MCGEE, ELIZABETH HOLT, Ph.D. Factors Associated with the Development of Anxiety Among Children with Attention-Deficit/Hyperactivity Disorder. (2007) Directed by Dr. Arthur D. Anastopoulos. 103 pp.

Given that children with Attention-Deficit/Hyperactivity Disorder (AD/HD) are known to experience difficulties with anxiety at a higher rate than the general population, this study's overall goal was to examine potential risk factors that may explain the link between AD/HD and comorbid anxiety. Sixty-three male and female children diagnosed with AD/HD and their mothers were administered various questionnaires to assess six domains (demographics, faulty thinking, parenting factors, school functioning, treatment, and AD/HD severity) to ascertain possible risk factors for the development of comorbid anxiety. In addition, mothers were administered a diagnostic interview to assess their children's AD/HD and separate anxiety diagnoses. Results supported variables most consistently in the faulty thinking, parenting factors, and school functioning domains as potential risk factors contributing to comorbid anxiety in children with AD/HD. Contrary to expectations, AD/HD severity made no direct contribution to comorbid anxiety above and beyond the other variables. However, it most likely makes an indirect contribution by its impact on the faulty thinking, parenting factors, and school functioning domains. Further, although causality cannot be addressed by the current research design, the three identified domains serve as a foundation for future research targeting directionality and risk in the development of anxiety among children with AD/HD. Based on this study's findings, implications for assessments and treatments for clinical practice were also discussed.

FACTORS ASSOCIATED WITH THE DEVELOPMENT OF ANXIETY AMONG CHILDREN WITH ATTENTION-DEFICIT/ HYPERACTIVITY DISORDER

by

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

Greensboro
2007

Approved by

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To my family

APPROVAL PAGE

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

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ACKNOWLEDGMENTS

I would like to thank those people who provided support and encouragement throughout the course of this research project. Specifically, I appreciate the guidance and patience of my adviser and dissertation committee chair, Dr. Arthur Anastopoulos, and would also like to thank Dr. Wesley Allan, Dr. Heidi Gazelle, and Dr. Walter Salinger for serving on my dissertation committee. I would also like to express my gratitude to Shelly Ball and Dr. Kelly Graves for statistical assistance and to those students who helped with data collection, John Palembas, Lucas Thomas, and John David Odom. Finally, I would like to thank my family and friends for all their encouragement throughout this process.

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CHAPTER I

INTRODUCTION

One of the most common difficulties for which children are referred for psychological evaluations and treatment is Attention-Deficit/Hyperactivity Disorder (AD/HD; American Psychiatric Association, 1994; Cantwell, 1996; National Institutes of Health, 2000). Approximately 3% to 5% of school-aged children develop this disorder. Many of these children display or go on to develop secondary or comorbid conditions including learning difficulties, social skills deficits, externalizing problem behaviors, and internalizing difficulties (Barkley, 1998; Barkley, Anastopoulos, Guevremont, & Fletcher, 1992; Befera & Barkley, 1985; Cantwell & Baker, 1992; Cuffe, Moore, & McKeown, 2005; DuPaul & Stoner, 1994; Pelham & Bender, 1982). Although a significant percentage of children with AD/HD exhibit problems beyond those due to the core symptoms of the disorder, little is known about the development of comorbid conditions. Compared to research on comorbid learning difficulties and externalizing problems among children with AD/HD, even less is known regarding the development of comorbid internalizing problems, specifically anxiety difficulties (Jensen et al., 2001).

What is known is that anxiety disorders are present among children with AD/HD at a higher rate than in the general population (Angold, Costello, & Erkanli, 1999). Studies have indicated that anywhere from 25% to 50% of children with AD/HD display some type of anxiety disorder (Anderson, Willliams, McGee, & Silva, 1987; August,

Realmuto, MacDonald, Nugent, & Crosby, 1996; Biederman, Faraone, & Lapey, 1992; Bird, Gould, & Staghezza, 1993; Busch et al., 2002; Cohen, Cohen & Kasen, 1993; Jensen, Martin, & Cantwell, 1997; Russo & Biedel, 1994) compared to 5% to 21% of children without AD/HD in the general population (Busch et al., 2002; Kashani & Orvaschel, 1990; Pliszka, 2000). Although many children with AD/HD exhibit comorbid anxiety, many do not develop this type of problem. What is unknown is how anxiety develops in children with AD/HD. In the AD/HD population, multiple pathways are likely to exist to bring about comorbid anxiety, and the timing of these pathways is unclear. Core anxiety symptoms may precede the development of AD/HD symptoms, the opposite may occur, or perhaps the presence of both difficulties occurs simultaneously.

To facilitate understanding of how comorbid anxiety may develop in children with AD/HD, a review of each of these disorders will be presented first, including sections on current diagnostic criteria, epidemiology, developmental course, and etiology. Next, an examination of the research on AD/HD and comorbid anxiety and its limitations will be highlighted to aid in determining gaps that current and future research may target. Finally, a conceptual framework of potential risk factors associated with the presence of anxiety in children with AD/HD will be presented as a needed next step in this line of research.

Overview of AD/HD

AD/HD is a disorder characterized by developmentally deviant levels of inattention and/or hyperactivity-impulsivity (American Psychiatric Association, 1994). Inattentive symptoms include behaviors such as having difficulty sustaining attention,

making careless errors in one's work, and being easily distracted. Hyperactive-impulsive symptoms include behaviors such as fidgeting and squirming when seated, running around or climbing excessively in inappropriate situations, being "on the go" or acting as if "driven by a motor," and having difficulty waiting for a turn. Not only must a diagnosis be based upon the presence of these symptoms, but a child must also display a pervasive and chronic pattern of inattentive and/or hyperactive-impulsive behaviors in excess of what would be expected of other children of the same age (American Psychiatric Association, 1994).

Current diagnostic criteria

In the current Diagnostic and Statistical Manual of Mental Disorders - Fourth Edition (DSM-IV), two lists of symptoms are provided, one for inattentive behaviors and one for hyperactive-impulsive behaviors. Six of nine symptoms from the inattention list and/or six of nine symptoms from the hyperactivity-impulsivity list must be endorsed as developmentally deviant in order to meet the symptom frequency criteria for a diagnosis of AD/HD. In addition, the symptoms must have persisted for at least 6 months, be present prior to age seven, and be associated with clinically significant impairment in two or more settings (e.g., home, school, daycare). Finally, these symptoms cannot be attributable to other mental or behavioral disorders. One of three major types of AD/HD diagnoses can be determined from criteria met for either or both symptoms lists: Predominantly Inattentive Type (AD/HD-I), Predominantly Hyperactive-Impulsive Type (AD/HD-HI), and Combined Type (AD/HD-C; American Psychiatric Association, 1994). AD/HD-I is diagnosed when at least six of nine inattentive symptoms and five or less

hyperactive-impulsive symptoms are endorsed. AD/HD-HI is diagnosed when at least six of nine hyperactive-impulsive symptoms and five or less inattentive symptoms are exhibited. Finally, AD/HD-C can occur when at least six of nine inattentive symptoms and at least six of nine hyperactive-impulsive symptoms are present.

Epidemiology

The prevalence of AD/HD in school-aged children is estimated at 3-5% of the general population (American Psychiatric Association, 1994; Cuffe et al., 2005). However, studies examining AD/HD have reported prevalence rates ranging from a low of 2% to as high as 25% (Gadow et al., 2000; Gaub & Carlson, 1997; Nolan, Gadow, & Sprafkin, 2001; Szatmari, 1992). The prevalence of AD/HD has been found to vary significantly as a function of gender, with the disorder being evident much more frequently in males than in females. Male-to-female ratios range from 4:1 to 9:1 depending on the sample (i.e., general population or clinic population). Prevalence rates are also affected by age considerations with rates of the disorder appearing to decrease with age (DuPaul et al., 1997). The role of socioeconomic status (SES) on prevalence rates of AD/HD is inconsistent, with some research reporting slightly higher rates in lower SES groups while others show comparable rates across social classes when comorbid conditions are controlled (Lambert, Sandoval, & Sassone, 1978; Szatmari, 1992). Ethnic differences have also been documented suggesting a higher rate of AD/HD symptoms among ethnic minority children when compared to Caucasian children (Bauermeister, Berrios, Jimenez, Acevedo, & Gordon, 1990; DuPaul et al., 1997).

Developmental Course

The expression of AD/HD symptoms changes across development. The onset of these symptoms usually occurs during the preschool years at about 3 to 4 years old (Barkley, 1996). Hyperactive-impulsive behaviors typically characterize the disorder at this age followed by developmentally deviant inattentive behaviors between 5 and 7 years of age. Hyperactive-impulsive symptoms have been found to steadily decrease over time, while inattentive symptoms remain relatively constant (DuPaul et al., 1998; DuPaul et al., 1997). As children diagnosed with AD/HD develop into adolescents, about 50% to 80% will continue to experience difficulties and meet full diagnostic criteria for the disorder (Barkley, Fischer, Edelbrock, & Smallish, 1990; Mannuzza & Klein, 1992). An estimated 30% to 50% of these adolescents with AD/HD will continue to display symptoms into adulthood (Barkley, 1996). Even though as adults, individuals often do not continue to meet criteria for a formal AD/HD diagnosis, they still carry residual symptoms.

Etiology

Because most research points to the etiology of AD/HD as biological in nature, these factors receive much more attention than purely psychosocial causes. Psychosocial factors are generally not thought to play a major etiological role (Barkley, 1990; Cantwell, 1996). The main biochemical theory of AD/HD has been based on a catecholamine hypothesis, but its impact on AD/HD still remains unclear (Pliszka, McCracken, & Maas, 1996). Studies on the place of action of methylphenidate point to dopamine being the most influential neurotransmitter involved in the presence of AD/HD

symptoms (Swanson, Castellanos, Murias, LaHoste, & Kennedy, 1998). Rogeness, Javors, and Pliszka (1992) hypothesize that AD/HD can be seen as a result of dysregulation or imbalance of three neurotransmitter systems: the dopaminergic system, the noradrenergic system, and the serotonergic system. It is unclear, however, the exact imbalance that is necessary for the development of AD/HD (Quist & Kennedy, 2001).

Studies are increasingly reporting possible genetic linkages for AD/HD. To date, the strongest evidence for heritability of AD/HD comes from twin studies with greater concordance for AD/HD symptoms occurring between monozygotic (MZ) twins compared to dizygotic (DZ) twins. In a review of 20 national and international twin studies of AD/HD, Faraone et al. (2005) found a mean heritability estimate of 76%. The estimated heritability of AD/HD ranges from 0.5 to 0.9. However, it has been proposed that the higher estimates are found in studies using symptom counts rather than categorical diagnoses. When children who meet clinically significant diagnostic criteria for AD/HD were used in a study assessing heritability, the results revealed a heritability of 64 % for hyperactivity and inattention. This finding may indicate genetics may be contributing more to the development of AD/HD in children who exhibit clinically significant levels of AD/HD symptoms (Todd, 2000).

Results from family studies and adoption studies provide further evidence that is consistent with genetics being a major contributor to the development of AD/HD. For example, there is a higher prevalence of psychopathology in the parents, siblings, and other family members of children with AD/HD. More specifically, it has been found that between 10% and 35% of parents and siblings of children with AD/HD also have the

disorder (Biederman, Faraone, Keenan, & Tsuang, 1991; Faraone & Biederman, 2000; Pauls, 1991). Research from adoption studies also demonstrates that adopted children were more likely to show levels of hyperactivity that resembled the behavior of their biological parents rather than their adoptive parents (Sprich, Biederman, Crawford, Mundy, & Faraone, 2000; Todd, 2000).

More recently, researchers have begun to investigate more extensively the genes involved in AD/HD. Molecular genetic studies suggest that AD/HD may be very complex and involve the contribution of several genes. Mixed support has been found in candidate gene studies of AD/HD which have focused on catecholaminergic, noradrenergic, and serotonergic genes. Faraone et al. (2005) reviewed candidate gene studies of AD/HD based on case-control and family-based designs and found substantial support for the following gene variants mostly associated with the dopamine and serotonin systems: DRD4, DRD5, DAT, DBH, 5-HTT, HTR1B, and SNAP-25. Whether AD/HD is transmitted by one or more related or independent genes is still unknown, although there is mounting evidence that AD/HD is likely caused by many genes of small effect (Faraone et al. 2005; Khan & Faraone, 2006; Waldman & Gizer, 2006).

Overview of Childhood Anxiety

Compared to disruptive behavior disorders, less research has focused on childhood anxiety (Chorpita & Barlow, 1998; Pine, 1999). However, in recent years researchers have begun to study more extensively the epidemiology and course of childhood anxiety disorders. Children with clinical levels of anxiety are characterized by

experiencing developmentally inappropriate fear or worry that is out of proportion to the context of the situation causing impairment in functioning. The DSM-IV currently defines 10 clinical anxiety disorders that can affect children: separation anxiety disorder, specific phobia, generalized anxiety disorder, social phobia, panic disorder with and without agoraphobia, agoraphobia without panic disorder, obsessive-compulsive disorder, posttraumatic stress disorder, and acute stress disorder (American Psychiatric Association, 1994).

Current diagnostic criteria

Due to the large number of childhood anxiety disorders, only the two of most interest to this study and which have been found to be commonly comorbid with AD/HD - Generalized Anxiety Disorder (GAD) and Social Phobia - will be described here (Manassis, Tannock, & Barbosa, 2000; Pine, 1997; Willcutt, Pennington, Chhabildas, Friedman, & Alexander, 1999). According to criteria in the DSM-IV, in Generalized Anxiety Disorder, the anxiety symptoms are not bound to any discernible stimulus situation. Excessive worry pervades a wide range of events and activities. This excessive worry and anxiety are characteristic of the child's life for at least 6 months and are evidenced by one of six symptoms, i.e., restlessness, fatigue, sleep problems, inability to concentrate. In Social Phobia, the anxiety is marked by a fear or worry of social or performance situations in which the child may feel embarrassed. Children may or may not recognize that their worry is unreasonable. These feelings of worry must have existed for 6 months in order to receive a diagnosis (American Psychiatric Association, 1994).

