

SMITH, TAYLOR F., M.A. Risk and Protective Factors Associated with Depressive Symptomatology in Youth with AD/HD. (2009)  
Directed by Dr. Arthur D. Anastopoulos. 71 pp.

Youth diagnosed with Attention-Deficit/Hyperactivity Disorder (AD/HD) are at approximately five-times greater risk for developing depression (Angold, Costello, & Erkanli, 1999). In an effort to explain this relationship, both risk and protective factors from the Failure Model (Patterson & Capaldi, 1990) were examined in an AD/HD sample. A total of 30 youth with rigorously defined AD/HD and their maternal caregivers participated in this cross-sectional study. Consistent with study hypotheses familial risk (i.e., maternal depression, youth aggression, and negative parenting practices) was associated with increased depressive symptomatology, whereas youth perceived competence was associated with decreased depressive symptomatology in AD/HD youth. These findings suggest that the Failure Model (Patterson & Capaldi, 1990) has utility in explaining the relationship between AD/HD and depression.

RISK AND PROTECTIVE FACTORS ASSOCIATED WITH DEPRESSIVE  
SYMPTOMATOLOGY IN YOUTH WITH AD/HD

by

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A Thesis Submitted to  
The Faculty of The Graduate School at  
The University of North Carolina at Greensboro  
in Partial Fulfillment  
of the Requirements for the Degree  
Master of Arts

Greensboro  
2009

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To my parents, Leigh and Mary Smith.

APPROVAL PAGE

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## ACKNOWLEDGMENTS

I would like to thank my advisor, Dr. Arthur Anastopoulos for his guidance and valuable contribution to this project. In addition, a special thanks to my committee members, Dr. Susan Calkins and Dr. Julia Mendez, for their advice. I am also grateful to Dr. Erin Kane, Dr. Joanne Joseph, Dr. Mark Lewis, Dr. Susan Farrell and Nicole Schatz who assisted in recruitment efforts. Lastly, thank you to those families who kindly volunteered to serve as participants.

## TABLE OF CONTENTS

	Page
LIST OF TABLES .....	v
LIST OF FIGURES .....	vi
CHAPTER	
I. INTRODUCTION .....	1
II. METHOD .....	19
III. RESULTS .....	30
IV. DISCUSSION .....	38
REFERENCES .....	46
APPENDIX A. TABLES AND FIGURES .....	62
APPENDIX B. MEASURES .....	70

## LIST OF TABLES

	Page
Table 1. Sample Characteristics. ....	62
Table 2. Diagnostic and Treatment Sample Characteristics. ....	63
Table 3. Correlations among Predictor Indices/Variables and Outcome Variables . . . .	64
Table 4. Summary of Hierarchical Regressions for the Failure Model Predicting Composite Youth Depression . . . . .	65
Table 5. Summary of Hierarchical Regressions for the Failure Model Predicting Youth Self-Report of Depression . . . . .	66
Table 6. Summary of Hierarchical Regressions for the Failure Model Predicting Mother-Rated Youth Depression . . . . .	67

## LIST OF FIGURES

	Page
Figure 1. The Failure Model with latent variables from Patterson and Capaldi (1990). . . . .	68
Figure 2. Simple slopes analysis for maternal-rated youth depression on familial risk at levels of youth perceived competence. . . . .	69

## CHAPTER I

### INTRODUCTION

Attention Deficit/Hyperactivity Disorder (AD/HD) is diagnosed in 3-7% of the childhood population (American Psychiatric Association, 2000). In addition to the primary symptoms of the disorder (i.e., inattention, hyperactivity and impulsivity), AD/HD is also associated with a number of secondary deficits, including dysfunction in academic, behavioral, emotional, familial and social domains (e.g., Anastopoulos & Shelton, 2001). Along with increasing risk for more severe externalizing behavior trajectories, especially the development of Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD; August, Realmuto, MacDonald, Nugent & Crosby, 1996), AD/HD also increases risk for future internalizing disorders such as depression (e.g., Jensen, Shervette, Xenakis & Richters, 1993).

Although some argue that the increased rates of co-occurrence result from AD/HD-related impairment and not “true depression” (Hoza, Pelham, Milich, Pillow, & McBride, 1993), the majority of evidence recognizes that children with AD/HD are at heightened risk for developing depression. Moreover, youth with AD/HD and comorbid depression are more impaired than youth with AD/HD or depression alone (e.g., Lewinsohn, Rohde, & Seeley, 1996). In community samples, ~13.7% of youth diagnosed with AD/HD, compared to ~2.7% of the general population, have a depressive disorder.

In other words, youth with AD/HD are at approximately five-times greater risk for depression compared to their same aged peers (Angold et al., 1999).

Despite being at heightened risk, many youth with AD/HD do not develop depressive disorders. Therefore, it is important not only to assess factors that put children with AD/HD at risk for depression, but also factors that protect these children against the development, relapse or maintenance of depression. Unfortunately, the study of resilient outcomes in children with AD/HD has been limited. An understanding of the route to adaptation in youth with AD/HD is critical to conceptualizing the varied developmental outcomes of this at-risk population (Rhoads, 2006). Therefore, the purpose of this project was to better understand the relationship between AD/HD and comorbid depression by examining both risk and protective factors. Since little is known about the etiological specificity between early onset Major Depressive Disorder (MDD) and Dysthymic Disorder (DD), and considering the two depressive disorders are commonly comorbid (e.g., McGee, Williams, & Feehan, 1990), this project examined the severity of depressive symptom dimensions across categories.

As background for this study, it is first necessary to provide overviews of AD/HD, depression and the comorbidity of AD/HD and depression. Next, the concept of resiliency is introduced and factors associated with risk for, and protection against, depression are reviewed. Then a conceptual overview of the link between AD/HD and depression is presented. Finally, the research questions and hypotheses of this study are stated within a risk and protection framework.

## *AD/HD and Depression*

### *Etiology*

AD/HD is a polytypic syndrome that results from multiple causes of mostly biological origins (Swanson, Castellanos, Murias, LaHoste, & Kennedy, 1998). Despite the varied presentation of the disorder, some proposed etiological commonalities have surfaced. An underlying genetic susceptibility seems to be the most common factor related to the genesis of AD/HD, though environmental components should not be overlooked. Evidence from family, twin, adoption, association and linkage studies indicate that AD/HD is of predominantly genetic origin (Brassett-Harknett & Butler, 2007). Though single genes have only small to medium effect sizes across the population, genes associated with the transmission of dopamine (e.g., DAT1, DRD4) and other neurotransmission systems have been major foci of study. Conceptually, such genes may be precursors to a dopamine deficiency (Pliszka, McCracken, & Maas, 1996), which in turn may lead to neurophysiological anomalies (Castellanos, Giedd, Marsh, & Hamburger, 1996) in specific brain regions (e.g., frontal lobe and basal ganglia). Although structural and functional imaging studies do not provide a universal explanation for the phenotypic variability of AD/HD, areas that are consistently identified as dysfunctional (e.g., dorsolateral prefrontal cortex) map onto the executive dysfunction characteristics of AD/HD (Barkley, 1997).

In contrast to the presumed genetic/neurobiological etiology of AD/HD, there is a greater emphasis on the etiological heterogeneity of depression. This is reflected in multifactorial and transactional theories of depression in youth (e.g., Cicchetti & Toth,

1998; Hammen & Rudolph, 1996; Hankin & Abela, 2005). In adult research there is consensus in regard to neuroanatomical, neurochemical and neuroendocrine correlates of depression; however, the data in children are far less consistent (Kaufman, Martin, King, & Charney, 2001) and for that reason will not be elaborated upon here. Instead, major theories of child and adolescent depression tend to incorporate transactional relationships between biological, cognitive, social and emotional factors (e.g., Patterson & Capaldi, 1990; Hammen & Rudolph, 1996; Cicchetti & Toth, 1999; Hankin & Abela, 2005).

#### *Developmental Course*

The mean age of onset for AD/HD is between 3 and 4 years of age (Mcgee, Williams, & Feehan, 1992). Although hyperactive-impulsive symptoms typically occur earlier than inattentive symptoms, they appear to attenuate with age (DuPaul, Power, Anastopoulos, Reid, McGoey, & Ikeda, 1997). Although symptom presentation may change over time (i.e., heterotypic continuity; Cicchetti & Rogosch, 1997), AD/HD often persists into adulthood. Around 50-80% of children display clinically significant symptom levels into adolescence (Barkley, Fischer, Edelbrock, & Smallish, 1990), which persist into adulthood, albeit at relatively lower rates (Mannuzza, Klein, Bessler, Malloy, & Hynes, 1997).

In contrast to AD/HD, depression generally does not begin in early childhood. Though the age of onset appears to be decreasing for individuals born more recently, currently the mean age of onset for MDD is in the mid-twenties (American Psychiatric Association, 2000). There is great variability in the onset of depression but trends in the course of the depression do exist. For instance, prevalence for depression increases

drastically during adolescence, especially within females (Lewinsohn, Clarke, Seeley & Rohde, 1994).

### *Co-Occurrence of AD/HD and Depression*

AD/HD and depression are individually associated with long-term morbidity, heightened risk for comorbidity (e.g., Conduct Disorder) and impairment in multiple domains. The co-occurrence of AD/HD and depression significantly exacerbates this risk and suggests that a subpopulation of children diagnosed with AD/HD and depression are at heightened risk for long term morbidity, comorbidity, impairment and for attempting and completing suicide (Weinberg, McLean, Snider, & Nuckols 1989; Biederman, Faraone, Milberger, & Guite, 1996; Brent, Perper, Goldstein et al., 1988, Lewinsohn, Rohde, & Seeley, 1993). The seemingly synergistic maladaptive consequences of AD/HD and depression are alarming. The combination of a deficit in impulse control, considered to be the core deficit of AD/HD (Barkley, 1997) and symptoms of early onset depression puts these individuals at heightened risk for behavior that is self-defeating (e.g., suicide).

