comprehensive interview that will help us determine more accurately whether she is experiencing serious emotional concerns or whether she was not feeling well on the days that she completed the questionnaire and brief interview. The interviews will be conducted by trained doctoral psychology students under the supervision of Kevin Stark, Ph.D., licensed psychologist. The interview of your daughter will be completed in a room at school that will protect her privacy. It takes 45 to 90 minutes to complete and asks specific questions about how your daughter is feeling, thinking and behaving and a range of experiences she may have encountered. The interview with you will cover the same topics and can be conducted in person or over the phone if that is preferable, at a time that is convenient for you. Participation in the interview will not cost you anything and there will not be any financial compensation for participation. Completed interviews will be stored in locked file cabinets in a secure office at UT to protect confidentiality. If she is, she may be eligible for participating in the ACTION program. If this wouldn't be the best program for her, we will provide you with possible resources from within the school and the community.

If a child reports that she is at risk of hurting herself or others, her parents would be immediately informed and she would immediately talk to her school counselor. In addition, she would be interviewed by Kevin Stark, Ph.D., a licensed psychologist, or one of the consulting psychiatrists at no cost to the family. If a child reports that she is being hurt, the school's standard procedures for reporting such instances to the relevant state agency would be followed.

The purpose of the project is to determine whether the ACTION coping skills program is helpful, and whether parent participation makes the program more effective. In addition, we are trying to learn whether adding follow-up meetings prevents future distress. If you have any questions about the study, you can call Kevin Stark, Ph.D. at (512) 471-0267 your school counselor, or principal.

If you choose to participate, you or your daughter may stop participation at any time. Participation in the study is entirely voluntary. You are free to say that you do not want to

participate by returning this form indicating that you do not want to participate. You can refuse to participate and this decision will not affect your relationship with your child's school or the University of Texas.

Researchers are required by Texas state law and professional ethics codes to report to Child Protective Services (or other appropriate regulatory agency) all instances of alleged child abuse and neglect. Please note that if your child completes the screening questionnaire or interview and is believed to be at risk for emotional, psychological or possible physical harm or neglect, then the investigator will report this information to the attending physician, Child Protective Services, and any other necessary regulatory agencies. Please note when a child reports neglect or being harmed, participants cannot stop the referral of their child's case to the authorities and any subsequent actions taken.

If you have questions about your rights as a participant, please contact Lisa Leiden, Ph.D., Chair, The University of Texas at Austin Institutional Review Board for the Protection of Human Subjects, (512-471-8871). Let him know that you are enquiring about the study entitled "Helpfulness of the ACTION Coping Skills Program with and Without Parent Participation."

Please check the appropriate box indicating that **YES** you have read this letter and are giving permission for you and your daughter to participate by completing the interview, or **NO** you do not want to complete the interview nor do you want your daughter to complete the interview. Regardless of your decision, please sign this form and return it to your child's teacher. You will be given a copy of this permission letter to keep for your records.

☐ **YES** I give my permission for my daughter and I to participate by completing the interview.

☐ **NO** I do not give my permission for my daughter and I to participate by completing the interview.

_____ _____

Parent's Signature Date

_____ _____

Researcher's Signature Date

_____ _____

Principal's Signature Date

Revised KDS 8/29/05

Appendix P

Treatment Consent Form

DEPARTMENT OF EDUCATIONAL PSYCHOLOGY
THE UNIVERSITY OF TEXAS AT AUSTIN

School Psychology Program •George I. Sanchez Building 504 •Austin, Texas 78712-1296
(512) 471-4470 •FAX (512) 471-1288 •Campus Code D5800 •http://edpsych.edb.utexas.edu

Dear Parent,

Based on results of the screening and interview that you and your daughter have participated in so far, we are requesting permission for you and your daughter to continue and participate in the evaluation of the ACTION coping skills program. If you give your permission for your daughter to participate, she will be randomly assigned to one of three groups: (1) ACTION coping skills program, (2) ACTION coping skills program plus parent participation, or (3) wait to receive the program in about 12 weeks.

If your daughter is randomly assigned to the ACTION coping skills program, she will meet 20 times over the next twelve to sixteen weeks with a group of girls to participate in a counseling program that is designed to teach her problem solving, coping skills for managing her emotions and stress, and strategies for thinking more positively about herself and things in general.