Epidemiology

The prevalence rate of anxiety is somewhat more complicated to estimate than that of AD/HD due to the number of separate disorders. Population studies generally find around 5% to 21% of children reporting symptoms consistent with anxiety disorders (Kashani & Orvaschel, 1990; Pliszka, 2000). As reviewed in Pine (1997), among the 10 DSM-IV childhood anxiety disorders, Separation Anxiety Disorder, Specific Phobia, and Generalized Anxiety Disorder are the most common with prevalence estimates in the 2% to 5% range. Panic Disorder is the rarest of the anxiety disorders occurring in <1% of children. In general, anxiety is experienced at equal rates between male and female children during childhood with a split emerging at some point during adolescence with girls experiencing more difficulties with anxiety than boys. This pattern varies somewhat across studies and anxiety disorders. In respect to the two primary anxiety disorders of interest in this study, GAD appears to follow the above pattern (see reviews, Costello, Egger, & Angold, 2004; Flannery-Schroeder, 2004) whereas research is mixed in the case of Social Phobia, with some supporting that equal rates appear to be maintained (Beidel, Turner, & Morris, 1999) and other research supporting a split in adolescence (Essau, Conradt, & Petermann, 1999).

Developmental Course

In a review of age of onset of childhood anxiety disorders in community and clinic samples, studies indicated that the onset age of anxiety disorders precedes that of depressive disorders in childhood (Kovacs & Devlin, 1998). Onset age was defined as the earliest age at which the participants experienced any core symptoms of a particular

anxiety disorder. The mean onset age of GAD has been reported as 8.8 years and the mean onset age of Social Phobia was 11 years (Last, Perrin, Hersen, & Kazdin, 1992). Other studies have found the mean age of onset of GAD ranging from 10.8 to 13.4 years and the mean onset of Social Phobia ranging from 11.3 to 12.7 years (see review Beidel & Turner, 2005).

Views on childhood anxiety have gradually undergone change. Whereas childhood worries and fears were historically thought to be transient in nature and were a neglected area of research, anxiety problems are now recognized as being a more stable and prevalent childhood difficulty. Last, Perrin, Hersen, and Kazdin (1996) found that 81.7% of clinically referred children 5-18 years old with anxiety disorders had recovered from their initial anxiety diagnosis at the end of a 3-4 year follow-up period with few cases (7.8%) of relapse during this period. However, approximately 30% of these children developed new psychological diagnoses, usually a different anxiety disorder but a few developed behavior disorders or depressive disorders indicating continued clinical problems. In a review by Beidel and Turner (2005), anxiety disorders across childhood and adolescence are present for considerable amounts of time. Although rates vary, approximately 50% of children were found to meet criteria for their initial anxiety diagnosis at follow-up which ranged from 6 months to 5 years. Twenty-five to 30% of the children were diagnosed with different anxiety disorders at follow-up which were hypothesized to have developed due to possible changes in the form of the disorder, child development, or diagnostic procedures. Examining the developmental changes of childhood anxiety symptoms and fears in a community sample emphasized the need to

keep in mind developmental changes when studying the developmental course of clinical anxiety (Weems & Costa, 2005). Separation anxiety was found to be most prominent in children 6-9 years old, death and danger fears in children 10-13 years old, and social anxiety in addition to fear of criticism in adolescents 14-17 years old. Although the pattern of anxiety in a clinical sample may not follow the same pattern as in a community sample, it is important to consider that developmental changes may change the clinical expression as well.

It is generally accepted that in many cases there may be connections from anxiety in childhood and adolescence to adult anxiety disorders. For example, findings suggest that approximately 45% of adolescents with anxiety disorders will have anxiety disorders in adulthood (Pine, Cohen, Gurley, Brook, & Ma, 1998). In addition, in studies of adults with anxiety disorders, most retrospectively reported suffering from an anxiety disorder in childhood (Newman et al., 1996; Pine et al., 1998). Prospective longitudinal studies are needed to determine the specific risk that childhood anxiety disorders may contribute to the pathway in developing adult anxiety disorders.

Etiology

It is generally recognized that children develop and maintain problems with anxiety through multiple pathways (i.e., equifinality; Cicchetti & Rogosch, 1996). Both a biological predisposition as well as interactions among environmental factors are thought to contribute to anxiety disorders in children, and pathways may differ among children leading to the same outcome (anxiety disorder). Children who may be at-risk for anxiety problems also do not react equally to the same environmental and psychosocial factors,

and thus, all at-risk children do not develop anxiety disorders (i.e., multifinality; Cicchetti & Rogosch, 1996; Morris, 2004). Although multiple factors play a role in the development of anxiety in children, including both biological explanations as well as environmental explanations, the focus of this next section will be biological in nature with a more detailed examination of prominent psychosocial factors that contribute to the development of anxiety to follow in subsequent sections.

Biological factors. Biological explanations have been a focus in studying the development of childhood anxiety as anxiety disorders have been shown to aggregate in families (Merikangas, Avenevoli, Dierker, & Grillon, 1999). Children of adults with anxiety disorders display increased rates of anxiety disorders (Fyer, Mannuzza, Chapman, Martin, & Klein, 1995; Last, Hersen, Kazdin, Orvaschel, & Perrin, 1991; Warner, Mufson, & Weissman, 1995). From another perspective, first-degree relatives of children with anxiety disorders also exhibit increased rates of anxiety disorders (Fyer et al., 1995; Last et al., 1991). More recently, studies have focused on examining the specificity of familial associations across the DSM-IV anxiety classifications (Pine, 1997). However, current theories and the majority of studies have revealed that there is a genetic predisposition to developing anxiety of various degrees rather than a specific disorder (Pine, 1999). Twin studies of anxiety are generally consistent with the trend in research on childhood disorders that indicate genetic and nonshared environmental factors contribute to their development (Pine, 1997). In a study of 6-year-old twins, Bolton et al. (2006) found that Separation Anxiety Disorder demonstrated a substantial heritability estimate of 73%, while Specific Phobias were 60%, suggesting that genetic

effects may be more significant than environmental effects on early-onset anxiety disorders. However, in general, child studies of anxiety have produced mixed results with some indicating substantial heritability estimates, while others indicating that familial transmission of anxiety was best explained by shared environment (Thapar & McGuffin, 1994; Topolski et al., 1997). Legrand, McGue, and Iacono (1999) estimated trait anxiety heritability at 45% and that more transient anxiety was accounted for by environmental factors. Other research has found that genetics contributes approximately 30% to anxiety heritability with shared environment 20% and the remaining variance in childhood anxiety explained by nonshared environmental factors (see review Eley & Gregory, 2004). Although there have been several studies exploring genes related to the serotonin and dopamine systems in attempts to identify genes underlying anxiety disorders, no specific genetic loci have been found and replicated in independent samples (Merikangas & Low, 2005).

The genetic component of anxiety is often thought to be manifested early on as a type of temperament in young children. The term temperament indicates an assortment of stable moods and behavioral patterns that are controlled or influenced by a person's biology. Emerging in early child development, temperament is a construct made up of both physiological and psychological processes. Temperament is the result of various environments acting on young children who inherit a particular physiology (Schwartz, Snidman, & Kagan, 1999). When applied to childhood anxiety specifically, Kagan (1994) describes two categories of children - behaviorally inhibited and uninhibited. Inhibited (shy) children at 2 years of age typically stop their ongoing behavior, stop

talking, seek comfort from a parent or withdraw when faced with unfamiliar people or situations. In contrast, uninhibited (sociable) children approach unfamiliar people and situations and are outgoing, sociable, and talkative.

Research examining Kagan's inhibited temperament in early childhood as a precursor to later anxiety problems has produced some support. Schwartz et al. (1999) found that there are certain components of an inhibited temperament that continue from 2 years of age to early adolescence that predispose them to social anxiety. Among adolescents who had been classified as inhibited or uninhibited at 2-years-old with both standardized interviews and observation, 34% were found to be impaired by generalized social anxiety compared to 9% who had been identified as uninhibited (Schwartz et al., 1999). These findings indicate that inhibition at an early age may be a risk factor for developing later social anxiety. However, 66% of the original inhibited 2-year-olds did not develop impairing social anxiety, suggesting that environment and other psychosocial factors play an influential role in the development of anxiety.

Prior, Smart, Sanson, and Oberklaid (2000) also conducted a longitudinal study to assess relationships between inhibited children and anxiety problems later in adolescence. As part of the Australian Temperament Project (ATP), children were identified as inhibited "shy" and uninhibited "not shy" at 4-8 months of age and were assessed during several subsequent waves of data collection. Results showed that 42% of children rated as shy during 6 or more of 8 assessments in childhood developed anxiety problems in adolescence compared to 11% of the participants who were never identified as shy. However, only 20% of the children with anxiety problems in early adolescence were

rated as persistently shy in childhood, and only 9% of a subsample of the cohort who had been given an anxiety diagnosis had been rated as persistently shy in childhood. Results again suggest other environmental factors most likely play a role in anxiety development.

Shamir-Essakow, Ungerer, and Rapee (2005) examined the relationships among behavioral inhibition, attachment, and anxiety in 3-4 year old children to aid in clarifying the development of childhood anxiety by adding an environmental factor, parent-child relationship (attachment). The greatest anxiety (indicated by the sum of the number of anxiety disorders for which a child met diagnostic criteria) in children was shown by those who were identified as behaviorally inhibited and insecurely attached, as well as having anxious mothers. However, causal and directional conclusions could not be made due to concurrent data collection of the various indices.

Because there are few longitudinal studies examining the effects of temperament and the existing ones used different methods to obtain their samples and used different measurements of this construct, it is difficult to make meaningful generalizations. However, it appears that this biological trait may put at least some behaviorally inhibited children at increased risk for developing anxiety problems. With the interaction of additional environmental and psychosocial factors, behaviorally inhibited children may be at an even greater risk for anxiety disorders.

Psychosocial factors. Although various psychosocial factors (i.e., attachment, adverse peer relations, and stressful life events) have been proposed to increase risk in the pathway to difficulties with anxiety, only those of interest to this study will be examined. Supported by the Weems and Stickle (2005) model discussed in the next section and

previous research, cognitive processing distortions, cognitions regarding limited control, over-control and negative parenting practices, and parent psychopathology are some of the conceptual factors that have been identified as areas of risk for anxiety development.

Negative cognitive errors or cognitive processing distortions have been associated with childhood anxiety. Leitenberg, Yost, and Carroll-Wilson (1986) found that negative cognitive errors were used significantly more by both depressed and anxious children than unaffected children. More recently, mixed results have emerged regarding types of negative cognitive errors distinguishing between children who have problems with anxiety and those who have problems with depression (Epkins, 2000; Leung & Poon, 2001; Weems, Berman, Silverman, & Saavedra, 2001). Examples of these cognitive errors are overgeneralizing (believing that a single negative outcome will occur in all similar future cases), catastrophizing (expecting the worst possible outcome based on the belief that it is most likely to happen), personalizing (believing oneself to be responsible for all bad things), and selective abstraction (focusing on only the negative aspects of a situation) and are the same ones that were first described as used by depressed adults by Beck, Rush, Shaw, and Emery (1979). Kendall, Pimental, Rynn, Angelosante, and Webb (2004) proposed that the way in which children cognitive process is important because it gives behavior meaning and over time, children will interpret similar behaviors they experience in a similar style. Thus, eventually children will expect behavior and become anxious in advance.

Chorpita and Barlow (1998) conceptualize the development of childhood anxiety also as a result of faulty cognitions specifically in relation to children's perception of

control. This key construct is defined as having the ability to influence events and outcomes in the environment related to reinforcement. During their early development, children experience limited control over events in their lives which leads to an increased generalized propensity to perceive various aspects of life as not within their personal control. Anxiety is hypothesized as developing from limited control, unpredictability, or "helplessness" at one end of a continuum with the other end potentially being an extreme sense of no control, “hopelessness,” and depression (Alloy, Kelly, Mineka, & Clements, 1990; Chorptia & Barlow, 1998).

More recently, Chorpita, Brown, and Barlow (1998) found that restricted opportunity for personal control that permeated family environments was associated with anxiety in children. Thus, it is not necessarily the occurrence of a traumatic event in their early development that is associated with the development of childhood anxiety, but everyday interactions and outcomes within the family environment that children believe are not influenced by their own behavior. This learned perception contributes to the adoption of using an external locus of control which is associated with anxiety.

In regard to the development of anxiety, parenting practices that are characterized by high protection and over-control especially in conflictual situations may be associated with later anxiety disorders (Bogels & Brechman-Toussaint, 2006; Turner, Beidel, Roberson-Nay, & Tervo, 2003). Over-control parenting practices include a parent who restricts the child from interacting in developmentally appropriate activities. These parents may be likely to discourage children to think and act independently, disregard children’s different opinions, and frequently disrespect children’s viewpoints (Whaley,

Pinto, & Sigman, 1999). These kinds of parenting behaviors can also encourage a child to have an external locus of control which is thought to lead to feelings of helplessness. In addition it can lead to a family environment that is low on expressiveness, that is feeling limited in being able to openly express feelings, opinions, etc. which can contribute to children developing a perception of limited personal control leading to anxiety (Chorpita et al., 1998; Turner et al., 2003). Parental negativity is conceptualized as parenting practices that are low in warmth, critical, and rejecting. These negative parenting practices are hypothesized to affect childhood anxiety by contributing to children's cognitive processes and thereby developing a tendency to view their environment as more threatening. In a review, Bogels and Brechman-Toussaint (2006), suggest further research is needed on both types of parenting practices, parental over-control and parental negativity, to clarify individual, interactional, and causal contributions to the development of childhood anxiety.

These types of parenting practices have often been found in parents who have psychological difficulties themselves. Mothers who have anxiety difficulties have been rated as more critical and controlling of their children compared to control mothers (Whaley et al., 1999). They also demonstrated less affection, smiled less frequently, and were more negative while interacting with their children. Mothers with high levels of anxiety have also been found to exhibit lower expectations of their anxious children's coping abilities in stressful or challenging situations compared to mothers of children without anxiety disorders. These mothers may unintentionally encourage anxious

behavior and coping styles in their children (Kortlander, Kendall, & Panichelli-Mindel, 1997).