Understanding the temporal sequence of AD/HD and comorbid depression will provide insight into the nature of the complex phenomena by providing clues to potential mechanisms that may intercede the onsets of the disorders. AD/HD is believed to have early developmental origins and symptoms appear before the age of seven. In contrast, depression generally has a later onset and prevalence rates tend to increase with age (American Psychiatric Association, 2000). In addition, early externalizing behavior problems positively predict later internalizing problems, whereas early internalizing

problems negatively predict later externalizing problems once earlier externalizing problems are controlled for (Gjerde, 1995; Gilliom & Shaw, 2004; Mesman, Bongers, & Koot, 2001). In addition, AD/HD has been shown to be primary among affective and behavioral disorders, meaning it is not predicted by previously existing disorders but is a starting point of heterotypic continuous pathways that involve ODD, CD, anxiety and depression (Burke, Loeber, Lahey, & Rathouz, 2005). Furthermore, since the most proximal etiological factors of depression tend to emerge later in development (e.g., negative cognitions; Hammen & Rudolph, 1996; Hankin & Abela, 2005), it seems less likely that children would develop depression prior to, or concurrent with, AD/HD. Taken together, the distinctions between the onset, course and etiology of the disorders suggests at least in the majority of cases, the onset of AD/HD precedes the onset of depression.

Currently, it is unclear whether AD/HD and depression are comorbid due to an underlying shared etiology or whether depression results from AD/HD related impairment. Biederman, Mick and Faraone (1998) maintain that AD/HD related depression represents “true depression” and not just AD/HD associated demoralization. AD/HD and comorbid depression were found to have independent and distinct courses, and AD/HD features (i.e., symptom severity and school difficulty) were not significant predictors of major depression persistence. Therefore, the authors argued that AD/HD and depression have a partially shared genetic etiology, a view that is corroborated by the familial comorbidity between AD/HD and depression (Faraone & Biederman, 1997; Nigg & Hinshaw, 1998). Alternatively, others argue that negative sequelae related to AD/HD

in family (i.e., maladaptive parent management techniques; Ostrander & Herman, 2006) and social (Ostrander, Crystal, & August, 2006) domains were found to separately mediate the relationship between AD/HD and depression. In contrast to previous findings, this suggests that AD/HD related impairment may be a causal mechanism linking AD/HD and depression.

### *Risk and Protection*

There are many possible explanations for the comorbidity between AD/HD and depression. Risk factors for depression (e.g., maternal depression, maladaptive parenting and aggression) seem to congregate in individuals diagnosed with AD/HD. Considering the additive/synergistic nature of risk factors (Sameroff, Seifer, Zax, & Barocas, 1987), the congregation of multiple risk factors, across the ecology, is especially alarming.

Therefore to help prevent the development of depression within the AD/HD population, it is important to first understand what factors put these individuals at risk for, and protect them against depression.

Research on risk and protective factors associated with childhood depression has focused on children of parents with depressive disorders and children diagnosed with, or having symptoms of, depression. Though youth with AD/HD are overrepresented in the aforementioned groups, they constitute a distinct group of children. This distinct subset of children at-risk for depression may respond differently to factors of risk and protection. Additionally, this subgroup of at-risk children may have risk and protective factors that are highly salient in their specific risk setting.

Luthar (2006) refers to resilience as the phenomenon of “positive adaptation despite experiences of significant adversity or trauma (p. 742).” Clinically disordered populations are not often thought of as being positively adapted. The majority of resiliency research has been conducted on populations exposed to environmental stress (e.g., maternal depression and child maltreatment) and individuals who are considered resilient do not develop psychopathology (e.g., depression). Despite previous precedent, children with AD/HD are at increased risk for developing depression and therefore constitute a population capable of displaying resiliency, in this case emotional resiliency (Kline & Short, 1991). To understand the route to resilient outcomes, it is important to identify factors that make the at-risk population vulnerable to maladaptation. Though the terms *risk* and *vulnerability* tend to be used interchangeably, this paper will use the term “risk factors” to denote variables that exacerbate risk for depression among youth with AD/HD (Luthar, 2006).

Considering the multitude and severity of risk factors associated with depression in youth with AD/HD, it is surprising that the rates of comorbidity are not higher between the disorders. To mitigate risk between AD/HD and depression, factors must be present that protect children from the adverse influences related to the disorder. Protective factors alter the effects of risk in a positive direction (Luthar, 2006). For instance, these factors may be protective (i.e., protective main effect of factor across levels of risk) or protective-stabilizing (i.e., factor confers stability in competence despite increased risk; Luthar, Cicchetti, & Becker, 2000), and general (i.e., reduce risk for general psychopathology) or specific (i.e., reduce risk for just depression). Though the majority

of risk and protective factors are bipolar in nature, and confer risk and protection at opposite poles (Masten, 2001), the next section discusses risk and protective factors to be consistent with their valence in this study (Cohen, Cohen, Aiken & West, 2002).

### ***Risk Factors***

#### *Parents*

Parents of children with AD/HD are at increased risk for depression, AD/HD, anti-social personality disorder, anxiety disorders, alcoholism and learning disorders (Faraone & Biederman, 1997; Nigg & Hinshaw, 1998). Although research addressing the impact of each of these specific psychopathologies on parent-child relations is still emerging, the negative parenting sequelae associated with maternal depression is better established. For instance, depressed mothers display more negative (e.g., negative affect, criticism, negative facial expression) and disengaged behavior (e.g., ignoring, withdrawal) and fewer positive parenting behaviors (e.g., pleasant affect, praise, and affectionate contact) than non-depressed mothers (Lovejoy, Grazyk, O'Hare, & Neuman, 2000). These maladaptive parental behaviors are consistent with those seen in parents of children with AD/HD (Danforth, Barkley, & Stokes, 1991; DuPaul, McGoey, Eckert, & VanBrakle, 2001).

#### *Comorbid Externalizing Disorders*

Although AD/HD is clearly associated with depression above what would be expected by chance (Angold et al., 1999), depression is also strongly associated with ODD and CD. Therefore, the relationship between AD/HD and depression may resemble an epiphenomenon (Angold et al., 1999) that develops due to the association between

AD/HD and ODD/CD. When additional externalizing comorbidity is considered, much of the association between AD/HD and depression seems to be accounted for by the presence of ODD/CD (Angold et al., 1999; Burke et al., 2005; Fischer, Barkley, Smallish, & Fletcher, 2002; Treuting & Hinshaw, 2001).

#### *AD/HD Symptoms or Subtype*

Although AD/HD subtypes have been shown to be differentially associated with comorbid profiles (Milich, Balentine, & Lynam, 2001), traditional AD/HD subtyping (e.g., AD/HD-combined type, predominantly Inattentive type and predominantly Hyperactive-Impulsive type) is ill-suited for predicting comorbid depression, cross-sectionally. Since symptoms of inattention are related to both AD/HD and depression it would be difficult to determine how the distinct disorders individually impact inattention. Angold et al. (1999) have argued that increased vulnerability for comorbid outcomes may result from increased symptom severity, though in regard to AD/HD and depression, evidence is mixed (e.g., August et al., 1996; Jensen et al., 1993).

#### *Negative Life Events*

Negative life experiences, such as divorce, are found to independently contribute to depression (e.g., Kendler & Karkowski-Shuman, 1997; Kim, Conger, Elder, & Lorenz, 2003). The combination of chronic stressors and episodic stressors is likely to put a child at heightened risk for depression, even if the child has acquired appropriate coping skills (Hammen, Burge, & Adrian, 1991). Not only are children with AD/HD at increased risk of implementing maladaptive coping strategies (Melnick & Hinshaw, 2000) but they are

also at increased risk for experiencing negative life events such as divorce, marital conflict, and accidental injuries (Barkley, 2006).

### *Demographics*

In the general population, risk for depression tends to increase from childhood to adolescence, especially for females (e.g., Lewinsohn et al., 1994). In addition, low SES has been implicated in the development of child and adolescent depression in community samples (Garrison, Schluchter, Schoenbach, & Kaplan, 1989). Such factors are presumed to act similarly on youth with AD/HD.

### *Biology*

Parental psychopathology puts children at both biological and environmental risk for maladaptive outcomes. One possible explanation for the comorbidity is a shared genetic link between depression and AD/HD (Biederman et al., 1998), but since biological risk is not a focus of this study, it will not be elaborated upon here.

## ***Protective Factor***

### *Perceived Competence*

Youth with AD/HD are described as having perceptions of competence that are higher than are warranted (Hoza et al., 1993) and are heightened compared to control children (Hoza et al., 1993; Hoza, Pelham, Dobbs, Owens, & Pillow, 2002). These “adaptive” perceptions are utilized when AD/HD youth are confronted with failure and may represent contextually salient adaptation to protect against depression (Diener & Milich, 1997). Previous research has demonstrated that perceptions of competence are enhanced in academic and social domains, and may buffer against the development of

depression in middle childhood (Hoza et al., 2002). Since inflated perceptions of competence are associated with many negative outcomes including academic difficulty, aggression and psychopathy (Robins & Beer, 2001; Bushman & Baumeister, 1998), one must be cautious claiming that positive illusions lead to a resilient outcome. If defensive processing is overly persistent, what is considered emotionally adaptive in the present can give way to general maladaptation over time (McEwen & Lasley, 2003).

### ***Theory: From AD/HD to AD/HD and Depression***

A thorough theoretical explanation of how a child who develops AD/HD goes on to develop depression must allot for multiple factors, which interact to form probabilistic pathways leading towards and away from depression. Though increased risk for depression is theorized to be due, in part, to pre-existing vulnerabilities (e.g., genetic risk), given study limitations (i.e., cross-sectional nature and focus on psychosocial variables) preexisting vulnerabilities will not be elaborated upon here; instead, emphasis will be placed on AD/HD related impairment.