If your daughter is randomly assigned to the counseling plus parent participation, she will meet 20 times over the next twelve to sixteen weeks with a group of girls to participate in a counseling program that is designed to teach her problem solving, coping skills for managing her emotions and stress, and strategies for thinking more positively about herself and things in general. In addition, you would be asked to attend a total of 10 meetings over this period that will last about an hour and a half. The parent meetings will be held at school after hours and daycare and refreshments will be provided at no expense. During these meetings parents will have a chance to learn the skills that their daughter is learning, and parents will learn strategies for helping their daughter to use the skills.

The girls will meet in a small group during an elective class. Each meeting will last one class period. Steps have already been taken to ensure that she will receive any class materials that she misses. The group meetings will be led by a trained doctoral psychology student or Ph.D. level therapist and a counselor from your daughter's school. The group leaders will be supervised by Kevin Stark, Ph.D. It is not expected that your daughter will experience any discomfort or risks from participating in the ACTION coping skills program. In fact, past experience with the program indicates that the girls enjoy participating and benefit from it.

If your daughter is randomly assigned to wait to receive counseling in about 12 weeks, we will take the following steps to ensure that she is okay. A doctoral psychology student will meet with her each week to monitor how she is doing, she will be discreetly observed in school at lunch or recess for about fifteen minutes per week, and the staff member will check-in with her teacher each week. In addition, every other week, the staff member will check with you to see if you have any concerns. At the end of the waiting period, she will have the opportunity to participate in the coping skills program. If at any point during this waiting period she reports feeling worse or you would like to seek counseling elsewhere, we will provide you with information about community and school resources. You have the option at anytime to seek additional services including consultation with one of the project's consulting psychiatrists at no cost to you.

We will be monitoring each girl's progress and report this information to two psychiatrists who are being paid by us to oversee each child's welfare. If a participant is not improving as a result of the program, then parents will be informed and we will meet with you to discuss other options for providing your daughter with help. If you would like information about medications that might be of assistance, the psychiatrists are available to meet with you and discuss these options at no cost to you.

To determine whether the ACTION coping skills program is helpful, we are asking you and your daughter to complete some questionnaires that help guide, and evaluate the effectiveness of the ACTION program. The questionnaires will take your daughter about one hour to complete. It will take you about 30 minutes to complete your questionnaires. We are asking you to complete the questionnaires so that we can determine whether participation in the ACTION program also benefits you and your family. The questionnaires have been completed by other children and adults without any discomfort. In order to assess the potential benefits of ACTION on school performance, our staff collects the following general education information: grades from reporting periods, attendance, and discipline information for participants.

For one year after completion of the ACTION program, your daughter will have the opportunity to meet with her group and apply the skills to the new problems and stresses that she faces as she grows up and navigates her way through the many difficulties of being a teenager. The groups will meet three times a semester over the rest of the course of the study. In addition, to determine if your daughter needs additional help, once a year, we will ask you and your daughter to complete the interview and the questionnaires to determine whether we have achieved the goal of preventing the difficulties from recurring. Each time in the future that you and your daughter are asked to complete the measures, you will be paid $25.00 and your daughter will be paid $20.00.

If a participant reports at any time that she is feeling like she would like to hurt herself or someone else, then, she would be immediately interviewed by a trained staff member and the school counselor. In addition, if there is concern about a child's safety, the staff member would immediately contact the parents and Kevin Stark, Ph.D. or one of the

consulting psychiatrists. If at all possible, the psychiatrist on call would be available to meet with the girl and her parents to further evaluate the situation and to provide you with information about resources from within the community that could be of help. If it is not possible to immediately meet with one of the mental health professionals, then it would be recommended that the child and parents pursue the conventional procedure of driving to the emergency room of a local hospital. If a participant reports that she is being hurt, then the staff member and school counselor would follow the school's standard procedures for reporting such instances to the relevant state agency.

All of the services that we provide are available to you at no cost to your family.

The benefits to you and your daughter are that she may learn skills and strategies that will help her to be happy and healthy throughout adolescence. Similarly, you may learn strategies for helping her to successfully make it through adolescence. The benefit to society is that it will help us to determine whether teaching girls who are experiencing depression these skills helps to reduce the depression and whether it is even more helpful to involve parents. Furthermore, since girls are at very high risk for becoming depressed between the ages of 13 to 15, the results of this study will help us learn whether there is a procedure for preventing this from occurring.

The ACTION program meetings are audiotaped for quality assurance purposes. To ensure confidentiality, the following steps will be taken: (a) the cassettes will be coded so that no personal identifying information is visible on them; (b) they will be kept in a locked file cabinet in a secure office at UT; (c) they will be reviewed only for research purposes by the relevant research staff; and (d) they will be erased after they are checked and the study has been completed. Identifying information will be removed from all of the assessment materials completed during the study and the materials will be stored in a locked file cabinet in a locked research office at UT.