Integrative models. Some researchers have begun to develop theoretical models describing the etiology of childhood anxiety disorders that call for the inclusion of both biological factors as well as various environmental and psychosocial factors (Fredman, Hirshfeld-Becker, Smoller, & Rosenbaum, 2003; Morris, 2004; Weems & Stickle, 2005). Childhood anxiety disorders are hypothesized to be produced by a complex interaction of biological, cognitive, behavioral, and social factors in various contexts (i.e., home, school, community). Weems and Stickle (2005) proposed a general model of childhood anxiety with a tentative ordering of the theoretical processes. They hypothesized that:

> biological factors may appear early in the development of anxiety disorders, that the interpersonal or social aspects are likely to affect some of the other processes, and that the cognitive and learning factors are proximal to the development of anxiety disorder and may be responsible for some specificity in order development (Weems & Stickle, 2005, p.113).

As seen in Figure 1, the authors reiterate the concepts of equifinality and multifinality in that they suggest that different interactions of these factors may lead to the same outcome of an anxiety disorder. They also propose that biological, cognitive, behavioral, and social protective factors exist as well that may prevent a child who experiences many of the risk factors from developing anxiety disorders.

Fredman et al. (2003) also proposed a tentative general etiological model of anxiety acknowledging that questions regarding the specificity of risk factors for separate anxiety disorders and the mechanisms through which they contribute to disorders still

remain. They proposed that children have a biological predisposition of low tolerance for physiological arousal and/or easily acquire fear regarding certain objects, places, or situations. This predisposition may be exhibited as behavioral inhibition or a specific childhood anxiety disorder. Environmental factors such as parental modeling of poor coping, critical parenting style, stressful live events, and negative peer relationships may interact with children's "pre-existing biological diathesis" (Fredman et al., 2003, p.150) to shape children's beliefs that they are unable to cope with a dangerous world and that anxiety symptoms and interactions with others are uncontrollable.

Morris (2004) suggested one possible pathway to a specific childhood anxiety disorder, Social Phobia, in which an infant who is behaviorally inhibited is raised by a parent with anxiety difficulties which impairs the parent's ability to be nurturing and appropriately responsive to the child's needs. There may be a "poor fit between parent and child temperament styles" (Morris, 2004, p. 59) resulting in impaired attachment in parent-child relationships as well as subsequent relationships the child tries to initiate and maintain with peers. Unable to practice and develop healthy, appropriate social skills, a child may withdraw and become isolated which may generalize across social settings, promoting and maintaining a negative cycle. Morris (2004) explained that because there are "multiple entry points" (p. 59) on the pathway to the development of anxiety disorders in children, studying and understanding the etiology and course of anxiety disorders is challenging.

Comorbidity

Following from the two previously reviewed childhood disorders, an examination of comorbidity is necessary as AD/HD and anxiety are often experienced together in children. Historically, the term comorbidity has had various meanings. These meanings originated in the medical literature. In a diagnostic sense, comorbidity has been used to describe one disease exhibiting symptoms similar to a coexisting disease. In a prognostic sense, comorbidity refers to the increased risk of negative outcome due to combination or individual effects of having co-occurring diseases. In a pathogenetic sense, two diseases are related etiologically, not simply co-occurring. Finally, comorbidity can be used in a therapeutic sense to describe how a disease can affect the evaluation of treatment efficacy of a co-occurring disease (Kaplan & Feinstein, 1974; Lilienfield, Waldman, & Israel, 1994). More recently, Lilienfield et al. (1994) made the argument that the term comorbidity should not be used in the psychopathology literature because it creates confusion as to what meaning is intended, particularly co-occurrence or covariation. Co-occurrence is simply the presence of two diagnoses while covariation is the presence of two diagnoses occurring more often than by chance. In this paper, the term comorbidity will be used in the context of covariation; that is, that AD/HD and childhood anxiety have some similarities and occur together more frequently than by chance.

The question of primary diagnosis is also relevant in understanding comorbidity research in this area. Primary diagnosis can refer to the disorder that appeared first in a child's history. It can also refer to the diagnosis that is most important to the family, the diagnosis that is the focus of treatment, or the diagnosis that is causing the most

impairment in a child's life. In terms of this study, AD/HD is considered the primary diagnosis in that children who participated were recruited from an AD/HD clinic and research lab where they were referred for assessment due to questions of the presence of AD/HD.

AD/HD and Anxiety Comorbidity Rates

Multiple pathways may lead to the presence of anxiety in children with AD/HD. Prevalence studies of children whose primary diagnosis is an anxiety disorder have found comorbid AD/HD rates ranging from 10% to 17% (Beidel et al., 1999; Swedo, Rapoport, Leonard, Lenane, & Cheslow, 1989; Verduin & Kendall, 2003). In contrast, studies of children with AD/HD as the primary difficulty and comorbid anxiety have reported higher rates. In clinic-referred samples, anxiety disorders are present in approximately 25% to 50% of the AD/HD population (Biederman et al., 1992; Pliszka, 2000; Russo & Biedel, 1994). In community samples, comorbid anxiety disorders occur in 25% to 40% of the AD/HD population (Anderson et al., 1987; August et al., 1996; Bird et al., 1993; Cohen et al., 1993; Jensen et al., 1997). Within clinic and community samples, varying estimates are likely due to different diagnostic criteria and assessment tools, type and number of informants, and methods for obtaining samples (Jensen et al., 1997). Higher rates of comorbidity in clinic-referred AD/HD samples have been suggested as an overestimation of true comorbidity in the general population. Thus, elevated comorbidity in clinic populations is seen as a manifestation of more severe psychopathology and impairment in functioning, so that children with AD/HD and comorbid disorders are more likely to be referred for treatment than less severe noncomorbid AD/HD

(McConaughy & Achenbach, 1994). However, studies with both clinic-referred samples and community samples have consistently shown that children with AD/HD have "co-occurring" anxiety more often than by chance and cannot be attributed to solely factors such as referral biases and differences in diagnostic instruments, informants, or participant age (Angold et al., 1999).

Another area of research within AD/HD and comorbid anxiety is examining anxiety among AD/HD subtypes. In these studies, anxiety has often been subsumed under a broader construct of internalizing symptoms. Significant differences among comorbid internalizing symptoms have been found among AD/HD subtype groups in some research and not in other research (August et al., 1996; Eiraldi, Power, & Nezu, 1997; Willcutt et al., 1999). Some studies have found comorbid internalizing problems to be equally associated with AD/HD-I and AD/HD-C, while other research has demonstrated comorbid internalizing problems to be more strongly associated with only AD/HD-I or AD/HD-C alone. When studying AD/HD subtypes and patterns of comorbidity with anxiety specifically and not submerged in the category of internalizing disorders/symptoms, mixed results have also been found. Levy, Hay, Bennett, and McStephen (2005) found that higher rates of anxiety disorders were associated with AD/HD-C compared to AD/HD-I. Gender differences were also present with higher rates of anxiety among female children compared to male children. However, no differences have been found between children with AD/HD-I and children with AD/HD-C (Power, Costigan, Eiraldi, & Leff, 2004) and between genders in children with AD/HD (Gaub & Carlson, 1997) on levels of anxiety. Higher rates of anxiety among children

with AD/HD-C and among female children with AD/HD may be explained by anxiety being associated with greater impairment. AD/HD-C has been found to be a more severe disorder (i.e., greater number of symptoms among both hyperactivity-impulsivity and inattention dimensions, greater comorbidity, more academic problems) than AD/HD-I and AD/HD-HI in some studies (Faraone, Biederman, Weber, & Russell, 1998; Nolan, Volpe, Gadow, & Sprafkin, 1999; Wolraich, Hannah, Pinnock, Baumgaertel, & Brown, 1996). Research also suggests that although male children are more likely to be referred for evaluations, female children who are referred have more impairment (Eme, 1992). Nolan et al. (1999) found that within a group of children with AD/HD-C, female children had a greater number of symptoms in both inattention and hyperactive-impulsive dimensions, suggesting a more severe disorder. Research has indicated that lower thresholds may be needed to detect AD/HD symptoms in female children because DSM-IV symptom count criteria may underdiagnose female children who experience impairment and would benefit from further evaluation and treatment (Hinshaw, 2002; Waschbusch & King, 2006).

Limitations of research

Whereas research on children with AD/HD and comorbid anxiety has not investigated the question of whether or not anxiety experienced by children with AD/HD is comparable to that of children in the general population, research on AD/HD and comorbid anxiety has included the study of prevalence rates, patterns of AD/HD subtypes and comorbidity, and treatment outcome (August et al., 1996; Bird et al., 1993; Eiraldi et al., 1997; Faraone et al., 1998; Jensen et al. 2001; Nolan et al., 1999; Willcutt et al.,

1999). Although these studies contribute to furthering knowledge regarding AD/HD and anxiety, many questions are still left unanswered. For example, when performing different analyses, some studies often do not differentiate between comorbid mood disorders and anxiety, labeling comorbidity simply as internalizing problems (August et al., 1996). Also, when there is a distinction made between mood and anxiety difficulties, little is known about different types of anxiety disorders and some studies use only dimensional measures of anxiety (Eiraldi et al., 1997; Faraone et al., 1998; Nolan et al., 1999). As previously mentioned, the DSM-IV currently defines 10 clinical anxiety disorders that affect children (American Psychiatric Association, 1994). Although there have been mixed results, Social Phobia and Generalized Anxiety Disorder have been two of the most prevalent anxiety disorders in the smaller portion of studies that have attempted to address the prevalence rates of anxiety disorders among children with AD/HD (Manassis et al., 2000; Willcutt et al., 1999).

The current study attempts to target several of the shortcomings and ambiguous findings in research conducted so far addressing AD/HD and comorbid anxiety in children. First, it describes anxiety disorder rates in a population of children with AD/HD who have been diagnosed using comprehensive DSM-IV criteria in attempt to replicate and clarify previous findings. Thus, the participants were children who met full criteria for AD/HD, not simply symptom counts or absence of functional impairment. Second, the current study also attempts to clarify any differences in expression of levels of anxiety across AD/HD subtypes and/or between genders. The literature reviewed has shown mixed results with some indications that greater anxiety may be associated with

children who may experience more impairing forms of the disorder, AD/HD-C and female children. Finally, the current study takes the next step in this line of research by examining psychosocial factors that may predict the presence of anxiety in a clinic population of children with AD/HD using both dimensional and categorical measures of anxiety specifically and not "internalizing problems" in general. Use of both dimensional and categorical measures are used to aid in clarifying optimal methods of measuring anxiety in this population.

Conceptual Framework

Because most research has been limited to studying prevalence rates of comorbid anxiety and examining patterns of AD/HD subtypes and internalizing comorbidity, the next important step to take is to examine factors that may explain AD/HD and the presence of comorbid anxiety in children. Multiple pathways are likely to explain this particular comorbidity. Anxiety may precede AD/HD or vice versa. An alternative pathway may be that they develop concurrently. An examination of how children with AD/HD are susceptible to the influences of the previously mentioned psychosocial risk factors for childhood anxiety is necessary and is where understanding the common association of these childhood disorders can begin as depicted in Figure 2.

Because children with AD/HD have a disorder that is characterized by developmentally deviant levels of inattention and/or hyperactivity-impulsivity (American Psychiatric Association, 1994), they are inherently at risk for developing negative cognitions regarding control of their environment. First, without interventions, children with AD/HD simply have great difficulty physically controlling themselves as many of

their hyperactive-impulsive symptoms include behaviors such as fidgeting and squirming when seated, running around or climbing excessively in inappropriate situations, being "on the go" or acting as if "driven by a motor," and having difficulty waiting for a turn (American Psychiatric Association, 1994). In addition, children with AD/HD may feel they have little control over their attention as they may struggle with inattentive symptoms that include behaviors such as having difficulty sustaining attention, making careless errors in one's work, and being easily distracted (American Psychiatric Association, 1994). In turn, struggles with attention and impulsivity may make them vulnerable to adapting a negative cognitive style that not only affects their beliefs regarding control, but also contributes to a bias of making impulsive and automatic negative cognitive errors.

Other difficulties that may increase vulnerability to cognitive processing distortions and encourage negative cognitions regarding control of their environments include poor academic functioning and social functioning. Approximately 30-50% of children with AD/HD also have a learning disability (Barkley, 1990; Cantwell & Baker, 1992; Klein & Mannuzza, 1991; Lambert & Sandoval, 1980). Children with AD/HD experience increased academic failure including repeating a grade and contact with specialized services, such as academic tutoring and special classes compared to controls (Barkley, Anastopoulos, Guevremont, & Fletcher, 1991; Faraone et al., 1998). The persistent patterns of inattentive, intrusive, and excessive behaviors that children with AD/HD demonstrate also disrupt social interactions frequently leaving them with fewer friends than other children, less liked than others, and rejected by their peers (Erhardt &

Hinshaw, 1994; Karustis, Power, Rescorla, Eiraldi, & Gallagher, 2000; Pelham & Bender, 1982). Repeated struggles and failures in both academic and social functioning on a daily basis may make influential contributions to the perception of lack of control of their environment and use of negative cognitive errors in children with AD/HD.

Research has also demonstrated that parents of children with AD/HD may also exhibit similar negative parenting practices and parent psychopathology to those of parents of anxious children. Parents of children with AD/HD are more directive, giving more commands to them, are more critical toward them, are more negative, and are less likely to respond to social interactions initiated by them compared to parents of children without AD/HD (Barkley, 1990; Johnston & Mash, 2001). Mothers of children with AD/HD also have exhibited increased rates of anxiety compared to mothers of children without AD/HD (Chronis et al., 2003; Perrin & Last, 1996). August et al. (1996) found that mothers of children with AD/HD-only rated themselves as being more effective in disciplinary management and were less depressed than mothers of children with comorbid internalizing disorders. Thus, parenting practices and maternal psychopathology may be risk factors among children with AD/HD for developing internalizing disorders and anxiety specifically.

Research Questions and Hypotheses

In brief review, many children with AD/HD also experience comorbid anxiety disorders. Multiple pathways are likely responsible for AD/HD and comorbid anxiety; AD/HD may follow anxiety, the opposite may occur, or they may develop simultaneously. Although a longitudinal study would best address the unfolding of

factors that promote the presence of anxiety in children with AD/HD, the practical and budgetary constraints made it unfeasible for the current study. Alternatively, this study was designed as an initial step to begin addressing the question: What psychosocial factors increase the risk for anxiety in a population of children whose primary diagnosis is AD/HD? In addition, replicating and clarifying existing literature was addressed. Based on the conceptual and empirical literature previously reviewed, the following predictions were made:

- Consistent with prior research, children with AD/HD were expected to exhibit Social Phobia and GAD more often than other anxiety disorders.