To account for the strong relationship between ODD/CD and depression in youth, Capaldi and colleagues (Patterson & Capaldi, 1990; Capaldi, 1991; Capaldi, 1992; Capaldi & Stoolmiller, 1999) developed the Failure Model (see Figure 1). The model posits that difficulty in family, academic and peer domains emanate from conduct problems. This combined with the experience of consistent failure and the associated lack of positive reinforcement from one's environment increases risk for depression in this population. Regardless of ODD/CD diagnoses, youth with AD/HD are at increased risk for negative transactions across family, school and peer domains; therefore, this

study will examine the viability of the Failure Model in an AD/HD population. Considering the association between AD/HD and depression can be largely accounted for by the presence of ODD/CD (Angold, et al., 1999), it could be argued that if the relationship is epiphenomenal, then it is unnecessary to expand the Failure Model to an AD/HD population. On the contrary, the Failure Model has utility in explaining the relationship between AD/HD and depression because similar to ODD/CD, AD/HD leads to shared and unique maladaptation across family, social and academic domains. Considering differences in the onset, course and nature of impairment across the disruptive behavior disorders, the Failure Model may have strong explanatory value beyond ODD/CD and may provide a framework for understanding risk for depression in an AD/HD population. Finally, considering the varied trajectories of this at-risk sample, this study looks to elaborate upon the Failure Model to show that both maladaptation and adaptation within relevant domains informs risk for/protection against depression.

Consistent with operant conditioning principles, the Failure Model (Patterson & Capaldi, 1990; Capaldi, 1991; Capaldi, 1992; Capaldi & Stoolmiller, 1999), posits that disruptive behavior (e.g., aggression) undermines parental attempts to control behavior, and over time allows disruptive youth to escape punishment, which in turn negatively reinforces disruptive behavior (e.g., Patterson, 1982). Within this maladaptive transaction, disruptive behavior (e.g., noncompliance, aggression, defiance) is termed coercive behavior as it functions to gain social attention, tangible rewards, or getting one's way (Snyder and Stoolmiller, 2002). In turn, the child's coercive behavior results in poor parenting practices, which tend to reward negative child behavior and ignore (fail

to reward) positive behavior (e.g., Snyder and Stoolmiller, 2002) producing parental rejection (Patterson & Capaldi, 1990). To complete the coercive cycle, poor parent management skills further exacerbate disruptive behavior (Patterson & Capaldi, 1990), which helps to explain how children with AD/HD are at increased risk for comorbid disorders, most notably ODD and CD. The reciprocal nature of the coercive cycle has garnered empirical support from experimental studies. For instance, administering stimulant medication to AD/HD youth is associated with better parenting practices (positive attention; Barkley & Cunningham, 1979) and improving parenting practices through Parent Training programs results in improvement of disruptive behavior problems (Pelham & Fabiano, 2008). In addition to parenting practices, boys' externalizing behavior problems are reciprocally associated with maternal depression across time (Gross, Shaw, & Moilanen, 2008).

The Failure Model also posits that disruptive behavior leads to peer rejection and poor academic skills which further increase risk for depression. Coercive parent-child transactions generalize to peer relationships (Stoolmiller, Duncan, & Patterson, 1993; Ramsey, Patterson, & Walker, 1990). For example, AD/HD youth's behavior (e.g., inattentive, off-task) tends to result in controlling and negative behavior from peers (Hinshaw & Melnick, 1995; Clark, Cheyne, Cunningham & Siegel, 1988), especially if the child is aggressive and noncompliant (e.g., Hinshaw & Melnick, 1995), often resulting in social rejection and isolation for boys (Pelham & Bender, 1982; Milich, Landau, Kilby, & Whitten, 1982) and girls (Mikami & Hinshaw, 2003). Again, operant conditioning principles in the peer domain increase risk for peer rejection and reactive

aggression. Impairment that is uniquely related to AD/HD (e.g., being off task and day dreaming) also seems to be strongly associated with peer rejection (Coie, Dodge, & Kuppersmidt, 1990) and differentiates youth who are only aggressive, only rejected or both (Bierman & Wargo, 1995). These findings suggest that in order to better understand the variability in social functioning within youth with disruptive behavior problems, impairment that is uniquely related to AD/HD should be accounted for (Waschbusch, Pelham, Jennings, Greiner, Tarter, & Moss, 2002). Over time the absence of pro-social skills and presence of maladaptive social behavior in AD/HD youth promote dysfunctional interactions with peers. This leads to peer rejection, messages of social incompetence and a dearth of positive reinforcement from the social environment, which together increase risk for depression.

Familial risk may also lead to impairment in the academic domain for youth with AD/HD (Hinshaw, 1992). Academic achievement in an AD/HD population is strongly related to cognitive impairment that is unique to AD/HD (i.e., vigilance and short-term memory) and not significantly related to ODD/CD (Rapport, Scanlan, & Denney, 1999). This provides additional support for examining the viability of the Failure Model in an AD/HD population. Furthermore, in the AD/HD population, early academic achievement and aptitude is directly predictive of later emotional and behavioral adjustment (Latimer, August, Newcomb, Realmuto, Hektner, & Mathy, 2003). Taken together, this suggests that AD/HD related impairment hinders academic achievement in AD/HD youth, and over time youth with AD/HD are at greater risk to receive negative objective and subjective feedback about their competence in school.

Similar to adult theories of depression that emphasize excessive punishment and lack of reinforcement (e.g., Seligman, Abramson, Semmel & von Baeyer, 1979), youth who receive negative evaluation across developmentally relevant domains (Masten et al., 1995) may begin to view themselves as incompetent. The negative feedback may lead to maladaptive self-perceptions, which in turn, increases depressive symptomology in children and adolescents (Cole, 1990). The addition of a cognitive component to the Failure Model helps to explain the spike in prevalence rates of depression in adolescence. As adolescents may have a more differentiated self-concept, focal deficits in social and academic domains may become more relevant during this period of development (Hankin & Abela, 2005). Furthermore, the addition of a cognitive component in the Failure Model helps to explain the varied developmentally trajectory of youth with AD/HD as at least some youth diagnosed with AD/HD have developed a compensatory mechanism of inflated self-perceptions in the face of negative feedback (Diener & Milich, 1997).

Taken together, impairment uniquely related to AD/HD is associated with competency in family, school and peer domains. Thus, the Failure Model (Patterson & Capaldi, 1990) may also provide a framework for understanding the relationship between AD/HD and depression. The maladaptive distribution of behavioral contingencies in the family domain contributes to an environment of relatively greater risk. If in the face of familial adversity youth are still capable of feeling competent with peers and at school, such risk may be mitigated. Finally, broadening the Failure Model to include competencies, instead of “failures” per se, allows for greater explanation of the varied developmental trajectories of this population.

### *Research Questions and Hypotheses*

In review, children with AD/HD are at increased risk for developing depression. Not only do they seem to have an underlying genetic vulnerability to depression (Faraone & Biederman, 1997; Nigg & Hinshaw, 1998) but their environments also confer a multitude of additional risk factors. Considering the additive or synergistic nature of risk and protective factors (Sameroff et al., 1987), it may be helpful to view such factors collectively. For instance, within the Failure Model (Patterson & Capaldi, 1990), *Familial Risk* for youth depression may represent a congregation of maternal depression, negative parenting practices, and youth aggression. Similarly, youth perceived competence in social and academic domains may be viewed as youth *Perceived Competence* in developmentally relevant domains of functioning. Risk modifiers can be highly influential in some settings but not in others (Luthar, 2006). Therefore, if appropriate interventions are to be designed, it is extremely important to identify malleable factors associated with depression within an AD/HD population.

In response to this situation, this study addressed the question: *What factors contribute to the variability in depressive symptomatology within an AD/HD population?* Specifically this study examined factors of risk and protection that are both highly salient in AD/HD and malleable (i.e., responsive to environmental intervention). Thus, in an effort to explain the complex phenomenon of AD/HD and comorbid depression, the utility of the Failure Model (Patterson & Capaldi, 1990) was examined in an AD/HD population, with particular attention to the following hypotheses:

- Hypothesis 1 - Consistent with previous research in the ODD/CD population, *Familial Risk* for depression within AD/HD youth was hypothesized to be related to increased youth depressive symptomatology.
- Hypothesis 2 - In an extension of previous findings, youth *Perceived Competence* in the academic and social domains was hypothesized to predict lower levels of youth depression, above and beyond familial risk.
- Hypothesis 3 - Youth *Perceived Competence* was predicted to demonstrate enhanced protective benefits from youth depression, at high levels of *Familial Risk*.

## CHAPTER II

### METHOD

#### *Participants*

Males and females between the ages of 8 and 16 years old, with a documented history of AD/HD were recruited for participation in this study. At the time of assessment all youth needed to meet full Diagnostic and Statistical Manual of Mental Disorders-4<sup>th</sup> Edition (DSM-IV TR; American Psychiatric Association, 2000) criteria for AD/HD to be eligible for the project. AD/HD diagnostic status was confirmed from: 1) a positive AD/HD diagnosis on the mother-completed Computerized Diagnostic Interview Schedule for Children (C-DISC-IV; Shaffer, Fisher, Lucas, Dulcan & Schwab-Stone, 2000); and 2) T-scores at or above 65 on either the Attention or Hyperactivity clinical scales on the Behavior Assessment System for Children Second Edition - Parent Rating Scale (BASC-2 PRS; Reynolds & Kamphaus, 2004). Although all AD/HD subtypes were included in the study, to reduce AD/HD taxonomic heterogeneity (see Milich et al., 2001), youth were excluded if the maternal caregiver endorsed less than 3 hyperactive-impulsive symptoms. Since internalizing and externalizing disorders are of interest to this study, children with previous or current externalizing (e.g., ODD) or internalizing disorders (e.g., MDD) were included.

