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Around 25% of children with Attention-Deficit/Hyperactivity Disorder (AD/HD) have an anxiety disorder (Tannock, 2000), and children with AD/HD are three times more likely than children in the general population to have an anxiety disorder (Angold, Costello, & Erkanli, 1999). Past research has identified negative or controlling parenting practices, parental psychopathology, child externalizing symptoms, and child educational difficulties as potential risk factors for anxiety in children with AD/HD. However, the mechanisms underlying the relation between AD/HD and anxiety are unclear. The goal of this study was to further examine the parent factors related to symptoms of anxiety in children with AD/HD. It was hypothesized that parental symptoms of AD/HD and inconsistent parenting practices would predict symptoms of anxiety in children with AD/HD. Child perceptions of control were also hypothesized to moderate the relation between parenting practices and child anxiety symptoms.

The participants for the study included 28 children between the ages of 8 and 15 who had been diagnosed with AD/HD and at least one parent. A logistic regression analysis indicated that maternal psychopathology predicted a diagnosis of social phobia. Post-hoc analyses also indicated that mothers of children with AD/HD and social phobia had more symptoms of AD/HD than parents of children with AD/HD only. These findings have implications for theoretical understandings of AD/HD with comorbid anxiety, future research, and clinical practice with families of children with AD/HD.

PARENT FACTORS ASSOCIATED WITH ANXIETY IN CHILDREN WITH

ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

by

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

Attention-Deficit/Hyperactivity Disorder (AD/HD) and anxiety disorders are two

of the most common psychological disorders in children (Costello, Egger, & Angold,

2005; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Shaffer, Fisher, Dulcan, &

Davies, 1996). These disorders co-occur frequently in the same children; nearly 25% of

children with AD/HD also have an anxiety disorder, and around 10-15% of children with

an anxiety disorder also have AD/HD (Kendall et al., 2010; March et al., 2000; Tannock,

2000). A widely cited meta-analysis demonstrated that the odds of AD/HD and any

anxiety disorder being comorbid in a community sample range from 2.1 to 4.3, with a

median of 3.0 (Angold et al., 1999); therefore, a child with AD/HD or anxiety has a three

times higher chance of having the other disorder than a child without either of these

disorders.

Despite the substantial comorbidity[1] between AD/HD and anxiety disorders,

relatively little research has been conducted regarding the factors responsible for the

overlap between these disorders. Past cross-sectional research has demonstrated that

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[1] The term "comorbidity" has been used in many ways in the psychological literature (Angold et al., 1999; Kaplan & Feinstein, 1974; Lilienfeld, 2003; Lilienfeld, Waldman, & Israel, 1994; Piotrowski, 2007). It has been used to refer to the co-occurrence of one or more disorders, though different uses and interpretations of the term have varied with respect to whether the disorders must co-occur in time, how many disorders constitute a comorbidity, and whether symptoms (as opposed to clinical disorders) should be included. One common definition is two disorders that co-occur more frequently than would be expected by chance because they are etiologically related (Kaplan & Feinstein, 1974). This is the meaning of comorbidity that was used for the purposes of this project; that is, AD/HD and anxiety disorders co-occur in children at a rate that is significantly higher than would be predicted by chance based on their base rates in the general population.

overprotective and controlling parenting practices, lack of positive parenting, parental psychopathology, child educational difficulties, child externalizing behavior, and symptoms of inattention are associated with anxiety in children with AD/HD (Acosta et al., 2008; Baldwin & Dadds, 2008; Kepley & Ostrander, 2007; McGee, 2007; Moulton, 2006). These studies have been limited by imprecise measurements of parental psychopathology and by the cross-sectional nature of most of this research.

Understanding this common comorbidity is important for two reasons. First, children with comorbid AD/HD and anxiety have been shown to exhibit greater impairment in the domains of life stress and parental psychopathology than children with AD/HD only (Jensen, Shervette, Xenakis, & Richters, 1993). Second, children with AD/HD and an anxiety diagnosis have been found to respond differently to various psychosocial and pharmacological treatments than children with AD/HD only or an anxiety disorder only. One notable example of this differential response comes from the Multimodal Treatment of AD/HD (MTA) study, which compared behavioral treatment, medication, combination treatment (behavioral treatment and medication), and community care for the treatment of AD/HD in children. Secondary analyses of these data indicated that children with comorbid AD/HD and anxiety responded best to the combination treatment with regard to several outcomes, and that the effect sizes for all treatments were larger for children with comorbid AD/HD and anxiety than for the general sample (March et al., 2000). Therefore, an improved understanding of the risk factors for anxiety in children with AD/HD may enhance clinical work with children who

have AD/HD and comorbid anxiety and may also suggest unique treatment strategies that address these risk factors.