- Due to experiencing both inattentive and hyperactive-impulsive symptoms, children with AD/HD-C often experience a higher degree of functional impairment than that associated with AD/HD-HI or AD/HD-I. Similar findings have been reported for girls with AD/HD. To the extent that greater impairment is frequently associated with developing comorbid difficulties, this may suggest a link to greater susceptibility for anxiety problems. Thus, higher levels of dimensional anxiety were expected to be found among children with AD/HD-C versus the other subtypes, as well as among girls versus boys regardless of subtype.

- Consistent with prior research and with the conceptual model presented earlier (Weems & Stickle, 2005), faulty thinking and parenting factors were expected to account for the greatest variance in both dimensional and categorical anxiety among children with AD/HD; to a lesser degree, school functioning, treatment, and demographic variables were expected to predict comorbid anxiety as well. Because it impairs daily functioning in ways that increase the risk for anxiety to occur, AD/HD symptomatology was also expected to explain variance in comorbid anxiety above and beyond that of the other variables.

CHAPTER II

METHOD

Participants

The present study used a clinical population of 63 children with AD/HD ranging in age from 8 to 12 years old and their mothers. Child participants were recruited from current and former clients of the UNCG AD/HD clinic and former participants in AD/HD Research Lab studies who had undergone comprehensive evaluations resulting in a diagnosis of AD/HD. They were identified as meeting DSM-IV criteria for AD/HD for this study based on a positive AD/HD diagnosis generated by maternal responses to the DISC-IV, accompanied by clinically significant scores on the attention problems and/or hyperactivity subscales of the BASC or ADHD Rating Scale – IV completed by parents. All three AD/HD subtypes were eligible.

Thirty-eight male children with AD/HD and 25 female children with AD/HD participated in the study. The participants' mean age was 9.8 years. Racial backgrounds represented that of the community with 76.2% Caucasian children, 20.6% African-American children, and 3.2% biracial children participating in the study. Their mothers' mean age was 39.4 years. This participant sample was similar to the population seen in the UNCG Psychology Clinic. A summary of demographic variables and frequencies of AD/HD subtypes are summarized in Table 1.

Measures

Several measures were employed to determine diagnostic status of the children in order to qualify for participation in the study and also to determine AD/HD subtype for subsequent analyses. Questionnaires assessing demographics, faulty thinking, parenting factors, school functioning, treatment, and AD/HD severity were selected and used to represent these domains in order to identify potential risk factors to predict association of anxiety in children with AD/HD.

AD/HD Diagnostic Status

Computerized Diagnostic Interview Schedule for Children - IV. (DISC; NIMH, 1997). The DISC-IV is a computerized structured diagnostic interview that evaluates for DSM-IV Axis I disorders. Responses to the interview are given in a yes/no format. Each question is read aloud to the parent exactly as presented on the computer, and the administrator enters the exact responses without interpretation. All aspects of the diagnosis are addressed. The interview has been found to be a reliable and valid measure (Schwab-Stone et al., 1996; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). Maternal responses to the AD/HD module were used to assess the presence of AD/HD in children.

Behavior Assessment System for Children. (BASC; Reynolds & Kamphaus, 1992). The BASC is a broad band rating scale, which consists of 109-148 items depending on the appropriate age form (child or adolescent in this study). There are both teacher and parent report versions, which have been shown to be psychometrically sound (Merenda, 1996). Each item is rated on a 4-point scale. The measure yields 9 clinical

scales, 3 adaptive scales, and 4 composite scores. The Hyperactivity and Attention Problems subscales were used to establish developmental deviance of these symptoms to aid in confirming AD/HD diagnoses.

Attention-Deficit Hyperactivity Disorder Rating Scale - IV. (DuPaul, Power, Anastopoulos, & Reid, 1998). The ADHD rating scale is an 18-item scale based on the DSM-IV criteria for AD/HD. There are teacher and parent report versions. This measure has been found to be a useful instrument for identification purposes in accordance with DSM-IV diagnostic criteria (DuPaul et al., 1998; DuPaul, et al., 1997) and has excellent reliability and validity (DuPaul et al. 1998). Each item is rated on a 4-point scale. The measure yields Inattentive, Hyperactive-Impulsive, and Total Scores, which were used to assess the developmental deviance of AD/HD symptoms in the children in order to confirm an AD/HD diagnosis.

Predictor Variables

Nowicki-Strickland Locus of Control Scale. (NSLOC; Nowicki & Strickland, 1973). This scale is a 40-item self-report measure designed to assess the degree of perceived control (success or failure) children think they have over the environment. Reliability and validity have been demonstrated (Kendall, Finch, Little, Chirico, & Ollendick, 1978; Nowicki & Strickland, 1973; Yates, Hecht-Lewis, Fritsch, & Goodrich, 1994). Responses are given in a yes/no format with higher scores indicating external locus of control. This measure was used as an indicator of the extent children used an external locus of control in their thinking.

Children's Negative Cognitive Error Questionnaire. (CNCEQ; Leitenberg et al., 1986). This questionnaire asks children to rate vignettes illustrating four types of cognitive distortions or negative cognitive errors (catastrophizing, overgeneralization, personalization, and selective abstraction) in three areas (social, academic, and athletic). Internal consistency and test-retest reliability have been demonstrated (Leitenberg et al., 1986). Children use a 5-point scale to rate how similar a thought is to how they would think in the situation. Total scores were used as an indicator of children's use of negative cognitive errors.

Parenting Stress Index - Short Form. (PSI-SF; Loyd & Abidin, 1985). The PSI-SF is a 36-item scale assessing parenting stress. This measure has excellent reliability and validity (Loyd & Abidin, 1985). Items are rated on a 5-point scale. The index is comprised of three domains: parental distress, parent-child dysfunctional interaction, and difficult child. The PSI-SF generates scores from each of these domains as well as a Total Stress Score. For the purposes of the present study, the PSI-SF Total Stress Score served as a parenting stress variable.

Symptom Checklist - 90- Revised. (SCL-90R; Derogatis, 1993). The SCL-90R is a 90-item scale assessing a broad band of psychological maladjustment in adults. The SCL-90R has adequate internal consistency and test retest reliability and validity (Pauker, 1999). Items are rated on a 5-point scale. The measure yields a General Severity Index Score and an anxiety subscale, which were employed in analyses. The measure was used to assess for general psychopathology and also anxiety in mothers.

Alabama Parenting Questionnaire. (APQ; Frick, Christian, & Wootton, 1999; Shelton, Frick, & Wootton, 1996). The APQ consists of 35 items assessing five parenting constructs. It includes a Parental Involvement scale, a Positive Parenting scale, a Poor Monitoring/Supervision scale, an Inconsistent Discipline scale, and a Corporal Punishment scale. Items assessing the first two scales are worded in the positive direction and items assessing the last three scales are worded in the negative direction. The items are rated using a 5-point scale (1=never to 5=always). The Parenting Composite score served as an indicator of negative parenting practices.

Child and Family Information Sheet. This form was completed by parents and designed to collect information on children's school functioning and treatment history. To assess school functioning, the frequencies of those children with learning disabilities, repeating grades, and obtaining special education services (i.e. tutoring, attending special classes, attending resource classes) were gathered. In addition, grades on their last report card served as a measure of academic performance. Due to various grading scales among different grade levels, academic performance was identified as "satisfactory" or "unsatisfactory" for analyses. Parents were also asked questions regarding their children's therapy and medication treatment as well as their own mental health treatment history to control for treatment effects in the analyses. Dichotomous groups were formed for analyses. For example, children who had received medication treatment for their AD/HD symptoms were coded as "1" and those who had not were coded as "0."

Attention-Deficit/Hyperactivity Disorder Rating Scale - IV. (DuPaul, Power, Anastopoulos, & Reid, 1998). See description above. This measure yields Inattentive,

Hyperactive-Impulsive, and Total Scores which were used to assess the severity of AD/HD symptoms in children. The adult self-report version was also completed by mothers to serve as a measure of their AD/HD symptomatology.

Outcome Variables

The presence of anxiety in children with AD/HD was measured through several different formats. Both parent and child perspectives were included. In addition, both dimensional and categorical anxiety were assessed.

Behavior Assessment System for Children. (BASC; Reynolds & Kamphaus, 1992). The Anxiety subscale from the parent version of the BASC was used as an outcome measure of dimensional anxiety.

Multidimensional Anxiety Scale for Children. (MASC; March, Parker, Sullivan, Stallings, & Conners, 1997). The MASC is a 4-point Likert self-report scale that represents anxiety in children aged 8 to 18 years. It has excellent internal reliability and satisfactory to excellent test-retest reliability. It demonstrates both convergent and divergent validity (March et al., 1997; March & Sullivan, 1999). The MASC consists of 39 items distributed across 4 major factors, 3 of which can be divided into 2 subfactors each. The factors include physical symptoms (tense/restless and somatic/autonomic), social anxiety (humiliation/rejection and public performance fears), harm avoidance (perfectionism and anxious coping), and separation anxiety. An overall score is a valid indicator of generalized anxiety and was used as an outcome measure of dimensional GAD.

Social Anxiety Scale for Children-Revised. (SASC-R; La Greca & Stone, 1993). The SASC-R is a 22-item scale that assesses three factors of child social anxiety: Fear of negative evaluation from peers; social avoidance and distress that is specific to new situations or unfamiliar peers; social avoidance and distress that is experienced more generally when with peers. Research has supported both reliability and validity of this scale. A self-report version uses a 5-point scale to rate how much each item is true for the child. In this study, the total score served as a measure of dimensional Social Phobia.

Computerized Diagnostic Interview Schedule for Children - IV. (DISC; NIMH, 1997). Parental responses to the anxiety modules (Social Phobia, Separation Anxiety Disorder, Specific Phobia, Panic Disorder, Agoraphobia, Generalized Anxiety Disorder, Obsessive-Compulsive Disorder, Posttraumatic Stress Disorder) were used to assess the presence of anxiety symptomatology in children and to determine categorical anxiety, specific anxiety diagnoses.

Procedure

Parents of current and former child clients of the UNCG AD/HD Clinic and former participants in AD/HD Research Lab studies who agreed to be contacted regarding potential participation in research projects were telephoned by the principal investigator in order to ascertain their interest in participation in the current study. Of those clients who were currently obtaining services in the AD/HD clinic who qualified for the study, their therapists informed their parent(s) of the study after receiving evaluation feedback, and the principal investigator then followed up with a telephone call to the client's parent if interested in hearing more about the study. Depending on

convenience for the participant's parent, research visits were conducted either at the UNCG Psychology Clinic or the participant's home. Before administering any assessment measures, the principal investigator explained the study in detail and obtained signed written consent from the parent and either the principal investigator or trained student assistant obtained and documented assent from the child. The parent was given a copy of the consent form.

In addition to the battery of interviews and questionnaires completed during evaluations of children for AD/HD in the UNCG AD/HD Clinic, this study added four written child self-report measures - the NSLOC, CNCEQ, SASC-R, and the MASC - and parent-completed anxiety modules of the DISC-IV. For those children whose evaluations were recently completed, parents were not asked to complete again the overlapping measures between those used in their child's evaluation and this study. For those children whose evaluations were completed beyond six months before being recruited for this study, mothers again completed the AD/HD Rating Scale-IV and the BASC to assess the current severity of their children's AD/HD symptoms and the status of comorbid anxiety. Participant's mothers were also administered the AD/HD and anxiety modules of the DISC-IV. To assess other areas of parental functioning, mothers completed the Self-Report Version of the AD/HD Rating Scale-IV, the PSI-SF, the SCL-90R, and the APQ. For those children who were recruited from prior AD/HD Research Lab studies, mothers were not asked to complete again the overlapping measures. Thus, mothers completed three additional anxiety modules of the DISC-IV, the Self-Report Version of the AD/HD Rating Scale-IV, the PSI-SF, the SCL-90R, and the APQ. All children

completed the NSLOC, CNCEQ, SASC-R, and MASC. These additional child self-report measures were administered by either the principal investigator or a trained student assistant if available and were each read aloud to child participants to account for different reading levels. The child-completed measures took approximately 45 minutes to 1 hour to administer. Before ending the research visit, children were also allowed to choose a small toy from a toy box and parents were paid $10 as compensation for their participation.

All administrations of the DISC-IV including those during the original evaluations and during the current study were conducted by doctoral students supervised by a licensed psychologist with a Ph.D. in clinical psychology who had received certified training in the administration of the DISC-IV. Doctoral students received didactic training in administration of the DISC-IV and conducted pilot cases before actual administration to clients and study participants, and thus, all doctoral students had extensive experience with DISC-IV administration.

CHAPTER III

RESULTS

Preliminary Analyses

Table 2 presents various means, standard deviations, and ranges of the data representing predictor and outcome variables. An examination of the distributions of each continuous variable, along with skewness and kurtosis statistics, revealed that the variables were approximately normally distributed with no significantly skewed variables or inflation of scores in the tails or centers of the distributions.

Rates of Anxiety Disorders

As seen in Table 3, the frequencies of the eight anxiety disorders assessed by the DISC are presented. Social Phobia (22.2%) and GAD (11.1%) are listed as the second and third most common anxiety diagnoses in this population of children with AD/HD. Because the DISC has been argued to produce false positive diagnoses of Specific Phobia, this disorder was not unexpectedly endorsed as the most common anxiety diagnosis (34.9%). Overall, 50.8% of the children in this study were positive for a DISC anxiety diagnosis.

Differences in Dimensional Anxiety by AD/HD Subtype and Gender

To aid in clarifying previous research studying subtype and gender differences in anxiety within AD/HD populations, a two-way factor analysis of variance (ANOVA) was conducted using the BASC anxiety subscale as the dependent variable. Results of the

ANOVA are given in Table 4. Within the analysis, only participants with AD/HD-IA or AD/HD-C were included as there were only two cases with AD/HD-HI, an insufficient number for the analysis. Neither main effect of AD/HD subtype or gender was found to be significant. However, there was a significant interaction effect of AD/HD subtype and gender on dimensional anxiety, $F(1, 57) = 5.116$, $p < .05$. Simple effects analyses revealed that male children with AD/HD-C were rated significantly more anxious than male children with AD/HD-IA. No other differences in anxiety were revealed by AD/HD subtype and gender.