Fifty youth and their parents initially agreed to participate in this study. However, due to the fact that participants either failed to return questionnaires or meet criteria for

the study, the final sample included a total of 30 youth diagnosed with AD/HD and their maternal caregiver. A summary of demographic characteristics for the sample are presented in Table 1. Youth participants ranged from 8 to 16-years-old ( $M = 10.8$ ,  $SD = 2.4$ ). Compared to the community as a whole, the sample was more racially/ethnically homogenous, as 80% of the youth in the sample identified as Caucasian ( $N = 24$ ) and 20% of the youth in the sample identified as African-American ( $N = 6$ ). On average participating families were of slightly higher Socioeconomic Status (SES;  $M = 46.6$ ,  $SD = 13.9$ ) as measured by the Hollingshead Four Factor Index (Hollingshead, 1975), than the community as a whole.

At the time when measures were completed, the entire sample had a documented history of an AD/HD diagnosis. Consistent with gender prevalence rates for the AD/HD population at large (American Psychiatric Association, 2000), this sample was 77% male ( $N = 23$ ) and 23% female ( $N = 7$ ). The majority of youth in the study met criteria for AD/HD Combined Type (80%;  $N = 24$ ), followed by Inattentive Type (17%;  $N = 5$ ) and Hyperactive-Impulsive Type (3%;  $N = 1$ ). Considering individuals with fewer than 3 Hyperactive-Impulsive symptoms were excluded, these findings are consistent with what would be expected in the AD/HD population (Biederman, Faraone, Weber, Russell, Rater & Park, 1997).

Parent report of youth diagnostic history for depression was attained from 25 of the 30 participating families. Of those reporting, 32% ( $N = 8$ ) of maternal caregivers believed that their youth should have received treatment for depression, whereas only 8% ( $N = 2$ ) of the youth sample had received a formal diagnoses of MDD. Consistent with

the predicted developmental sequence of AD/HD and depression, the reported (perceived or documented) onset for depression followed the reported onset of AD/HD ( $M = 3.7$  years;  $SD = 2.5$ ).

In terms of treatment, 70% ( $N = 21$ ) of youth from the reporting sample was taking medication for AD/HD. Of those taking medication for AD/HD, 89% ( $N = 16$ ) were taking stimulant medication, and 11% ( $N = 2$ ) were taking non-stimulant medication (i.e., *Strattera*<sup>®</sup>). Youth in the sample were also taking other psychotropic medication including an antiepileptic drug for mood stabilization ( $N = 2$ ), a mood stabilizer ( $N = 1$ ) and an anti-hypertensive for aggression ( $N = 1$ ). In addition 63% of youth, had received psychosocial treatment ( $N = 15$ ; i.e., individual, group or family therapy) for an average duration of 34.6 months ( $SD = 41.1$ ) and 46% of maternal caregivers had received some form of parent training ( $N = 11$ ). A summary of these sample diagnostic and treatment characteristics is provided in Table 2.

*Materials: AD/HD Diagnostic Status*

*Computerized Diagnostic Interview Schedule for Children – IV Parent Version* (C-DISC-IV; NIMH, 1997). The C-DISC-IV is a computerized structured diagnostic interview that assesses a broad range of youth psychopathology based on current DSM-IV criteria. An interviewer reads each item to the parent, who provides a yes or no response indicating whether or not the item applies to their child. The AD/HD module of the DISC-IV has been demonstrated to have adequate test-retest reliability in clinic samples (.79; Shaffer et al., 2000). Research on a previous version of the DISC investigated the relationship between interview diagnoses and clinician ratings, and

reported adequate criterion validity ( $\kappa = .72$ ) for the AD/HD module (Schwab-Stone, Shaffer, Dulcan, & Jensen, 1996). Mothers' responses to the C-DISC-IV AD/HD were used to determine eligibility for this research project.

*Behavior Assessment System for Children, 2<sup>nd</sup> Edition Parent Rating Scale* (BASC-2 PRS; Reynolds & Kamphaus, 2004). The BASC-2 is a broad-band rating scale that assesses a wide range of youth psychopathology and adaptation. The child version was administered to mothers with youth ranging from 8 to 11 years-old while the adolescent version was administered to mothers of youth from 12 to 16 years-old. Dependent on the version, the scale contains 150-160 items. The respondent rates the frequency of each item on a 4-point scale, from "never" to "almost always." It yields 9 clinical scales (e.g., attention, hyperactivity, and aggression scales), 5 adaptive scales, and 4 composite scores. Both the Hyperactivity and Attention Problems scales are positively skewed, which allows increased sensitivity in differentiating youth at the severe end of these continuous dimensions. The Attention Problems and Hyperactivity scales were used to establish the developmental deviance of the primary symptoms of AD/HD.

#### *Outcome Measures*

*BASC-2 PRS Depression Scale* (Reynolds & Kamphaus, 2004). The clinical Depression scale on the BASC-2 was used to measure mother rated, youth depression. Reynolds and Kamphaus (2004) report that the depression subscale is internally reliable ( $\alpha = .90$ ) within an AD/HD population and has adequate test-retest reliability (i.e., from

.85-.87). The scale also shows good convergent validity with similar scales and discriminant validity with externalizing problems (e.g., hyperactivity).

*Child Depression Inventory (CDI; Kovacs, 1992).* The CDI is a widely used measure of self-reported depression in youth between the ages of 7 and 17. This 27-item scale, which is based on a three point scale (i.e., 0 indicates the absence of a symptom, 1 indicates the mild presence of a symptom and 2 indicates a definite symptom) examines the severity of depressive symptomatology. The scale has been shown to have adequate test-retest reliability (.74-.83), internal consistency (.71-.89) and discriminant and concurrent validity (Kovacs, 1992). Internal consistency within this sample ( $\alpha=.84$ ) is consistent with previous findings. Scores on the CDI range from 0-54 with higher scores indicating greater depression severity. Scores of 19 or higher are considered to be associated with clinically significant depression. In this study, the total score T-score, based on age and gender, was used as a measure of youth depression. In addition to youth self-report of depression, the CDI was modified to the perspective of a parent, and mothers rated their child on perceived depressive symptomatology.

#### *Predictors Variables – Risk*

*Beck Depression Inventory – II (BDI-II; Beck, Steer, & Brown, 1996).* This 21-item inventory measures frequency and severity of adult depressive symptomatology. Responses range from zero to three indicating increases in the severity of depression. For each item, mothers identified the statement that most closely matched their feelings over the past two weeks. This widely used measure of depression has a range from 0-63, with higher scores indicating more severe depression. The scale has demonstrated adequate

test-retest reliability, concurrent validity (Beck et al., 1996) and high internal consistency (Beck, Steer, Ball & Renieri, 1996). The raw total score served as a measure of maternal depression.

*BASC-2 PRS Aggression scale (Reynolds & Kamphaus, 2004).* The Aggression scale measures the propensity for an individual to do physical or emotional harm to others by assessing both verbal and physical aggression (Reynolds & Kamphaus, 2004). Reynolds and Kamphaus (2004) reported that the aggression subscale has excellent internal consistency within an AD/HD population ( $\alpha = .88-.91$ ) and test-retest reliability (.72-.84). The aggression subscale has good convergent validity with similar measures (e.g., Conner's Parent Rating Scales; Conners 1997). In addition, the distribution of the aggression subscale is positively skewed allowing for sensitivity to degree of aggression in the more severe range. Higher scores on the aggression scale represent higher levels of aggressive behavior, and T-scores served as predictors of youth depression.

*Alabama Parenting Questionnaire (APQ; Shelton, Frick, & Wootton, 1996).* The APQ is a multidimensional measure of parenting practices. The mother reported on her own parenting behavior. The questionnaire is made up of 42 questions and is based on a five point frequency scale (from 1 = never to 5 = always). The questionnaire consists of five different subscales, which include involvement, positive parenting, poor monitoring/supervision, inconsistent discipline and corporal punishment. The scales have moderate internal consistencies ( $\alpha = .47$  to  $.80$ ) and have been normalized on children (from 6-13 years-old), in both clinic and community samples (Shelton et al., 1996). These subscales have strong external validity in predicting child externalizing

problems. The measure has also been found to have good test-retest reliability and construct validity (Shelton et al., 1996).

*Modified Version of the Life Events Checklist (LEC; Johnson & McCutcheon, 1980).* Negative life events were measured using a modified version of the LEC which did not include 12 developmentally inappropriate items for children from the original version (10, 24, 25, 26, 28, 32, 38, 39, 41, 42, 43, and 46). The modified version of the LEC consisted of 34 positive and negative life events appropriate for children and adolescents. On each item, the youth marks whether or not the event had occurred in their life in the past 12 months. Next, if the event occurred the youth indicates whether the event was good or bad and how large of an impact the event had on their life (no effect, small effect, moderate effect, or large effect). The LEC produces both a positive and negative change score which are calculated by summing the impact of both positive and negative events, respectively. The negative change score has related to a number of different outcomes (e.g., depressive symptoms; Johnson & McCutcheon, 1980) and served as predictor of youth vulnerability.

*ADHD Rating Scale-IV (ADHD-RS; DuPaul, Power, Anastopoulos, & Reid, 1998).* The ADHD RS-IV is an 18-item questionnaire based on the current DSM-IV criteria for AD/HD. Respondents rate the occurrence of each symptom on a scale from 0 (not at all) to 3 (very often). Items tapping inattentive and hyperactive-impulsive symptoms are presented in alternating fashion yielding both symptom totals and symptom severity scores in three different domains: Inattentive, Hyperactive-Impulsive, and Total. The scale has excellent internal consistency (alpha coefficient = .88-.92), test-

retest reliability ranging from .78-.86, and adequate criterion validity (DuPaul et al., 1998). Hyperactivity-Impulsivity severity scores range from 0-27 with higher scores indicating greater symptom severity. The home version of the scale was given to the mother and she rated the youth's symptoms of AD/HD; Hyperactivity-Impulsivity percentiles, based on age and gender, were used as a predictor of youth risk for depression.