Participation in the ACTION coping skills program is entirely voluntary. You are free to refuse to be in the study, you are free to discontinue participation for any reason at any time, and your refusal or discontinuation will not influence current or future relationships with The University of Texas at Austin or your child's school district

Researchers are required by Texas state law and professional ethics codes to report to Child Protective Services (or other appropriate regulatory agency) all instances of alleged child abuse and neglect. Please note that if your child is believed to be at risk for emotional, psychological or possible physical harm or neglect, then the investigator will report this information to the attending physician, Child Protective Services, and any other necessary regulatory agencies. Please note when a child reports neglect or being harmed, participants cannot stop the referral of their child's case to the authorities and any subsequent actions taken.

If you have any questions about the study, you can call Kevin Stark, Ph.D. at (512) 471-4407, your school counselor, or principal. You may also contact the project coordinator, Jennifer L. Hargrave, Ph.D., with questions, concerns, or to withdraw from the study at any time at (512) 471-0218.If you have questions about your rights as a participant, please contact Lisa Leiden, Ph.D., Chair, The University of Texas at Austin Institutional Review Board for the Protection of Human Subjects, (512) 471-8871. Let her know that you are enquiring about the study entitled "Helpfulness of the ACTION Coping Skills Program with and Without Parent Participation."

Please check the appropriate box indicating that **YES** you have read this letter and are giving permission for you and your daughter to participate in the ACTION coping skills program and to complete the questionnaires, or **NO** you do not want to participate in the ACTION coping skills program and you do not want to complete the questionnaires. Regardless of your decision, please sign this form and return it to your child's counselor. With this permission letter, you should have received a copy to keep for your records.

NOTE: TWO COPIES OF THIS LETTER ARE PROVIDED; ONE IS TO KEEP FOR YOUR RECORDS

PLEASE RETURN ONE COPY OF THIS PORTION TO THE SCHOOL COUNSELOR

☐ **YES** I give my permission for my daughter, _____, and me to participate in the ACTION coping skills program and to complete the questionnaires. This includes permission for ACTION staff to access report card information, discipline referrals, and attendance records during participation.

☐ **NO** I do not give my permission for my daughter, _____, to continue any further with the ACTION project.

_____ _____

Parent's Signature Date

_____ _____

Kevin D. Stark, Ph.D. Date

NOTE: TWO COPIES OF THIS LETTER ARE PROVIDED; ONE IS TO KEEP FOR YOUR RECORDS

Appendix Q

Parent Consent and Youth Assent: Control Group

Dear Parent,

You and your child are invited to participate in a study about thoughts, feelings, relationships and psychological adjustment in children and adolescents. We are researchers at The University of Texas at Austin, Department of Educational Psychology. We are looking for children and adolescents to participate in the study. Your child was selected as a possible participant because she is in the relevant age range, and is a student enrolled in the Pflugerville Independent School District. The purpose of this study is to learn more about the relationship between thoughts, behaviors, family characteristics and emotional adjustment. Approximately 25 students from PISD will have an opportunity to participate. Selection for participation will be determined by achieving the closest match in terms of age, gender, ethnicity, and family composition to youngsters who previously participated in the study. This study will be conducted under the supervision of Kevin Stark, Ph.D., a Professor at the University of Texas at Austin and will be coordinated by staff at your child's school. If you and your student are chosen to participate, your family will receive an honorarium of $50.00 immediately following completion of the measures.

Should you decide to participate, a researcher from The University of Texas will ask you and your child to participate in a semi-structured interview regarding your child's feelings and behaviors. For each of you, the interview should take, at most, 45 minutes to complete. You and your child will also be asked to complete a number of questionnaires regarding your child, your family, and yourselves. Your child will be asked to complete a questionnaire that assesses his or her adjustment (Beck Youth Inventory), self-perceptions, things in general and the future (Cognitive Triad Inventory), a questionnaire that assesses your child's thoughts about what causes good and bad things to happen (Children's Cognitive Styles Questionnaire), a questionnaire about his or her perceptions of the way the family works (Self-Report Measure of Family Functioning), a questionnaire about his or her perceptions of messages that parents communicate (Family Messages Measure), and a questionnaire about stressful experiences (Life Events Questionnaire). In addition, your child would be asked to complete a story telling task entitled the Thematic Apperception Test. The school counselor has copies of all of these materials available for your review at this time as well as any time in the future. You would be asked to complete a questionnaire about your own emotional well-being (Symptom Checklist 90-R), a questionnaire about your self perceptions, things in general and the future (Cognitive Triad Inventory) and a questionnaire about your perceptions of the way your family functions (Self-Report Measure of Family Functioning). You and your child may complete the interviews and questionnaires in more than one meeting if you would like to do that. In sum, it would take you approximately an hour and a half to two hours to complete the interview and the measures and a total of 1.5 to 2.5 hours for your child to complete the interview and measures. The interview, questionnaires, and