Inter-correlations Among Demographic, Predictor, and Outcome Variables

A summary of correlations is presented in Table 5. Among predictor and outcome variables, gender (female) was associated with increased dimensional social anxiety ($r = .28$, $p < .05$). Children who had more external locus of control were associated with increased dimensional generalized ($r = .37$, $p < .01$) and social anxiety ($r = .52$, $p < .01$). Children who made more negative cognitive errors were also associated with having increased dimensional generalized ($r = .45$, $p < .01$) and social anxiety ($r = .73$, $p < .01$). As parenting practices became more negative they were associated with an increase in BASC dimensional anxiety, $r = .28$, $p < .05$. Children who had to repeat a grade in school were more likely to acquire a DISC diagnosis of Social Phobia, $r = .39$, $p < .01$. Having at least one special education service was also associated with an increase in BASC dimensional anxiety ($r = .25$, $p < .05$) as well as MASC generalized anxiety ($r = .25$, $p < .05$) and a DISC diagnosis of Social Phobia ($r = .27$, $p < .05$). Children who had therapy services were associated with an increase in BASC dimensional anxiety, $r = .33$,

$p < .01$. Children whose mothers had therapy were more likely to have a DISC anxiety diagnosis, $\underline{r} = .29$, $p < .05$.

Among predictor variables, an increase in age was associated with a decrease in AD/HD severity ($\underline{r} = -.27$, $p < .05$), decrease in Hyperactivity-Impulsivity severity ($\underline{r} = -.33$, $p < .01$), and decrease in external locus of control ($\underline{r} = -.32$, $p < .05$). Gender (female) was associated with increased academic performance ($\underline{r} = .25$, $p < .05$). Gender (male) was associated with having mothers with greater AD/HD symptom severity ($r = -.30$, $p < .05$) and with mothers who had therapy ($r = -.28$, $p < .05$). Children who used more external locus of control also made more negative cognitive errors, $\underline{r} = .41$, $p < .01$. Increased parenting stress was associated with increased general psychopathology ($\underline{r} = .30$, $p < .05$) and anxiety in mothers ($\underline{r} = .27$, $p < .05$) as well as with negative parenting practices ($\underline{r} = .57$, $p < .01$). In addition, children with increased total AD/HD severity ($\underline{r} = .37$, $p < .01$), Inattention severity ($\underline{r} = .29$, $p < .05$), and Hyperactivity-Impulsivity severity ($\underline{r} = .34$, $p < .01$), and children who had therapy ($\underline{r} = .36$, $p < .01$) and medication treatment ($\underline{r} = .26$, $p < .05$) were associated with increased parenting stress. Increased general psychopathology in mothers was associated with negative parenting practices ($\underline{r} = .26$, $p < .05$), increased anxiety in mothers ($\underline{r} = .82$, $p < .01$), increased mother AD/HD severity ($\underline{r} = .55$, $p < .01$), and mothers having had therapy ($\underline{r} = .44$, $p < .01$) and medication treatment ($\underline{r} = .42$, $p < .01$). Anxious mothers were more likely to have greater AD/HD severity ($\underline{r} = .49$, $p < .01$) and therapy ($\underline{r} = .31$, $p < .05$) and medication treatment ($\underline{r} = .28$, $p < .05$). Mothers with increased AD/HD severity were more likely to have had therapy treatment ($\underline{r} = .35$, $p < .01$) and medication treatment ($\underline{r} = .31$, $p < .05$).

Having at least one special education service was associated with decreased academic performance ($r = -.32$, $p < .05$) and increased total AD/HD severity ($r = .47$, $p < .01$), Inattention severity ($r = .42$, $p < .01$), and Hyperactivity-Impulsivity severity ($r = .40$, $p < .01$). Increased academic performance was associated with less Inattention severity, $r = -.25$, $p < .05$.

Among outcome variables, increased BASC anxiety was significantly associated with the presence of at least one DISC anxiety diagnosis ($r = .37$, $p < .01$) including DISC GAD ($r = .52$, $p < .01$) and DISC Social Phobia ($r = .29$, $p < .05$). Increases in SASCR social anxiety were associated with increases in MASC generalized anxiety, $r = .65$, $p < .01$.

Notable correlations among demographic variables included race being associated with increased maternal psychopathology ($r = .33$, $p < .05$), repeating a grade ($r = .25$, $p < .05$), and dimensional social anxiety ($r = .31$, $p < .05$).

Regression Analyses

As summarized in Table 6, predictor variables measuring the domains of demographics, faulty thinking, parenting factors, school functioning, treatment, and AD/HD severity were entered into hierarchical stepwise regressions and hierarchical forward stepwise logistic regressions to assess predictability of dimensional and categorical anxiety. Multicollinearity was checked for and determined not a problem.

Predictor Variables and Dimensional Anxiety

To determine the variance explained by the predictor variables in dimensional anxiety as measured by the BASC anxiety subscale, a hierarchical stepwise regression

analysis was conducted. Results of the regression are presented in Table 7. In the first step, the demographic variables (age and gender) were entered into the regression and were not significant in explaining variance in dimensional anxiety. Thus, they were excluded from the model as were the faulty thinking variables (NSLOC total score and CNCEQ total score) entered in step 2 which were also not significant. In step 3, the parenting factors variables were entered as a block (parenting stress, maternal general psychopathology, maternal anxiety, maternal AD/HD severity, negative parenting practices). Negative parenting practices as measured by the APQ Parenting Composite emerged as a significant predictor and was retained in subsequent steps (Adj. $R^2 = .060$, $p < .05$). In step 4, school functioning variables (learning disabilities, repeating grades, special education services, and academic performance) were entered into the regression model. Special education services were found to account significantly for an additional 6.1 % of the variance in dimensional anxiety above and beyond that of negative parenting practices (Cum. Adj. $R^2 = .121$, $p < .05$). In step 5, treatment variables (child therapy, child medication, maternal therapy, and maternal medication) were entered, and none was significant in explaining the variance in dimensional anxiety resulting in removal from the model. In the final step, AD/HD severity variables (ADHD-RS-IV total score, inattention score, and hyperactivity-impulsivity score) were entered, but were not significant in explaining any additional variance above and beyond that of negative parenting practices and special education services. Overall, the combination of negative parenting practices and special education services accounted for 12.1% of the variance in dimensional anxiety.

Predictor Variables and Dimensional GAD

To determine the variance explained by the predictor variables in dimensional GAD as measured by the MASC total score, a hierarchical stepwise regression analysis was conducted. Results of the regression are presented in Table 8. Using the same measures in the same order as the predictor variables in the above regression analysis, the demographic variables were entered into the regression first and were not significant. In step 2, the faulty thinking variables (NSLOC total score and CNCEQ total score) were entered to predict dimensional GAD. Children's negative cognitive errors (CNCEQ total score) emerged as a significant predictor in explaining dimensional GAD and was retained in the model (Adj. $R^2 = .187$, $p < .01$). Parenting factors, school functioning variables, treatment variables, and AD/HD severity variables were then entered into the regression as steps 3, 4, 5, and 6, respectively. None of these variables was significant in explaining any additional variance above and beyond that of children's negative cognitive errors in predicting dimensional GAD. Overall, children's negative cognitive errors accounted for 18.7% of the variance in dimensional GAD.

Predictor Variables and Dimensional Social Phobia

To determine the variance explained by the predictor variables in dimensional Social Phobia as measured by the SASCR total score, a hierarchical stepwise regression analysis was conducted. Results of the regression are presented in Table 9. Using the same measures in the same order as the predictor variables in the above regression analysis, the demographic variables (age and gender) were entered first to predict dimensional Social Phobia. Gender (female children) emerged as a significant predictor

and was retained in subsequent steps (Adj. R^2 = .065, p <.05). In step 2, the faulty thinking variables (NSLOC total score and CNCEQ total score) were entered. Both children's negative cognitive errors (CNCEQ total score) and children's locus of control (NSLOC total score) resulted as significant predictors in explaining dimensional Social Phobia and were retained in the model (Cum. Adj. R^2 = .561, p < .01; Cum. Adj. R^2 = .625, p < .01). Parenting factors were then entered as a block (parenting stress, maternal general psychopathology, maternal anxiety, maternal AD/HD severity, negative parenting practices) in step 3. Maternal general psychopathology emerged as a significant predictor (Cum. Adj. R^2 = .646, p < .05). School functioning variables, treatment variables, and AD/HD severity variables were then entered into the regression as steps 4, 5, and 6, respectively. None of these variables was significant in explaining any additional variance above and beyond that of gender, children's faulty thinking, and maternal psychopathology in predicting dimensional Social Phobia. Overall, gender (female children), children's faulty thinking, and maternal psychopathology accounted for 64.6% of the variance in dimensional Social Phobia.

Predictor Variables and Any Anxiety Diagnosis

To determine the ability of the variables in this model to predict the presence or absence of any anxiety disorder as measured by the DISC, a hierarchical forward stepwise logistic regression analysis was performed. Results are presented in Table 10 and Table 11. Using the same measures in the same order as the predictor variables in the above regression analyses, the demographic variables, faulty thinking variables, parenting factors, and school functioning variables were entered into the regression as

steps 1, 2, 3, and 4, respectively, to predict the presence or absence of any DISC anxiety disorder. None of these variables was significant in predicting the presence or absence of any DISC anxiety disorder and were removed from the model. In step 5, treatment variables (child therapy, child medication, maternal therapy, and maternal medication) were entered, and maternal therapy was found to be significant and retained in the model ($X^2 = 4.883$, $p < .05$). In the final step, AD/HD severity variables were entered, but were not significant in adding any predictive ability above and beyond that of maternal therapy to the presence or absence of any DISC anxiety disorder. Overall, mothers having therapy treatment accounted for 7.8% of the variance in predicting any DISC anxiety disorder above and beyond that of chance. Children whose mothers had had therapy treatment were 3.35 times more likely to have a DISC anxiety diagnosis. Maternal therapy correctly predicted absence of an anxiety disorder 75.9% of the time and presence of an anxiety disorder 51.6% of the time with overall predictive ability of 63.3%.

Predictor Variables and GAD Diagnosis

To determine the ability of the variables in this model to predict the presence or absence of GAD as measured by the DISC, a hierarchical forward stepwise logistic regression analysis was performed. Using the same measures in the same order as the predictor variables in the above logistic regression analysis, none of the predictor variables was found to be significant. Thus, none of the predictor variables significantly improved predicting GAD above and beyond that of chance.

Predictor Variables and Social Phobia Diagnosis

To determine the ability of the variables in this model to predict the presence or absence of Social Phobia as measured by the DISC, a hierarchical forward stepwise logistic regression analysis was performed. Results are presented in Table 12 and Table 13. Using the same measures in the same order as the predictor variables in the above logistic regression analyses, the demographic variables, faulty thinking variables, and parenting factors were entered into the regression as steps 1, 2, and 3, respectively, to predict the presence or absence of Social Phobia. None of these variables was significant in predicting Social Phobia resulting in removal from the model. In step 4, school functioning variables (learning disabilities, repeating grades, special education services, and academic performance) were entered into the logistic regression model, and repeating grades emerged as significant ($X^2 = 18.180$, $p < .01$). Treatment variables and AD/HD severity variables were then entered into the regression as steps 5 and 6, respectively. None of these variables was significant in adding any predictive ability above and beyond that of repeating grades to the presence or absence of Social Phobia. Overall, children who have repeated a grade in school accounted for 26.1% of the variance in predicting Social Phobia above and beyond that of chance. Children who had repeated a grade were 17 times more likely to have a DISC diagnosis of Social Phobia. Repeating grades correctly predicted absence of Social Phobia 95.7% of the time and presence of Social Phobia 50% of the time with overall predictive ability of 85%.

Differences in Dimensional and Categorical Anxiety by Child Medication Status

To clarify whether or not child medication status affected significant findings in regression analyses, child medication status was examined to determine if differences in dimensional anxiety and categorical anxiety existed simply due to medication treatment of AD/HD symptoms. Appropriate t-tests and chi-square tests were run and no significant differences in child anxiety resulted between the two medication groups. Thus, children who had been treated with medication for their AD/HD symptoms did not significantly differ from those children not taking medication in their level of anxiety, measured either dimensionally or categorically.

CHAPTER IV

DISCUSSION

Children with AD/HD develop difficulties with anxiety at a higher rate than the general population. Determining what processes may underlie this difference in rates of anxiety development is important to add to the existing research on children with AD/HD and comorbid anxiety, which currently focuses mainly on rates of anxiety comorbidity and patterns of anxiety/internalizing symptoms among AD/HD subtypes. Given that multiple pathways may lead to the co-occurrence of AD/HD and anxiety in children, this study's overall goal was to examine the factors that may link these two disorders in a clinical AD/HD population. In particular, this study asked: *What psychosocial factors increase the risk for comorbid anxiety in children with AD/HD?* Three hypotheses related to this question were investigated to clarify understanding of comorbid anxiety in children with AD/HD.

Hypothesis One

Consistent with prior research, children with AD/HD were expected to exhibit Social Phobia and GAD more often than other anxiety disorders.

Replicating previous research, children in this study were positive for anxiety disorders at a higher rate than the general population with 50.8% of the children meeting DISC criteria for at least one anxiety diagnosis. Hypothesis One was supported in that Social Phobia and GAD were found to be the second and third most common anxiety

disorders, respectively, in this population of children with AD/HD. As mentioned earlier, Specific Phobia was endorsed as the most common anxiety disorder. However, without additional clinical interviewing, the DISC tends to overdiagnosis this type of anxiety disorder due to many of the fears assessed being developmentally appropriate for children of this age (e.g., fear of the dark).

Hypothesis Two

Due to experiencing both inattentive and hyperactive-impulsive symptoms, children with AD/HD-C often experience a higher degree of functional impairment than that associated with AD/HD-HI or AD/HD-I. Similar findings have been reported for girls with AD/HD. To the extent that greater impairment is frequently associated with developing comorbid difficulties, this may suggest a link to greater susceptibility for anxiety problems. Thus, higher levels of dimensional anxiety were expected to be found among children with AD/HD-C versus the other subtypes, as well as among girls versus boys regardless of subtype.