#### *Predictors Variables - Protection*

*Modified Version of the Child Impairment Rating Scale (CIRS; Fabiano et al., 2006).* The original CIRS was developed to measure a child's need for treatment based on impairment in multiple contexts (i.e., social, parent-child, academic, self and family). Parents are asked to rate their child's level of impairment by placing an x on a continuum from "no problem" to "extreme problem". This measure was modified by including both a parent and child report version, with each rater indicating how well the child functions in the identified domains (Appendix A).. Parents and children placed a mark on a continuum from very poor to very excellent. The continuum was 100 mm in length and each mark was converted into 0-100 mm units.

#### *Procedure*

A majority of the sample was clinic-referred, with most (90%) of participating families drawn from the AD/HD Clinic at the University of North Carolina at Greensboro. In addition, one family was recruited from each of the following sites: Moses Cone's Developmental and Psychological Center in Greensboro, Sitrin Health Services an outpatient mental health care organization in New York, and a Greensboro

based community support group for families of children with AD/HD. Flyers were placed in these locations to increase awareness of the research project. Interested parties contacted the Principal Investigator (PI) by phone. Eligible families at the AD/HD Clinic were also verbally informed of the opportunity to partake in this research project. If families expressed an interest in participating, they were contacted directly by the PI and were given additional information about the study.

If the interested family had not received an assessment at the AD/HD Clinic at UNCG, the ADHD-RS (DuPaul et al., 1998) was administered to the mother over the phone to determine the potential presence of AD/HD in the identified youth. If the mother positively endorsed 6 or more out of 9 symptoms of either inattention or hyperactivity-impulsivity, then, if willing, the family (i.e., mother and youth) was scheduled to complete assessments at any of the identified clinics or the family's home, whichever was most convenient for the family.

If the family came to the AD/HD clinic, they were greeted by the clinic administrative assistant or by a graduate student. A graduate student then provided an overview of the research project and obtained consent from the mother and assent from the child. Next, the mother and child were directed into different assessment rooms. A graduate student then provided directions for completing the CDI, the modified version of the LEC, and a modified version of the CIRS-Youth Report. The graduate student then administered the measures to the youth or remained in the room to field any questions. After the youth completed their questionnaire, they were given an opportunity to ask questions about the research project.

During this time another graduate student, trained by a licensed psychologist in the administration of the C-DISC-IV, administered the C-DISC-IV AD/HD module to the mother. Next, the mother was given a packet of measures and directions on how to fill out the measures. The packet included the following measures in order: Child and Family Information sheet, ADHD-RS, BASC-2 PRS (either child or adolescent version), a modified version of the CIRS, BDI, and APQ. The mother then completed the packet at the visit or mailed the packet back to the researcher when completed.

Since many of the materials that were administered are also in the standard assessment battery at the AD/HD Clinic, families recruited from the clinic had slightly different administration procedures than families recruited from other agencies. If no more than 90 days had elapsed since the youth's clinic evaluation, mothers had the option to authorize the disclosure of measures (i.e., C-DISC-IV, BASC-2 PRS, ADHD-RS, and Child and Family Information sheet) that were completed during assessment at the AD/HD Clinic. All families granted authorization to disclose previously completed measures, thus for eligible families those measures were not re-administered.

At the conclusion of the study, mothers and youth were given more information about the study and received an individualized summary of results on selected measures. In addition, families received five dollars for participating.

### *Design*

The current study employed a cross-sectional, observational research design. The outcome of interest, depression symptomatology, was analyzed dimensionally. Considering the cross-sectional nature of the study, and the fluctuating nature of

depression, dimensional depression was considered to have greater explanatory power over categorical depression, and is consistent with the continuous nature of depression (e.g., Hankin & Abela, 2005).

## CHAPTER III

### RESULTS

#### *Data Reduction and Composite Variables*

*Composite Depression.* The best practice for measuring youth depression is using a combination of youth and parent report (Silverman & Saavadra, 2004). Therefore, a composite measure of depression was developed by combining the youth report CDI and mother report BASC-2 Depression subscale. T-scores were transformed to z-scores and then averaged together (Piacentini, Cohen, & Cohen, 1992). The BASC-2 Depression subscale was used in Composite Depression instead of the modified CDI Parent Version, so that mothers were not required to provide information about internal youth depression symptomatology (Piacentini et al., 1992).

*Familial Risk Composite.* Following the Failure Model (See Figure 1; Patterson & Capaldi, 1990), a Familial Risk Composite was created by aggregating parenting, maternal depression and youth aggression measures. Coercive interactions between parents and their children are an important feature of the Failure Model. Negative parenting practices such as inconsistent discipline often elicit disruptive behavior from youth and are a contextually salient risk factor for depression in AD/HD youth (Ostrander & Herman, 2006). Thus, a Negative Parenting Practices Composite was created by standardizing all APQ indices, using within sample statistics. Next, after multiplying positive parenting and involvement by -1, the average of all APQ indices was calculated.

The resulting Negative Parenting Practices Composite was created so that higher scores were indicative of maladaptive parenting practices. Antisocial child behavior also presents contextually salient risk for depression (Treuting & Hinshaw, 2001) and is associated with negative parenting practices and parental rejection (Patterson & Capaldi, 1990). Therefore antisocial child behavior was included in the composite and was operationalized using the BASC-2 PRS Aggression subscale T-scores based on age and gender. Lastly, given that a direct measure for parental rejection was not available, and the contextually salient risk for maternal depression in mothers of AD/HD youth, maternal depression was entered into the Familial Risk Composite. Maternal depression was operationalized as the total score on the maternal caregivers' BDI. Finally, the Negative Parenting Practices Composite, BASC-2 PRS Aggression subscale, and the BDI were  $z$ -standardized and the average of the three measures represented the Familial Risk Composite which served as a predictor of youth depression.

*Perceived Competence Composite.* In line with competency domains in the Failure Model (Patterson & Capaldi, 1990), a composite measure was created by averaging  $z$ -standardized scores from perceived social and academic competence items on the modified version of the CIRS-Youth report (see Appendix A).

#### *Preliminary Analyses*

To examine whether assumptions for regression analyses were met, the predictor and outcome variables were inspected. All variables were within the normal limits for skewness and kurtosis. Though visual inspections of variable distributions showed that

maternal BDI scores were bimodal in nature, the distribution of the Familial Risk Composite measure was normally distributed; therefore, maternal BDI scores remained in the calculation of the Familial Risk Composite. Statistical significance ( $\alpha$ ) was set a .05 for all analyses. Statistics presented in text are significant at the .05 level, unless otherwise noted.

#### *Demographic Variables and Predictor/Outcome Variables*

Consistent with prior research, younger children had significantly higher levels of hyperactivity ( $r = -.42$ ), and aggression ( $r = -.45$ ) but were less likely to be taking medication ( $r = .42$ ). Girls had significantly higher Familial Risk Composite scores ( $t = -2.38$ ) and mother-rated youth depression ( $t = -3.25$ ). Lower SES was associated with greater Familial Risk Composite scores ( $r = -.42$ ).

In the child domain, higher levels of inattention ( $r = .38$ ), hyperactivity-impulsivity ( $r = .39$ ), and aggression severity ( $r = .66$ ) were associated with increased levels of mother rated depression but not youth rated depression. Negative life events were not associated with any measure of youth depression.

At the family level, higher levels of maternal depression ( $r = .53$ ) were related to increases in mother-rated youth depression but not self-report of youth depression. No other parental indices were related to either measure of youth depression.

#### *Relationships within Composite Variables and Between Predictor and Outcome Variables*

A summary of correlations among predictor/outcome variables appears in Table 3.

*Depression.* The CDI, CDI-Parent Modification, and the BASC PRS Depression subscale were used to measure depression in this study. Both forms of maternal caregiver rated youth depression were positively correlated ( $r = .58$ ) and the relationship between the CDI and CDI-Parent Modification ( $r = .35, p < .10$ ) as well as the CDI and BASC Depression subscale ( $r = .32, p < .10$ ) were positively associated at a trend level.

To further explore the relationship between the two indices of the composite measure of depression (i.e., CDI and BASC-2 PRS Depression subscale), a test of homogeneity of variance showed that the CDI and BASC-2 Depression subscale had equivalent variances, thus a paired sample t-test was computed to examine differences in mean ratings. The results demonstrated that mother's rating of youth depression was significantly higher than youth self-report of depression ( $t = -3.56$ ). This suggests that though the variability of depression scores is similar across reporters, ratings of depression are higher for mothers compared to youth. Therefore, in addition to exploring research hypotheses with Composite Depression (i.e., CDI and BASC PRS Depression Subscale) as the primary outcome variable, independent analyses were also conducted using the CDI and the BASC PRS Depression subscale.

*Familial Risk Composite.* Though only inconsistent discipline was significantly related to depression ( $r = .42$ ), other parenting subscales (e.g., involvement) were related to other familial risk indices (e.g., maternal depression). Therefore, the Negative Parenting Practices composite, which included all five APQ subscales, was retained. Higher scores on the Negative Parenting Practices composite were associated with higher scores on mothers ( $r = .45$ ) and combined ( $r = .42$ ) measures of depression but not self-

report of youth depression. Among variables entered into the Familial Risk Composite, increases in maternal depression were related to increases in the Negative Parenting Practices composite ( $r = .39$ ). Aggression was not significantly related to either Negative Parenting Practices ( $r = .23$ ) or maternal depression ( $r = .05$ ). In contrast, increases in aggression ( $r = .66$ ), Negative Parenting Practices ( $r = .45$ ) and maternal depression ( $r = .53$ ) were all associated with increases in maternal rated youth depression. Although the conceptual relationships among familial risk indices put forth by the Failure Model (Patterson & Capaldi, 1990) were not fully supported, all indices increased risk for youth depression, which supports the creation of a composite measure of familial risk.