story telling task are commonly used to evaluate the emotional functioning of youths and adults. They have been completed by hundreds of individuals without any adverse effects. This study will be beneficial in that it should serve to identify psychosocial factors relevant to emotional disorders in children and adolescents, an area largely unexplored to date. Any information in connection with this study that can be identified with you will remain confidential and will be disclosed only with your permission. However, if your child reports an intent to harm herself or others, we would immediately notify the school counselor and you.

For research purposes, we would like your permission to audio-tape the interviews. The tapes are used to determine whether the interview was administered correctly. The tapes will be kept in a locked file cabinet without any identifying information on them and they will be erased once the study has been completed.

Your decision whether or not to participate will not affect your present or future relations with The University of Texas or Pflugerville Independent School District. If you decide to participate, you are free to discontinue participation at any time. Should you decide to allow your child or adolescent to participate, he/she will also have a chance to decide whether or not to participate.

If you have any questions, feel free to contact Dr. Kevin Stark. Dr. Stark can be reached by telephone at 512-471-4407, or in writing: SZB 504, The University of Texas at Austin, Austin, TX 78712. If you have any questions or concerns about your treatment as a research participant in this study, call Professor Clarke Burnham, Chair of the University of Texas at Austin Institutional Review Board for the Protection of Human Research Participants, at (512) 475-7129.

Please keep this form for your records.

*****PLEASE RETURN THIS FORM TO YOUR SCHOOL COUNSELOR*****

You are making a decision whether or not to participate and to allow your child to participate. Your signature indicates that you have read the information provided and have decided to participate and to allow your child to participate should (s)he choose to. By signing this form you are agreeing to participate both by completing the questionnaires and the clinical interview; you are also giving permission for the interview to be audio-taped. You may withdraw at any time after signing this form, should you choose to discontinue participation in this study.

_____ _____

Signature of Parent or Legal Guardian Date

_____ _____

Signature of Staff/Researcher Date

_____

Phone Number (to be contacted over the summer)

*****PLEASE RETURN THIS FORM TO YOUR SCHOOL COUNSELOR*****

Child/Adolescent Assent Form

I agree to participate in a study that is interested in evaluating the relationship between thoughts, feelings, and interpersonal behaviors in children and adolescents. I understand that this study has been explained to my parent or guardian and that he or she has given permission for me to participate. I understand that I may decide at any time that I do not wish to continue this study and that it will be stopped if I say so. Information about what I say and do will not be given to anyone else unless I say so.

I understand that I will be asked to complete an interview about my current feelings, behaviors, and thoughts as well as a number of questionnaires about myself and my family. I understand that by signing this form I am giving permission for the interview to be audio-taped for research purposes and that these tapes will be erased as soon as the study is completed.

I understand that it is all right if I decide to stop my participation in this study at any time. When I sign my name to this page I am indicating that this page was read to me and that I am agreeing to participate in this study. I am indicating that I understand what will be required of me and that I may stop my participation at any time.

_____ _____

Child/Adolescent Signature Date

_____ _____

Staff/Researcher Signature Date

References

Abela, J., & D'Alessandro, D. (2002). Beck's cognitive theory of depression: A test of the diathesis-stress and causal mediation components. *British Journal of Clinical Psychology, 41*, 111-128.

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VITA

Jennifer Ann Herren was born in Nashville, Tennessee, the daughter of Patricia Herren and William Herren. After graduating from Hillwood Comprehensive High School in 1999, she attended Maryville College where she graduated *Summa Cum Laude* in December 2002 with a major in Psychology and minor in Sociology. Jennifer entered the School Psychology Doctoral Training Program at the University of Texas at Austin in the fall of 2003. At the University of Texas, she received the Joseph L. Henderson and Katherine D. Henderson Foundation Student Fellowship (2004-2005) and the Christine Warren Anderson Memorial Fellowship (2005-2006). Jennifer earned her Master of Arts degree in 2007, and her professional experience includes serving as a research assistant and teaching assistant. She will complete her internship at Children's Hospital of Boston in August 2009.

Permanent Address: 7163 Old Harding Road
Nashville, TN 37221

This dissertation was typed by the author.