Although there were no significant main effects of AD/HD subtype or gender on dimensional anxiety as measured by the BASC, a significant interaction between these two independent variables did exist; male children with AD/HD-C were more anxious than male children with AD/HD-IA. Thus, the hypothesis was partially supported in regard to subtype, but only in male children. Contrary to the hypothesis, there were no gender differences in anxiety. However, this finding is consistent with research on childhood anxiety with levels of anxiety being relatively equal until adolescence when girls generally experience increased anxiety compared to boys, a stage somewhat later in

development than that experienced by the child participants in this study. The findings of the current study may also aid in explaining mixed results in past AD/HD studies as all-male samples have been often used. Thus, gender composition of the samples may be critical in finding significant differences in anxiety among subtypes. Differences among anxiety levels may exist among subtypes in all-male samples but not in samples including boys and girls.

Hypothesis Three

Consistent with prior research and with the conceptual model presented earlier (Weems & Stickle, 2005), faulty thinking and parenting factors were expected to account for the greatest variance in both dimensional and categorical anxiety among children with AD/HD; to a lesser degree, school functioning, treatment, and demographic variables were expected to predict comorbid anxiety as well. Because it impairs daily functioning in ways that increase the risk for anxiety to occur, AD/HD symptomatology was also expected to explain variance in comorbid anxiety above and beyond that of the other variables.

Three separate hierarchical stepwise regression analyses were conducted using predictor variables representing six domains (demographics, faulty thinking, parenting factors, school functioning, treatment, and AD/HD severity) and three different dimensional anxiety outcome variables (BASC anxiety, MASC anxiety, and SASCR anxiety). In the first analysis, both negative parenting practices and special education services were significant in predicting BASC anxiety. Thus, those children whose mothers used more negative, critical parenting practices characterized by poorer

involvement in children's activities, poorer monitoring and supervision of children, more inconsistent in applying discipline, and greater use of corporal punishment were more likely to develop dimensional anxiety. This is consistent with the conceptual framework presented earlier in which it was suggested that mothers of children with AD/HD use similar negative parenting practices as those used by mothers of anxious children, and thus, may put children with AD/HD at greater risk of developing anxiety problems. In addition, children obtaining special education services, such as tutoring, special classes, and/or resource classes, were more likely to show greater anxiety, above and beyond that of having mothers who used negative parenting practices. Because school functioning is often negatively affected by AD/HD symptoms, children with AD/HD frequently need special education services and may feel self-conscious about experiencing learning difficulties leading to greater anxiety. The other school functioning variables, learning disabilities, repeating grades, and academic performance, were not significant in predicting dimensional anxiety. Compared to these variables, receiving special education services may have been a significant predictor due to this measure of school functioning being more "visible" to children leading those with AD/HD who receive special education services to be more aware of being different than those children with AD/HD not receiving special education services.

In the second analysis, children's negative cognitive errors were significant in predicting dimensional GAD. So, children who tended to make more negative cognitive errors such as catastrophizing, overgeneralization, personalization, and selective abstraction were more likely to experience generalized anxiety. Because of their

impulsivity and the daily impairment they experience in various areas, children with AD/HD may quickly jump to faulty conclusions and be at risk for employing this type of thinking style which has been implicated as a potential causal factor in childhood anxiety.

In the third analysis, female gender, children's negative cognitive errors, children's locus of control, and maternal psychopathology were significant in predicting dimensional Social Phobia. Thus, girls may be more at-risk than boys for developing anxiety problems at least when measuring social anxiety. This finding may appear contrary to the findings in Hypothesis 2 where no gender differences were found measuring anxiety with the BASC. However, the difference in findings is likely due to different forms of anxiety being measured, social anxiety and more general trait anxiety. Perhaps, girls with AD/HD are more socially impaired than boys with AD/HD due to social norms making it more acceptable for boys to be hyperactive. Thus, because girls with AD/HD appear so different from typical girls, they may experience repeated social failures more often than boys resulting in feeling more socially anxious. Children's faulty thinking, making negative cognitive errors and having an external locus of control, was also predictive of dimensional Social Phobia, demonstrating an expected link between children with AD/HD and anxiety. As stated earlier, children with AD/HD may be susceptible to making impulsive conclusions about situations and develop a feeling of life being out of their personal control as a result of daily impairment in interpersonal and school functioning. In respect to maternal psychopathology as a significant predictor, past research has indicated that parent anxiety is associated with greater anxiety in children, but not associated with the development of specific anxiety disorders in children

(Pine, 1999). Although, in this analysis, maternal psychopathology in general and not anxiety specifically predicted dimensional Social Phobia in children with AD/HD, maternal functioning is still supported as playing a role in their child's anxiety.

In sum, when accounting for variance in dimensional anxiety, the hypothesis that the domains of faulty thinking and parenting factors would result as the most influential predictive factors was partially supported. At least one predictor variable in one of these domains emerged as significant in explaining variance in each of the three dimensional outcome variables. In fact, these predictor variables are the only ones that emerged as significant except for obtaining special education services (predicting 6.1% of the variance in BASC anxiety) and female gender (predicting 6.5% of the variance in dimensional Social Phobia). The domains of faulty thinking and parenting factors were both represented in producing the models that explained the greatest amount of variance in dimensional anxiety.

Three separate hierarchical forward stepwise logistic regression analyses were also conducted using predictor variables representing the same six domains (demographics, faulty thinking, parenting factors, school functioning, treatment, and AD/HD severity) and three different categorical anxiety outcome variables (presence/absence of any DISC anxiety disorder, presence/absence of DISC GAD, presence/absence of DISC Social Phobia). The analyses examining categorical anxiety are more challenging to interpret. In the first analysis, whether or not mothers received therapy was significant in predicting the presence/absence of any DISC anxiety disorder. Children whose mothers had received therapy were 3.35 times more likely to have a

DISC anxiety diagnosis. This may indirectly indicate that mothers receiving treatment had been experiencing more severe psychological difficulties, but not reporting them at the time of study participation due to experiencing some relief from obtaining treatment. Their children may have been identified as positive for DISC anxiety diagnoses for various reasons including truly having a diagnosis or mothers having a bias for endorsing symptoms in their children as a result of their own distress. Past research has also shown that parents with their own anxiety difficulties reward more anxious and avoidant behavior in their anxious children (Barrett, Rapee, Dadds, & Ryan, 1996), which perpetuates their children's anxiety problems.

In the second analysis, none of the variables representing the six domains was significant in predicting the presence/absence of DISC GAD. Although children positive for DISC GAD represented one of the most common anxiety diagnoses in this sample, only seven children actually were identified with GAD which may have provided limited variability to find significant results.

In the third analysis, whether or not children repeated a grade emerged as significant in predicting the presence/absence of DISC Social Phobia. Children who repeated a grade were 17 times more likely to have a DISC Social Phobia diagnosis. Similar to special education services and dimensional anxiety, perhaps children with AD/HD who repeated grades were concerned that their peers were more aware of their school difficulties than of children with AD/HD who did not repeat grades leading them to be more anxious in their interaction with peers. From a different perspective, because direction cannot be determined from the correlational nature of the research design and

analyses, children with AD/HD and Social Phobia may be at risk for repeating grades. These children may have social anxiety that may make them miss school more often or simply impair their learning and academic performance so severely that they are required to repeat grades.

Unlike the regression analyses using dimensional anxiety outcome variables, the domains of faulty thinking and parenting factors were not supported as significant predictors in the development of categorical anxiety. Thus, the hypothesis was inaccurate in regard to explaining categorical DISC anxiety diagnoses. Only two variables emerged as significant in accounting for variance in categorical anxiety, maternal therapy and repeating grades. In addition, AD/HD severity was not significant in explaining variance in dimensional *or* categorical anxiety above and beyond the other variables, indicating that it may make an indirect rather than direct contribution to the development of anxiety. In sum, faulty thinking variables and parenting factors appear to be most consistent in predicting anxiety measured dimensionally rather than categorically in children with AD/HD.

Synthesis of Findings

Consistent with previous research on children with AD/HD (Anderson et al., 1987; Angold et al., 1999; August et al., 1996; Biederman et al., 1992; Bird et al., 1993; Busch et al., 2002; Cohen et al., 1993; Jensen et al., 1997; Russo & Biedel, 1994), 50.8% of the children in this study were identified as having an anxiety disorder as measured by the DISC representing a higher rate than in the general population. As hypothesized, Social Phobia and GAD were two of the most frequent comorbid anxiety disorders in this

population. These findings lend support to prevalence rates found in past studies. Finding no gender differences in BASC anxiety in this age group is consistent with research examining anxiety alone. For clinical purposes, being aware that both female and male children with AD/HD are at risk for comorbid anxiety is important so that it is not ignored in either population. However, potentially due to experiencing greater impairment, male children with combined type AD/HD may be at greater risk for comorbid anxiety relative to male children with the inattentive type.

Overall, this study lends support to faulty thinking variables and parenting factors being links to the development of anxiety in a population of children with AD/HD as represented in the etiological model presented in Figure 2. Children who tend to have a faulty thinking style, live in a more restrictive environment with negative parenting, and have mothers with greater psychopathology were predictive of experiencing more dimensional anxiety, and thus, may be at greater risk for developing anxiety problems. It is not surprising that the faulty thinking and parenting factor domains were significantly represented in predicting dimensional anxiety in children with AD/HD as these relationships are also indicated in the research on childhood anxiety alone (Chorpita & Barlow, 1998; Chorpita et al., 1998). The AD/HD severity domain did not directly explain any additional variance in dimensional or categorical anxiety. Because these children had diagnoses of AD/HD, there may have not been enough variability in the AD/HD predictor variables to achieve significance in predicting anxiety. Or, it can be concluded that the core AD/HD symptoms simply make no direct impact on the development of anxiety. Rather, they may affect other factors as visually depicted in

Figure 2, such as impairing school functioning and parenting practices which in turn may lead to an increase in faulty thinking and therefore contribute to anxiety difficulties.

Both dimensional and categorical measures of anxiety were examined as outcome variables in the analyses of this study. Based on the results of this study, potential risk factors were more consistent in predicting dimensional anxiety rather than categorical anxiety. Further, the risk factors identified using dimensional anxiety outcome measures were more consistent with past childhood anxiety research. The two significant predictors in the regression analyses using categorical outcome measures of anxiety were actually better at predicting the absence rather than the presence of anxiety. In addition, risk factors explained more variance in predicting dimensional measures of specific forms of anxiety rather than anxiety in general. Thus, based on the results, measuring anxiety dimensionally targeting specific types of anxiety may produce a more accurate picture when determining the developmental pathways of children with AD/HD.

Finally, results indicated that school functioning may also play an important role in the development of anxiety in this AD/HD population. Whether or not a child had repeated a grade, a measure of the school functioning domain, was predictive of DISC Social Phobia. Repeating a grade, coupled with special education services also being a significant predictor of BASC dimensional anxiety, lends support to school functioning being a possible risk factor that should be further studied to understand its role in facilitating the development of anxiety in children with AD/HD.

Implications for Clinical Practice

The findings of this study can aid in guiding evaluations of children who are demonstrating behaviors suggestive of AD/HD as well as potential treatments for children with AD/HD who experience difficulties beyond the core symptoms. Clinicians should be aware that children referred for AD/HD evaluations often have comorbid difficulties and these additional problems should be adequately addressed through appropriate assessments including those that can clarify the presence of any anxiety problems. This study lends support to including child-report measures to assess for anxiety in addition to parent-report measures as children may be more accurate in describing this internalizing problem. Early identification of comorbid anxiety and timely treatment may reduce the negative impact on daily functioning of these children and prevent additional difficulties such as depression from occurring. Because faulty thinking and parenting factors were indicated as possible risk factors in this study, treatments including cognitive-behavioral therapy as well as parent training may be beneficial to these children and families. Treating children with AD/HD who also have anxiety difficulties with stimulant medications alone in many cases would only be effective in improving the core AD/HD symptoms. Multi-modal treatment is clearly indicated in this specific population which is consistent with current research on treatments for children with AD/HD.

Limitations

One of the limits of this study is that it used a correlational research design. Thus, conclusive causal statements cannot be made. The possible risk factors identified in this

study may be responsible for anxiety as etiological factors, maintenance factors, or a combination of both. Thus, a longitudinal study would be ideal for examining more thoroughly the risk factors that contribute to the development of anxiety in children with AD/HD. It would also be helpful in determining the timing of the development of the disorders, although it is likely that it is different for different children; core symptoms of anxiety may precede the development of AD/HD, the opposite may occur, or the disorders may develop simultaneously. Further, as the symptom pattern of AD/HD changes as children age, how is comorbid anxiety affected? Does the pattern of risk factors change as children age? Are there different outcomes in impairment if both disorders are treated as early as possible? Due to time and budgetary constraints a longitudinal study was not possible for the current study.

Another limitation in this study is that the child participants who were identified as having an anxiety disorder categorically were not rated overall as clinically anxious by the dimensional measures of anxiety. Similarly, mothers did not rate themselves as significantly stressed, anxious, or with general psychopathology. This may suggest that children with AD/HD are at greater risk for developing anxiety disorders but a milder form of anxiety disorders than that of the general population. Results and interpretation may have been different with a population of children with more severe comorbid anxiety who have mothers with more severe psychopathology. Stronger relationships among more of the predictor and outcome variables may have emerged (e.g., mother anxiety and child anxiety). Because this study used a clinical population of children with AD/HD as participants, results cannot be generalized to all children with AD/HD in the general

population. Because not all children with AD/HD seek services, unknown biases may exist in this sample of children who did seek AD/HD evaluations.

For some of the analyses, increasing the number of participants would have increased power to find significant results that may truly exist. For example, when examining differences in anxiety by AD/HD subtype and gender, a greater number of participants may have aided in finding a significant main effect of AD/HD subtype as it approached significance (See Table 4) with current numbers. Likewise, a greater number of participants would have increased the variability to determine if any significant relationships between predictor and outcome variables were overlooked.

Another limitation of the study was the measure of academic performance within the school functioning domain. Because the child participants spanned ages (8 through 12 years old) and grade levels (1st through 7th grades), academic performance as measured by grades was coded as satisfactory/unsatisfactory. In school, younger children received labels as grades (i.e., satisfactory, needs improvement) while older children received letter grades. Thus, the measure of academic performance in this study was not standardized and was more of an estimate of academic functioning.

Although this study attempted to use both child and mother report measures to represent predictor and outcome variables to gain a more complete understanding of the development of anxiety in this population, again due to time constraints, equal representation of child report and mother report was unfeasible for this study. Given that children may be better reporters of internal processes, such as making negative cognitive errors and using an external locus of control, it is not surprising that these predictor

variables were significant in dimensional GAD anxiety and dimensional Social Phobia as these outcome measures were also child report measures. In addition, obtaining teacher-report questionnaires of childhood AD/HD and anxiety at the time of the study would have added to understanding in the school setting. On a similar vein, all measures used to assess anxiety were either in questionnaire or interview format. To obtain a more thorough measure of anxiety in this population, other assessment methods such as physiological measures, observation, and/or parent-child interactions could have aided in providing a clearer overall picture of anxiety.