*Perceived Competence Composite.* Youth perceptions of competence in social and academic domains were significantly and positively related ( $r = .38$ ). Higher perceived competence with both peers ( $r = -.60$ ) and in academics ( $r = -.68$ ) were related to lower youth self-report of depression. In addition, increases in the Perceived Competence Composite was also related to decreases in youth self-report of depression ( $r = -.77$ ). These associations support the creation of the Perceived Competence Composite.

Given the statistical, conceptual and item content overlap between the Perceived Competence Composite and the CDI, the Perceived Competence Composite and its individual indices were correlated with the CDI total score after subtracting the raw scores on the Interpersonal problems and Ineffectiveness subscales. These subscales include questions about peers and school performance, respectively. The difference in magnitude of the correlations between the Perceived Competence Composite ( $t = -.18$ ;  $p > .05$ ), perceived social competence ( $t = .24$ ,  $p > .05$ ) and perceived academic

competence ( $t = 1.63, p > .05$ ) and the full and reduced CDI were not statistically different. Therefore, the relationship between youth perceived competence and self-report of depression was not due to overlap in item content (e.g., Hoza et al., 1993). Thus the results support the use of the CDI as a measure of self-report youth depression.

#### *Risk and Protective Factors Associated with Depressive Symptomatology*

A hierarchical regression analysis was utilized to test the primary hypotheses of the study. Due to the discrepancy in youth and mother rated youth depression in this sample, subsequent hierarchical regressions utilizing the same sequence of steps were also regressed onto self-report of youth depression and maternal caregiver rated youth depression independently. Determination of the hierarchical sequence of predictor variables was based on the Failure Model (e.g., Patterson & Capaldi, 1990). In the first step, Composite Depression was regressed onto demographic factors, followed by the Familial Risk Composite in the second step and youth Perceived Competence Composite in the third step. Finally, to examine the function of youth perceived competence across levels of risk, the interaction of the Familial Risk Composite and the youth Perceived Competence Composite was entered in the fourth step of the model.

As summarized in Table 4, this regression analysis yielded a final model explaining approximately 73% of youth depression variance from the aggregation of youth and mother report. In the first step, gender, age and SES were entered into the regression equation and explained a significant portion of Composite Depression variance. Female gender was associated with increased depression and higher levels of SES were associated with higher levels of depression at a trend level. Due to the wide

variability in SES and wide age range among participating youth, all variables were retained to simplify interpretation. In the second step, Familial Risk Composite was entered and accounted for additional Composite Depression variance. Next, the youth Perceived Competence Composite was entered and predicted additional Composite Depression variance. As expected, increases in perceived competence were associated with decreases in depression. Finally, the interaction between the Familial Risk Composite and the Perceived Competence Composite was entered into the model but against predictions failed to predict Composite Depression above previous predictors. Taken together, the results from this analysis suggest that the Failure Model adequately explains the relationship between AD/HD and depression in an AD/HD sample. Furthermore protective factors, specifically youth perceived competence, help to explain additional variance in youth depression above familial risk. In addition, when using a combined report of youth depression, results suggest that the protective effects of perceived competence function similarly across levels of risk.

Due to the discrepancy in youth and mother-rated youth depression; the previously specified hierarchical steps were regressed onto the CDI and the BASC-2 PRS Depression subscale, separately. When the model was regressed onto youth report of depression, with the exception of the youth Perceived Competence Composite, all steps were non-significant (see Table 5). In contrast, when mother rated youth depression was regressed onto the same hierarchical model (see Table 6), both gender and SES significantly predicted depression. In the next step, the Familial Risk Composite significantly predicted mother rated depression above demographic variables. Although,

the youth Perceived Competence Composite failed to predict mother rated youth depression, the interaction of the Familial Risk Composite and the Perceived Competence Composite predicted additional mother-rated youth depression variance at a trend level.

Simple slopes analyses (see Figure 2) revealed that familial risk for depression may moderate the function of youth perceived competence, such that perceived competence may protect youth from depression more at low levels of familial risk compared to high levels of familial risk. Thus, when depression is measured using only mothers' report, youth perceived competence may function as a "protective but reactive" factor (Luthar et al., 2000) in protecting youth with AD/HD from depression.

## CHAPTER IV

### DISCUSSION

Previous research on AD/HD and comorbid depression has focused on risk factors that may exacerbate risk for depression. Despite being at heightened risk for depression (Angold et al., 1999) many youth with AD/HD do not develop depression. Therefore, from a risk and protective perspective, this study examined whether the Failure Model (Patterson & Capaldi, 1990), could provide a useful framework for conceptualizing the complex comorbidity of AD/HD and depression. Consistent with predictions, results showed that the Failure Model, together with demographic variables, explained ~73% of depression symptom variance in stringently diagnosed youth with AD/HD. Thus, in addition to research demonstrating its utility with ODD/CD populations (Capaldi, 1991; Capaldi, 1992; Burke et al., 2005), the Failure Model appears to explain the presence of depression within an AD/HD youth population as well.

Consistent with the first hypothesis, the Familial Risk Composite predicted both composite and mother rated depression. However, the Familial Risk Composite only approached a trend level in predicting youth rated depression. Consistent with the second hypothesis, this study also found that after controlling for demographic variables and the Familial Risk Composite, youth perceived competence in social and academic domains was associated with lower depression severity on both the Composite Depression and youth report of depression. This supports the extension of the Failure Model to an

AD/HD population, and also suggests that AD/HD youth's perceptions of competence in developmentally relevant domains protect against depression.

In regards to the first hypothesis, the Familial Risk Composite emerged as a risk factor for youth depression. With the Failure Model (Patterson & Capaldi, 1990) as a conceptual framework, this suggests that negative parenting practices, maternal depression and youth aggression may increase risk for general psychopathology. Compared to specific risk/protective factors, general risk/protective factors provide a more parsimonious explanation for why youth with AD/HD tend to develop multiple comorbid disorders (Angold et al., 1999).

Increased rates of co-occurrence between internalizing and externalizing problems have been a focus of study across many perspectives. Within this study, the Familial Risk Composite represents multiple risk factors at different levels of the ecology. At a micro level of analysis, individual differences in temperament, such as difficulties with self-regulation, are also related to the development of both externalizing and internalizing problems (Calkins & Fox, 2002; Gilliom & Shaw, 2004; Oldehinkel, Hartman, De Winter, Veenstra, & Ormel, 2004). Temperament factors such as low effortful control and high negative emotionality, have been conceptually related to AD/HD, ODD/CD and depression (e.g., Nigg & Casey, 2005) and with all other factors in the Failure Model (e.g., Calkins & Fox, 2002; Feng, Shaw, Kovacs, Land, O'Rourke, & Alarcon, 2008; Graziano, Reavis, Keane, & Calkins, 2007). Thus, converging evidence, from multiple perspectives, may suggest that a heterotypic continuous pathway of externalizing problem behavior across development contributes to a congregation of general risk.

Though Angold et al., (1999) suggested that the relationship between AD/HD and depression is epiphenomenal, an alternative explanation is that the duration and severity of impairment in social and academic domains may interfere with the experiences of success or contribute to experiences of failure across development. Placing greater emphasis on competency in social and academic domains is more proximally related to depression (Luthar, 2006) and accounts for variability in adaptation in social and academic domains, across disruptive behavior disorders.

With respect to the second hypothesis, mother rated youth competence and discrepancy scores between mother and youth rated competence were associated with youth and combined report of youth depression. These findings are similar to other work that suggests that both familial risk (Ostrander & Herman, 2006) and youth social competency (Ostrander, Crystal & August, 2006) increase risk for depression in an AD/HD and general population (Cole, 1990).

In relation to the third hypothesis, generally, findings did not support the notion that perceived competence had greater protective benefits at high levels of familial risk. Instead when mothers-rated youth depression, youth perceived competence may have had greater protective benefits at low levels of familial risk. Specifically, youth perceived competence appears to function as a protective but reactive factor (Luthar et al., 2000). Though this finding only approaches significance, it suggests that youth perceived competence does not protect against a conglomeration of risk in youth with AD/HD. Thus, aiming to promote adaptation in social and academic domains, while neglecting to enhance family functioning, may have limited effectiveness.

Hoza et al. (1993) have called into question the nature of the relationship between AD/HD and depression, suggesting that the disorders are found to be comorbid due to overlap in AD/HD related impairment and depressive symptomatology. After removing scales from the CDI that contained questions about social and academic functioning, analysis revealed that magnitude of the relationship between the Perceived Competence Composite and the full CDI and the reduced version of the CDI did not differ. This indicates that the relationship between AD/HD and depression was not artificially inflated due to overlap in measurement content.

The findings from this study are promising but need to be considered in light of the limitations inherent in this research design. First, though the Failure Model has been supported across different populations, the cross-sectional nature of the study limits interpretation of causal relationships. In addition, all measures were based on youth or mother report. Utilizing observational instruments or using teacher reports for academic and social competence would increase measurement validity. Of particular note, mothers rated youth significantly higher than youth rated their own depressive symptomatology. Since youth with AD/HD are at five times greater risk to develop depression than their same aged peers, one explanation is that youth minimized their report of depression. Another limitation of the study is the reliability of the Perceived Competence Composite. Though the two items that created the Perceived Competence Composite had good face validity and construct validity, the small number of items contributing to the composite may make the competency ratings/interaction terms involving competency ratings less reliable. In addition, since the study lacks a “gold standard” measure of youth

competency (i.e., standardized achievement scores or sociometric status), it is difficult to understand the relationship between youth perceived competence, “true competence” and rater bias. Behavioral rather than cognitive adaptation in social and academic domains may also be a relevant protective factor and contextually salient in AD/HD, but was not directly assessed in this study.