Conclusion

Despite these limitations, this study adds to the research literature on children with AD/HD and comorbid anxiety. Consistent with past research, children with AD/HD in this study were shown to exhibit anxiety disorders at higher rates than the general population. This study added to the AD/HD literature in measuring comorbid anxiety both dimensionally and categorically, specifically demonstrating that Social Phobia and GAD were two of the most common anxiety disorders in this population. Results also supported anxiety differences among AD/HD subtypes with greater anxiety occurring in male children with AD/HD-C compared to male children with AD/HD-I. Even more importantly, this study examined factors to aid in determining the link between children with AD/HD and comorbid anxiety, identifying potential risk factors for the development of anxiety in this particular population. Results most consistently supported variables represented in the faulty thinking and parenting factor domains, and to a lesser degree in the school functioning domain, as possible risk factors affecting the developmental

pathway of anxiety in children with AD/HD. Although AD/HD severity made no direct contribution to comorbid anxiety above and beyond the other variables, it most likely makes an indirect contribution by its impact on the faulty thinking, parenting factor, and school functioning domains.

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Appendix A. Tables

Table 1
Demographic Variables and AD/HD Subtypes

Participant	Variable	
Child		
	Mean Age	9.8
	Gender	
	Male	38 (60.3%)
	Female	25 (39.7%)
	Race	
	Caucasian	48 (76.2%)
	African American	13 (20.6%)
	Biracial	2 (3.2%)
	AD/HD Subtype	
	Inattentive Type	29 (46.0%)
	Hyperactive-Impulsive Type	2 (3.2%)
	Combined Type	32 (50.8%)
Mother		
	Mean Age	39.4

Table 2
Description of Predictor and Outcome Variables

Variable	Male	Female	Frequency (%) no	(%) yes	*M*	*SD*	Min	Max
Predictor								
Child Age					9.83	1.41	8	12
Gender	38	25						
NSLOC					16.95	4.66	7	34
CNCEQ					51.41	16.96	24	90
PSITOT					86.97	23.53	44	144
SCLSEV					57.77	9.78	32	79
SCLANX					53.15	10.72	37	79
Maternal AD/HD					14.85	10.17	0	42
APQPC					-2.21	2.78	-8.00	5.73
LD			82.5	17.5				
Repeat Grd			79.4	20.6				
Special Ed Ser			54	46				
AcadPerf			20.6	79.4				
Child Therapy			57.1	42.9				
Child Med			11.1	88.9				
Maternal Therapy			63.5	36.5				
Maternal Med			52.4	47.6				
MTOT					35.97	10.02	17	54
MIA					20.19	4.45	10	27
MHI					15.78	7.06	0	27
Outcome								
MBANX					54.25	11.06	34	86
MASC					54.73	11.44	28	89
SASCR					45.43	15.58	18	82
DISCanxdx			49.2	50.8				
DISCgad			88.9	11.1				
DISCsopho			77.8	22.2				

Note. NSLOC = Nowicki-Strickland Locus of Control Scale total score; CNCEQ = Children's Negative Cognitive Error Questionnaire total score; PSITOT = Parenting Stress Index total stress score; SCLSEV = Symptom Checklist – 90 – Revised general severity index t-score; SCLANX = Symptom Checklist – 90 – Revised anxiety subscale t-score; Maternal AD/HD = ADHD Rating Scale – IV – Self-Report Version maternal AD/HD total severity for past 6 months; APQPC = Alabama Parenting Questionnaire parenting composite score; LD = learning disorder ; RepeatGrd = repeated a grade; SpecEdSer = special education services; AcadPerf = satisfactory academic performance; Child Therapy = child therapy treatment; Child Med = child medication treatment; Maternal Therapy = maternal therapy treatment; Maternal Med = maternal medication treatment; MTOT = child total AD/HD severity on ADHD Rating Scale – IV; MIA = child inattention severity on ADHD Rating Scale – IV; MHI = child hyperactivity-impulsivity severity on ADHD Rating Scale – IV; MBANX = BASC anxiety t-score; MASC = Multidimensional Anxiety Scale for Children total t-score; SASCR = Social Anxiety Scale for Children – Revised total score; DISCanxdx = any DISC anxiety diagnosis; DISCgad = DISC diagnosis of Generalized Anxiety Disorder; DISCsopho = DISC diagnosis of Social Phobia.

Table 3
Rates of Anxiety Disorders as Measured by the Diagnostic Interview Schedule for Children-IV (DISC)

Anxiety Disorder	Frequency (%)
Specific Phobia	34.9
Social Phobia	22.2
Generalized Anxiety Disorder	11.1
Separation Anxiety Disorder	9.5
Obsessive-Compulsive Disorder	1.6
Posttraumatic Stress Disorder	0
Panic Disorder	0
Agoraphobia	0
Any DISC Anxiety Disorder	50.8

Table 4
Two-Way ANOVA for Dimensional Anxiety as a Function of AD/HD Subtype and Gender

Source	*df*	*MS*	*F*	*p*
Gender	1	178.934	1.674	.201
Subtype	1	349.265	3.268	.076
Gender * Subtype	1	546.743	5.116	.028
Error	57	106.864		

Table 5
Correlations for Demographic, Predictor, and Outcome Variables

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. Age	-																
2. Gender	-.13	-															
3. Race	-.07	.18	-														
4. Grade	.92**	-.15	-.09	-													
5. Mother Age	.23	-.06	-.17	.21	-												
6. NSLOC	-.32*	-.03	-.09	-.30*	.07	-											
7. CNCEQ	-.12	.10	.19	-.15	-.07	.41**	-										
8. PSITOT	-.05	-.03	-.15	.01	.11	.14	.10	-									
9. SCLSEV	-.03	-.12	.33**	.00	-.12	-.12	.03	.30*	-								
10. SCLANX	-.03	-.12	.12	.02	-.05	-.08	-.03	.27*	.82**	-							
11. Mother AD/HD	.04	-.30*	.10	.04	-.18	-.03	.06	.07	.55**	.49**	-						
12. APQPC	.06	.05	.01	.09	-.08	-.03	.08	.57**	.26*	.14	.16	-					
13. LD	.15	-.20	-.15	.12	.02	-.05	-.03	.12	-.05	-.05	.05	-.04	-				
14. RepeatGrd	.04	-.09	.25*	-.19	-.03	-.03	.01	-.01	.03	-.03	-.01	-.04	.08	-			
15. SpecEdSer	-.02	-.10	.06	-.03	-.14	-.07	.07	.16	.01	.04	.03	-.06	-.01	.16	-		
16. AcadPerf	-.15	.25*	.06	-.16	.21	.11	.00	-.16	-.17	-.12	-.01	-.16	-.18	.07	-.32*	-	
17. Child Therapy	-.12	.02	-.01	-.07	.22	.21	.11	.36**	.09	-.03	-.03	.22	.02	.19	.17	.05	-
18. Child Med	.03	-.23	-.07	-.04	.03	-.15	-.08	.26*	.03	.06	.13	.14	.03	.18	.12	-.06	.10
19. Mother Therapy	-.05	-.28*	-.05	-.04	.06	.19	.04	.24	.44**	.31*	.35**	.18	-.18	.02	.09	-.02	.21
20. Mother Med	.07	-.06	-.01	.11	-.01	-.13	-.14	.06	.42**	.28*	.31*	.14	-.02	-.17	-.18	.02	.07
21. MTOT	-.27*	-.14	.11	-.27*	-.23	.18	.11	.37**	.13	.11	.01	.17	-.01	.12	.46**	-.23	.07
22. MIA	-.09	-.15	.07	-.12	-.27*	.15	.02	.29*	.13	.10	.11	.17	.07	.15	.42**	-.25*	-.16
23. MHI	-.33**	-.10	.12	-.31*	-.16	.16	.14	.34**	.11	.10	-.05	.14	-.06	.07	.40**	-.16	.21
24. MBANX	.22	-.16	-.03	.22	-.06	.17	.07	.10	.20	.20	.16	.28*	.16	.04	.25*	-.25	.33**
25. MASC	-.01	.04	.20	-.06	-.12	.37**	.45**	-.03	.12	.08	-.02	-.06	-.10	.14	.25*	-.10	.07
26. SASCR	-.17	.28*	.31*	-.19	-.22	.52**	.73**	.07	.11	-.02	.01	.13	-.06	.02	-.01	.05	.09
27. DISCanxdx	-.12	-.05	-.13	-.14	-.01	.17	.17	.08	.11	.15	.12	.14	.04	.19	.02	.13	.21
28. DISCgad	.08	.02	-.17	.04	-.09	.06	.08	-.05	.04	.02	.09	.14	.10	.07	.08	-.07	.20
29. DISCsopho	-.07	.04	.11	-.14	-.13	.08	.16	-.15	.10	.09	.09	-.07	-.25	.39**	.27*	.08	.23

Note. NSLOC = Nowicki-Strickland Locus of Control Scale total score; CNCEQ = Children's Negative Cognitive Error Questionnaire total score; PSITOT = Parenting Stress Index total stress score; SCLSEV = Symptom Checklist – 90 – Revised general severity index t-score; SCLANX = Symptom Checklist – 90 – Revised anxiety subscale t-score; Mother AD/HD = ADHD Rating Scale – IV – Self-Report Version mother AD/HD total severity for past 6 months; APQPC = Alabama Parenting Questionnaire parenting composite score; LD = learning disorder ; RepeatGrd = repeated a grade; SpecEdSer = special education services; AcadPerf = academic performance; Child Therapy = child therapy treatment; Child Med = child medication treatment; Mother Therapy = mother therapy treatment; Mother Med = mother medication treatment; MTOT = child total AD/HD severity on ADHD Rating Scale – IV; MIA = child inattention severity on ADHD Rating Scale – IV; MHI = child hyperactivity-impulsivity severity on ADHD Rating Scale – IV; MBANX = BASC anxiety t-score; MASC = Multidimensional Anxiety Scale for Children total t-score; SASCR = Social Anxiety Scale for Children – Revised total score; DISCanxdx = any DISC anxiety diagnosis; DISCgad = DISC diagnosis of Generalized Anxiety Disorder; DISCsopho = DISC diagnosis of Social Phobia.

*p < .05. **p < .01.

Table 5 (continued)
Correlations for Demographic, Predictor, and Outcome Variables

Variables	18	19	20	21	22	23	24	25	26	27	28	29
1. Age												
2. Gender												
3. Race												
4. Grade												
5. Mother Age												
6. NSLOC												
7. CNCEQ												
8. PSITOT												
9. SCLSEV												
10. SCLANX												
11. Mother AD/HD												
12. APQPC												
13. LD												
14. RepeatGrd												
15. SpecEdSer												
16. AcadPerf												
17. Child Therapy												
18. Child Med	-											
19. Mother Therapy	.27*	-										
20. Mother Med	.03	.60**	-									
21. MTOT	.10	.09	-.11	-								
22. MIA	.10	.13	-.03	.79**	-							
23. MHI	.08	.04	-.14	.92**	.49**	-						
24. MBANX	-.02	.18	.08	.22	.22	.17	-					
25. MASC	-.09	.13	-.14	.12	-.02	.19	.18	-				
26. SASCR	-.23	-.03	-.11	.21	.09	.24	.05	.65**	-			
27. DISCanxdx	-.05	.29*	.18	.07	.01	.09	.37**	.18	.13	-		
28. DISCgad	-.04	.05	.07	-.01	-.02	.00	.52**	.05	.06	.35**	-	
29. DISCsopho	.07	.23	-.05	.24	.25	.19	.29*	.10	.08	.53**	.30*	-

Note. NSLOC = Nowicki-Strickland Locus of Control Scale total score; CNCEQ = Children's Negative Cognitive Error Questionnaire total score; PSITOT = Parenting Stress Index total stress score; SCLSEV = Symptom Checklist – 90 – Revised general severity index t-score; SCLANX = Symptom Checklist – 90 – Revised anxiety subscale t-score; Mother AD/HD = ADHD Rating Scale – IV – Self-Report Version mother AD/HD total severity for past 6 months; APQPC = Alabama Parenting Questionnaire parenting composite score; LD = learning disorder ; RepeatGrd = repeated a grade; SpecEdSer = special education services; AcadPerf = academic performance; Child Therapy = child therapy treatment; Child Med = child medication treatment; Mother Therapy = mother therapy treatment; Mother Med = mother medication treatment; MTOT = child total AD/HD severity on ADHD Rating Scale – IV; MIA = child inattention severity on ADHD Rating Scale – IV; MHI = child hyperactivity-impulsivity severity on ADHD Rating Scale – IV; MBANX = BASC anxiety t-score; MASC = Multidimensional Anxiety Scale for Children total t-score; SASCR = Social Anxiety Scale for Children – Revised total score; DISCanxdx = any DISC anxiety diagnosis; DISCgad = DISC diagnosis of Generalized Anxiety Disorder; DISCsopho = DISC diagnosis of Social Phobia.

*p < .05. **p < .01.

Table 6
Predictor Variables Entered into Hierarchical Regression Analyses

STEP	DOMAIN	PREDICTOR VARIABLES
1	Demographics	Age Gender
2	Faulty Thinking	NSLOC CNCEQ
3	Parenting Factors	PSI total score SCL-90R: General Severity, Anxiety Adult ADHD Rating Scale total APQ Parenting Composite
4	School Functioning	Learning disability, Repeating grade, Special education services, Academic Performance
5	Treatment	Child Therapy Child Medication Maternal Therapy Maternal Medication
6	AD/HD Severity	ADHD Rating Scale – IV: Total score, Inattention severity, Hyperactivity-Impulsivity severity

Note. NSLOC = Nowicki-Strickland Locus of Control Scale total score; CNCEQ = Children's Negative Cognitive Error Questionnaire total score; PSI = Parenting Stress Index total stress score; SCL-90R = Symptom Checklist – 90 – Revised general severity index t-score, anxiety subscale t-score; Adult ADHD Rating Scale total = ADHD Rating Scale – IV – Self-Report Version maternal AD/HD total severity for past 6 months; APQ = Alabama Parenting Questionnaire parenting composite score.

Table 7
Summary of Hierarchical Stepwise Regression Model Predicting Dimensional Anxiety

STEP	VARIABLES ENTERED	STD BETA	ADJUSTED R^2	F CHANGE	p
1	Negative parenting practices	.275	.060	4.840	<.05
2	Negative parenting practices Special education services	.293 .273	.121	5.062	<.05

Table 8
Summary of Hierarchical Stepwise Regression Model Predicting Dimensional GAD

STEP	VARIABLES ENTERED	STD BETA	ADJUSTED R^2	F CHANGE	p
1	Negative cognitive errors	.447	.187	14.765	<.001

Table 9
Summary of Hierarchical Stepwise Regression Model Predicting Dimensional Social Phobia

STEP	VARIABLES ENTERED	STD BETA	ADJUSTED R^2	F CHANGE	p
1	Gender	.284	.065	5.174	<.05
2	Gender	.216	.561	67.787	<.001
	Negative cognitive errors	.707			
3	Gender	.235	.625	10.918	<.01
	Negative cognitive errors	.588			
	Locus of control	.287			
4	Gender	.257	.646	4.324	<.05
	Negative cognitive errors	.569			
	Locus of control	.315			
	Maternal psychopathology	.163			

Table 10
Logistic Regression Predicting Having Any Anxiety Diagnosis

Variable	*β*	*SE*	*Odds ratio*	*p*
Maternal Therapy	1.210	.563	3.352	.032
Constant	-.383	.335	.682	.253

Table 11
Classification of Predicted Any Anxiety Diagnosis

Observed		Predicted		
		DISC anxiety diagnosis		Percentage Correct
		no	yes	
DISC anxiety diagnosis	no	22	7	75.9
	yes	15	16	51.6
Overall Percentage				63.3

Table 12
Logistic Regression Predicting Having Social Phobia Diagnosis

Variable	*β*	*SE*	*Odds ratio*	*p*
Repeating Grade	2.833	.903	17.000	.002
Constant	-1.580	.415	.206	<.001

Table 13
Classification of Predicted Social Phobia Diagnosis

Observed		Predicted		
		Social Phobia diagnosis		Percentage Correct
		no	yes	
Social Phobia diagnosis	no	44	2	95.7
	yes	7	7	50.0
Overall Percentage				85.0

Appendix B. Figures

Figure 1. A general model of the etiology of childhood anxiety disorders based on Weems and Stickle (2005).

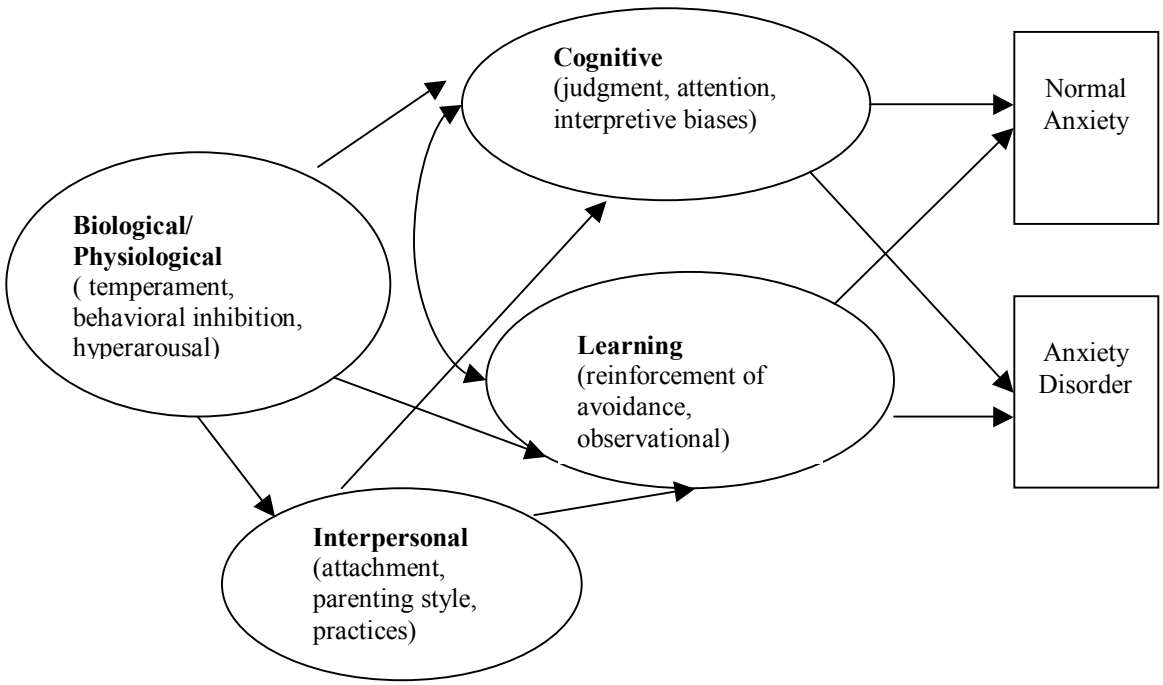
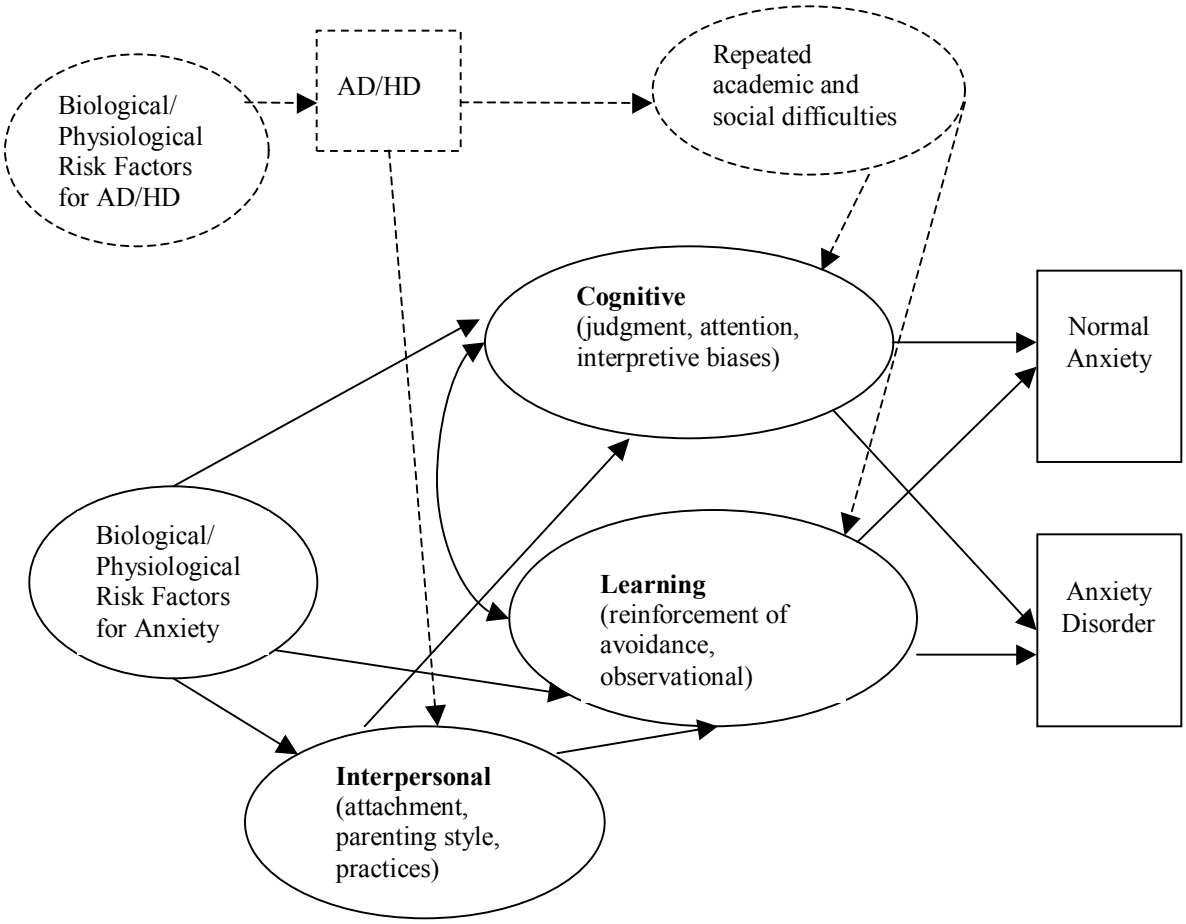


Figure 2. AD/HD added in dashed lines to depict a pathway of the development of AD/HD and comorbid anxiety based on Weems and Stickle (2005) model of anxiety.



Appendix C. Consent Forms

THE UNIVERSITY OF NORTH CAROLINA

GREENSBORO

CONSENT TO ACT AS A HUMAN PARTICIPANT:

Form for Parents/Guardians of Child Participants

Project Title: Factors Associated with the Development of Anxiety Among Children with Attention-Deficit/Hyperactivity Disorder

Project Director: Elizabeth McGee

Parent's Name: _____ Date of Birth: _____

Participant's Name: _____ Date of Birth: _____

Date of Consent: _____

Purpose:

Many children with AD/HD go on to develop secondary problems including difficulties with anxiety or worrying a lot about things in their lives. The purpose of this study is to aid in identifying risk factors for the development of anxiety among children with AD/HD. By identifying risk factors early in children's lives, certain treatments can then be used in specific areas of their lives to prevent the development of anxiety.

Procedure:

Your child will be asked to complete written questionnaires that will be read to them by a trained research assistant. These questionnaires will assess what they think about their environments including social, academic, and athletic scenarios. In addition, ideas about how their peers and authority figures perceive them will be measured. Finally, their report of physical symptoms will be collected, i.e., feelings of tension, restlessness. These questionnaires should take about one hour to complete. Your child will have the option to refuse to answer questions at any time.

This research project also requires a parent or caregiver to complete an interview and questionnaires. To assess the current severity of your child's AD/HD symptoms and the status of additional anxiety, you will complete two paper and pencil questionnaires on your child's behavior. In addition, a trained graduate student researcher will administer an interview to assess these behaviors. To assess other areas of parental functioning, you will complete questionnaires asking questions about your opinion of your child and your own psychological functioning. These questionnaires and interview should take about an hour to complete.

Risks and Discomforts:
The risks involved in this study are minimal. You and your child will be asked many personal questions regarding everyday life and how you respond in different situations. You or your child may feel uncomfortable or embarrassed when answering particular questions about personal information. If you feel uncomfortable at any time during the interview or when answering items on questionnaires, please notify the researcher immediately. If you continue to feel uncomfortable after sharing your concerns, you may withdraw from the study at any time without any consequences. If your child becomes uncomfortable or upset while answering the questionnaires, your child will also be given the option of withdrawing from the study at any time without any consequences.

Although the information collected during the interview and via questionnaires is kept confidential, the researcher will examine items that may indicate the possibility of you or your child endangering yourselves. For example, a researcher is required by law to report suspected physical abuse, sexual abuse, or other types of victimization. Should such a situation arise, the researcher will first discuss this with you. Emergency personnel or agencies will be provided if services are needed.

Benefits:
Children, families, schools, and society in general can benefit from this research. Children with AD/HD are at a much higher risk for developing difficulties with anxiety than children without AD/HD. This research can aid in determining more specifically factors that contribute to the development of anxiety in children with AD/HD, and thus, treatment can be aimed at lessening these effects and aid in preventing this comorbid difficulty. In addition, you will be mailed a summary of the results of the study, and suggestions for possible treatments and agencies that provide these services.

Confidentiality:
The answers to questions given by you and your child during the assessment visit will be kept confidential. As already mentioned, there may be a few cases in which the researcher cannot guarantee confidentiality. If the researcher learns of physical or sexual abuse that has not already been reported, the researcher must report this information to the Department of Social Services. In addition, if the researcher believes your child is in danger, the researcher is required by law to report this to the Department of Social Services. Again, you and your child have the right to refuse to answer questions at any time.

The data collected in this study will be identified by research numbers only and no individual's name will be directly associated with the data. It will be entered into password protected computer files. The original data will be stored in locked filing cabinets for 5 years and at that time the data will be shredded.

Consent:
By signing this consent form, you agree that you understand the procedures and any risks and benefits involved in this research. You and your child are free to participate or to withdraw your

consent to participate in this research at any time without penalty; your participation is entirely voluntary. Your privacy will be protected because you will not be identified by name as a participant in this project.

The research and this consent form have been approved by the University of North Carolina at Greensboro Institutional Review Board, which insures that research involving people follows federal regulations. Questions regarding your rights as a participant in this project can be answered by calling Dr. Beverly Maddox-Britt at (336) 334-5878. Questions regarding the research itself should be directed to Elizabeth McGee (334-5665, ext.2) or Dr. Arthur Anastopoulos (256-0006). Any new information that develops during the project will be provided to you if the information might affect your willingness to continue in the project.

By signing this form, you are agreeing to participate and to allow your child to participate in this project. Your child will also be given a description of the project. After hearing this, your child will be given the opportunity to give assent for participating in the project.

_____ _____

Parent/Guardian signature Witness to signature

ORAL PRESENTATION

(*When Obtaining Child Assent*)

Some children develop problems like worrying a lot about things in their lives. The purpose of this study is to learn more about children's worrying.

You will be asked to complete written questionnaires that will be read to you by a trained research assistant. These questionnaires will assess what you think about home, school, sports, and your friends. In addition, ideas about how you think about how your friends, classmates, parents, and teachers think about you will be measured. Finally, your report of feeling tense or restless will be collected. These questionnaires should take about one hour to complete. You will have the option to refuse to answer questions at any time.

You will be asked many personal questions regarding everyday life and how you respond in different situations. You may feel uncomfortable or embarrassed when answering particular questions about personal information. If you feel uncomfortable at any time during the interview or when answering items on questionnaires, please tell me immediately. If you continue to feel uncomfortable, you may withdraw from the study at any time.

If you should tell me something that makes me think that you may endanger yourself, I will contact your parent to ensure your safety.

Your answers to questions will be kept confidential. The data collected in this study will be identified by research numbers only and your name will not be directly associated with the data. It will be entered into password protected computer files.

_____

Signature of person obtaining consent on behalf of the University of North Carolina at Greensboro

_____

Date