The small sample size of the study limits the detection of small to moderate effects. For instance, significance tests of interaction effects aimed at exploring the function of protective factors are likely underpowered. To compensate for limited statistical power, the number of predictor variables was decreased by creating composite measures for Familial Risk and Perceived Competence. This in turn led to less specificity in interpreting results from regression analyses. Another limitation of the sample size is that it did not allow for separate age and gender analyses. Thus, differences between age groups or between males and females could not be explored. Due to the limited number of females with AD/HD, as well as youth with AD/HD-HI and AD/HD-PI subtypes, findings cannot be generalized to these populations. Lastly, to the extent that the importance of competency in family, social, and academic domains varies across individuals or cultures, the Failure Model may fail to explain the relationship between AD/HD and emotional functioning across all youth with AD/HD.

Bearing these limitations in mind, the results of this study nevertheless have implications for the assessment and treatment of youth diagnosed with AD/HD. In terms of assessment, the study demonstrates that both parent and child report of depression and associated risk and protective factors provide differing perspectives, which separately and

combined can provide clinically useful information. Since there are multiple causes for discrepancies between reporters, especially when youth self-report, it is important to probe for informant perspective and related attributions on the outcome of interest (see De Los Reyes & Kazdin, 2005).

Along with other research (e.g., Treuting & Hinshaw, 2002; Ostrander & Herman, 2006) these findings suggest that both perceptions of competence and attributions for success and failure that AD/HD youth make increase vulnerability to depression. Thus, when treating youth diagnosed with AD/HD, clinicians should aim to enhance youth strengths/competencies in social, academic or other domains. Additionally, if youth have focal deficits in social and academic domains it may be helpful broaden youth perceptions of competency and place greater emphasis on areas of relative success.

In terms of future research, though the Failure Model has helped to explain the relationship between AD/HD and depression, efforts to enhance functioning in familial, social and academic domains have led to small decreases in depressive symptomatology in AD/HD youth (Jensen, Hinshaw, Swanson, Greenhill, Conners, Arnold et al., 2001). This implies that current treatments that aim to reduce AD/HD related impairment do not optimally promote well-being across domains. To better enhance treatment for youth with externalizing problems, it is important to better understand the early developmental mechanisms of risk for depression. Such research should also look to model the Failure Model in an AD/HD population formally. Applying the Failure Model to AD/HD populations at earlier points in development (Gilliom & Shaw, 2004) may allow researchers to identify processes that are more malleable and are catalysts for future

competencies (Luther, 2006). Longitudinal research that looks to test cascading models (Masten, 2001) of externalizing behavior trajectories, depression and related competencies may also help to elucidate the nature of relationships among latent variables in the Failure Model. For instance, as adolescents sense of self becomes more differentiated (Abela & Hankin, 2005) perceptions of competence in social and academic domains may become extend to include romantic and occupational competency (Masten et al., 1995). With this developmental shift, emphasis in the Failure Model may move away from familial functioning and towards perceived competency in the multiple domains of functioning.

In addition, the majority of research conducted on the Failure Model has been conducted predominantly male samples. Little is known about how well the Failure Model explains the relationship between girls with AD/HD and depression. Future research should examine this gender issue. Moreover, experimental designs may help to explicate specific mechanisms that underlie the statistical association among latent variables in the Failure Model. Such research may better inform understanding of the comorbidity between AD/HD and depression and inform the development of new treatments that aim to promote emotional well-being in the AD/HD population.

In conclusion, the current study examined risk and protective factors associated with depressive symptomatology in AD/HD youth. Consistent with the Failure Model (Patterson & Capaldi, 1990), both familial risk for depression and youth perceived competence predicted a large proportion of depression related variance in an AD/HD population. From a risk and protective perspective, the results from this study suggest

that though perceived competence may protect youth from depression, it may do so more at low levels of familial risk. These findings suggest that the Failure Model has utility in explaining the presence of depressive symptomatology in AD/HD youth and provide an important link to previous research (e.g., Gilliom & Shaw, 2004; Capaldi, 1990; Capaldi, 1991; Capaldi & Stoolmiller, 1999).

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APPENDIX A. TABLES AND FIGURES

Table 1. *Sample Characteristics*

	<u>M</u>	<u>SD</u>
Age in years	10.8	2.4
Socioeconomic Status	46.6	13.9
	<u>Percentage</u>	<u>N</u>
Gender		
Male	77%	23
Female	23%	7
Race		
Caucasian	80%	24
African-American	20%	6

*Note.* N=30

Table 2. Diagnostic and Treatment Sample Characteristics

	<u>Percentage</u>	<u>N</u>
AD/HD Subtype		
Combined Type	80%	24
Inattentive Type	17%	5
Hyperactive-Impulsive Type	3%	1
Depression		
Perceived History of Depression <sup>a</sup>	32%	8
Documented History of Depression <sup>a</sup>	8%	2
Treatment		
On Medication	70%	21
Received Psychosocial Treatment <sup>a</sup>	63%	15
Received Parent Training <sup>a</sup>	46%	11
	<u>M</u>	<u>SD</u>
ADHD Symptom Severity		
ADHD RS Inattention Severity (%)	93.39	8.10
ADHD RS HI Severity (%)	92.75	11.47
BASC-2PRS Attention Problems	72.40	14.44
BASC-2 PRS Hyperactivity	67.83	7.38
Depressive Symptom Severity		
CDI T-score	49.40	10.31
BASC-2 PRS Depression T-Score	58.50	13.25

*Note.* ADHD RS = ADHD Rating Scale; HI = Hyperactivity/Impulsivity; BASC-2 PRS = Behavior Assessment Schedule for Children – 2<sup>nd</sup> Edition Parent Rating Scale; CDI = Child Depression Inventory; <sup>a</sup>=Information was collected from 25 out of 30 families.

Table 3. *Correlations among Predictor Indices/Variables and Outcome Variables*

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Gender	-													
2. Age	.03	-												
3. Socioeconomic Status	-.01	.25	-											
4. Familial Risk Composite	.41*	-.34 <sup>†</sup>	-.42*	-										
5. APQ: Composite	.07	-.15	-.39*	.63*	-									
6. BDI: Total Score	.31 <sup>†</sup>	.00	-.40	.64*	.39*	-								
7. BASC-2: Aggression	.36 <sup>†</sup>	-.45*	-.17	.76*	.23	.05	-							
8. Perceived Competence	-.11	.02	.28	-.37*	-.37*	-.19	-.25	-						
9. Youth CIRS: Social	-.05	.28	.40*	-4.6	-.47*	-.16	-.37*	.81*	-					
10. Youth CIRS: Academic	-.12	-.23	.09	-.17	-.17	-.16	-.06	.85*	.38*	-				
11. FRC X PC	-.10	.07	.02	-.37*	-.33 <sup>†</sup>	-.18	-.29	.75*	.69*	.56*	-			
12. CDI: Total Score	.30	.02	-.06	.37*	.20	.19	.33 <sup>†</sup>	-.77*	-.60*	-.68*	-.65*	-		
13. BASC-2: Depression	.52*	-.21	-.39*	.83*	.45*	.53*	.66*	-.27	-.31 <sup>†</sup>	-.15	-.14	.32 <sup>†</sup>	-	
14. Composite Depression	.52*	-.13	-.30	.77*	.42*	.47*	.63*	-.61*	-.54*	-.47*	-.45*	.76*	.86*	-

*Note.* APQ = Alabama Parenting Questionnaire; BDI = Beck Depression Inventory; BASC-2 Behavior Assessment Schedule for Children - 2<sup>nd</sup> Edition Parent Rating Scale; CIRS = Modified Version of Child Impairment Rating Scale – Youth Version; FRC = Familial Risk Composite; PC = Perceived Competence Composite; CDI = Child Depression Inventory. <sup>†</sup>p < .10. \*p < .05.

Table 4. *Summary of Hierarchical Regressions for the Failure Model Predicting Composite Youth Depression.*

Step	Variable	Std. $\beta$	Cum. Adj. $R^2$	F $\Delta$	Sig. F $\Delta$
1			.29	5.00*	.007
	Gender	.52*			
	Age	-.08			
	SES	-.28 <sup>†</sup>			
2			.60	20.83*	.000
	Gender	.23 <sup>†</sup>			
	Age	.11			
	SES	-.03			
	Familial Risk Composite	.70*			
3			.73	13.02*	.001
	Gender	.25*			
	Age	.05			
	SES	.03			
	Familial Risk Composite	.56*			
	Perceived Competence Composite	-.38*			
4			.73	1.44	.243
	Gender	.24*			
	Age	.04			
	SES	.09			
	Familial Risk Composite	.60*			
	Perceived Competence Composite	-.53*			
	Familial Risk Composite X Perceived Competence Composite	.19			

Note.  $N = 30$ . SES=Socioeconomic Status

<sup>†</sup> $p < .10$ . \* $p < .05$ .

Table 5. Summary of Hierarchical Regressions for the Failure Model Predicting Youth Self-Report of Depression.

Step	Variable	Std. $\beta$	Cum. Adj. $R^2$	F $\Delta$	Sig. F $\Delta$
1			-.01	.88	.464
	Gender	.30*			
	Age	.03			
	SES	-.06			
2			.05	2.81	.106
	Gender	.13 <sup>†</sup>			
	Age	.14			
	SES	.08			
	Familial Risk Composite	.40			
3			.61	36.84*	.000
	Gender	.17			
	Age	.02			
	SES	.20			
	Familial Risk Composite	.11			
	Perceived Competence Composite	-.77*			
4			.60	.09	.763
	Gender	.18			
	Age	.02			
	SES	.18			
	Familial Risk Composite	.09			
	Perceived Competence Composite	-.73*			
	Familial Risk Composite X Perceived Competence Composite	-.06			

Note.  $N = 30$ . SES = Socioeconomic Status

<sup>†</sup> $p < .10$ . \* $p < .05$ .

Table 6. *Summary of Hierarchical Regressions for the Failure Model Predicting Mother-Rated Youth Depression*

Step	Variable	Std. $\beta$	Cum. Adj. $R^2$	F $\Delta$	Sig. F $\Delta$
1			.38	6.82*	.002
	Gender	.52*			
	Age	-.14			
	SES	-.35*			
2			.69	27.55*	.000
	Gender	.23 <sup>†</sup>			
	Age	.05			
	SES	-.10			
	Familial Risk Composite	.71*			
3			.68	.16	.693
	Gender	.23 <sup>†</sup>			
	Age	.06			
	SES	-.11			
	Familial Risk Composite	.72			
	Perceived Competence Composite	.05			
4			.71	3.83 <sup>†</sup>	.062
	Gender	.20			
	Age	.04			
	SES	-.01			
	Familial Risk Composite	.80*			
	Perceived Competence Composite	-.20			
	Familial Risk Composite X Perceived Competence Composite	.32 <sup>†</sup>			

Note.  $N = 30$ . SES = Socioeconomic Status

<sup>†</sup> $p < .10$ . \* $p < .05$ .

Figure 1. The Failure Model with latent variables from Patterson and Capaldi (1990). Related variables and composites within this study are presented in italics and bold text, respectively.

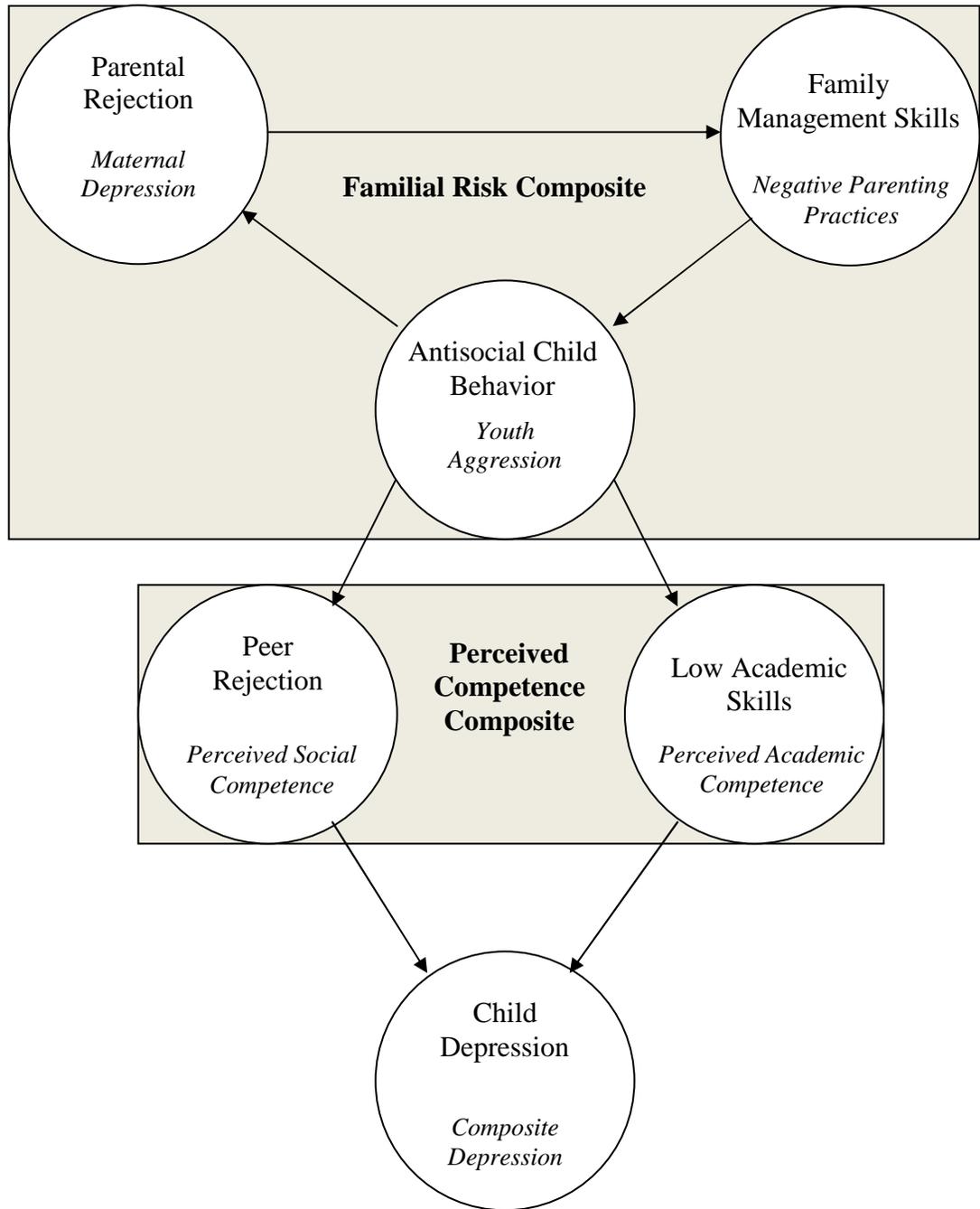
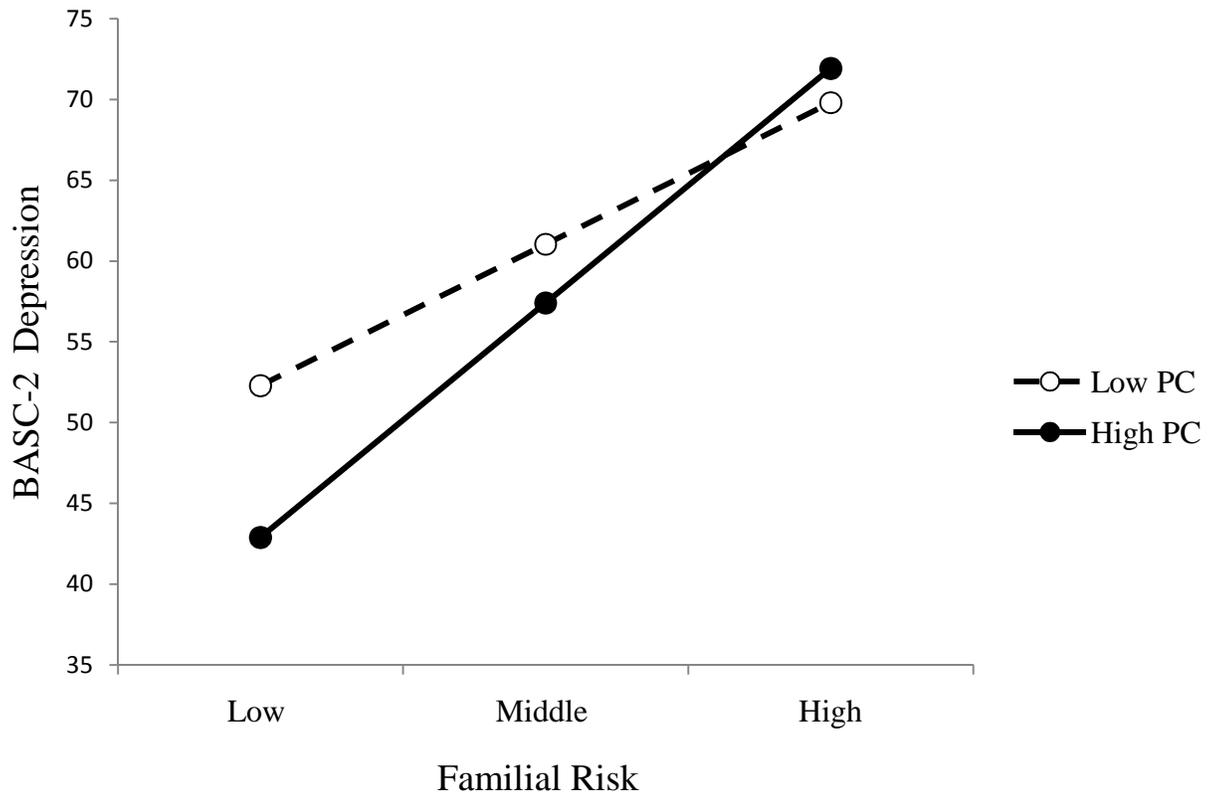


Figure 2. Simple slopes analysis for maternal-rated youth depression on familial risk at levels of youth perceived competence.



Note. PC = Perceived Competence Composite

APPENDIX B. MEASURES

Modified CIRS (Parent Version)

Instructions: Please mark an "X" **anywhere** on the lines TO INDICATE how well your child functions in each area.

1. How well does your child get along with his/her peers?

|\_\_\_\_\_|

**Very poorly**

**Very well**

2. How well does your child get along with you (and your spouse if present)?

|\_\_\_\_\_|

**Very poorly**

**Very well**

3. How is your child's academic progress at school?

|\_\_\_\_\_|

**Very behind**

**Very advanced**

4. How is your child feel about himself/herself?

|\_\_\_\_\_|

**Very low self-esteem**

**Very high self-esteem**

5. What kind of influence does your child have on your family

|\_\_\_\_\_|

**Very negative**

**Very positive**

6. Overall, how well does your child function in daily life?

|\_\_\_\_\_|

**Functions poorly in many areas**

**Very successful in all areas**

Modified CIRS (Youth Version)

Instructions: Please mark an "X" anywhere on the lines TO INDICATE how well you function in each area.

1. How well do you get along with your peers?

|\_\_\_\_\_|

**Very poorly**

**Very well**

2. How well do you get along with your mother (and your father if present)?

|\_\_\_\_\_|

**Very poorly**

**Very well**

3. How is your academic progress at school?

|\_\_\_\_\_|

**Very behind**

**Very advanced**

4. How do you feel about yourself?

|\_\_\_\_\_|

**Very low self-esteem**

**Very high self-esteem**

5. What kind of influence do you have on your family?

|\_\_\_\_\_|

**Very negative**

**Very positive**

6. Overall, how well does your do you function in daily life?

|\_\_\_\_\_|

**Function poorly in many areas**

**Very successful in all areas**