As a step towards clarifying the nature of the relationship between AD/HD and anxiety in children, this study was designed to further examine parent and parenting characteristics that are associated with anxiety in children with AD/HD. Specifically, this study examined inconsistent parenting practices as well as parental anxiety, depression, and AD/HD as predictors of anxiety in children with AD/HD. As a background to this investigation, brief overviews about what is known about AD/HD and anxiety are provided, followed by a review of the existing literature regarding theoretical reasons for the substantial comorbidity between AD/HD and anxiety and empirical research on this topic.

CHAPTER II

REVIEW OF THE LITERATURE

Attention-Deficit/Hyperactivity Disorder

AD/HD is characterized by developmentally deviant levels of inattention, hyperactivity, and/or impulsivity (American Psychological Association, 2000). Symptoms of inattention include lack of attention to details, making careless mistakes on schoolwork, difficulty sustaining attention, difficulty organizing tasks, and being easily distracted. Symptoms of hyperactivity include fidgeting with hands or feet, running about excessively in situations in which it is inappropriate to do so, acting "as if driven by a motor," and talking excessively. Symptoms of impulsivity include difficulty waiting for a turn and interrupting others.

In order to meet *Diagnostic and Statistical Manual—Fourth Edition—Text Revision* (*DSM-IV-TR*; American Psychological Association, 2000) criteria for AD/HD, a person must experience functional impairment in two or more domains, including social, academic, or occupational functioning, and this impairment must be related to symptoms of inattention, hyperactivity, or impulsivity (American Psychological Association, 2000). The person must display at least six of nine symptoms of inattention and/or at least six of nine symptoms of hyperactivity-impulsivity for at least six months, and these symptoms must be developmentally deviant. The symptoms must be present before age seven. Lastly, the symptoms must not be better accounted for by another disorder.

The average onset of AD/HD occurs between three and five years of age, and symptoms of hyperactivity, rather than inattention, are typically observed at this young age (Barkley, 2006). Children are likely to be diagnosed with AD/HD after entering formalized schooling, when the expectations of sitting still, listening to the teacher, following instructions, and sustaining attention are higher than for younger children; parents of school-age children may also struggle to help their child organize tasks and assignments and complete homework. Longitudinal research following individuals with AD/HD has found that while symptoms of hyperactivity may decline some in adolescence, 70-80% of children with AD/HD are likely to continue to experience significant symptoms of AD/HD into adolescence (Barkley, Fischer, Edelbrock, & Smallish, 1990). Regarding persistence of AD/HD into adulthood, research has been limited but seems to suggest that many adults diagnosed with AD/HD as children continue to experience difficulty with inattention, hyperactivity, and/or impulsivity as adults (Barkley, 2006).

Though estimates vary based on the assessment methods used and other methodological variations, most epidemiological studies have found that approximately 3-5% of children meet diagnostic criteria for AD/HD (Costello et al., 2003; Shaffer et al., 1996). Estimates of the ratio of boys to girls with the disorder also vary, but the average is 3:1 in non-clinic-referred children and between 5:1 and 9:1 in clinic-referred children (Barkley, 2006b). Children with AD/HD are more likely than children in the general population to experience difficulties in adaptive functioning, academic and intellectual functioning, and family and peer functioning (Frazier, Demaree, & Youngstrom, 2004;

Goode, 2002; Nixon, 2001; Stein, Szumowski, Blondis, & Roizen, 1995). Children with AD/HD are also more likely to meet criteria for other psychological disorders including Oppositional Defiant Disorder (ODD), Conduct Disorder (CD), depression, substance abuse, and, as previously reviewed, anxiety (Angold et al., 1999).

Current theories of the etiology of AD/HD posit that genetic factors that influence neurodevelopment likely cause AD/HD (for review, see Bradley & Golden, 2001). Family and twin studies have shown that the majority of variation in AD/HD-like symptoms can be accounted for by genetics and that other variability is accounted for by non-shared environmental factors, such as events that occur prenatally (Barkley, 2006a). Neurotransmitter dysfunction, particularly in the dopamine and norepinephrine systems, has also been found to be associated with AD/HD and implicated in its etiology (Pliszka, McCracken, & Maas, 1996). Genetics and early environmental effects likely contribute to structural and functional brain changes that cause the symptoms of AD/HD.

Studies of parenting of children with and without AD/HD have found that mothers of children with AD/HD were more likely to use power-assertive and controlling parenting techniques than parents of children that did not have AD/HD (Gerdes & Hoza, 2006; Moulton, 2006). Mothers and fathers of children with AD/HD have also been found to be less consistent in their parenting practices than parents of children without AD/HD (Ellis & Nigg, 2009); this effect was also found even when ODD and CD diagnostic status was controlled. Recent research suggests that parental AD/HD symptoms may influence parenting. Mothers of children with AD/HD who also had AD/HD themselves were less consistent disciplinarians than mothers who did not have

AD/HD (Chronis-Tuscano et al., 2008; Murray & Johnston, 2006). Other research indicates that maternal responses to child behavior are warmest when the mother and child both have high levels of AD/HD symptoms (Psychogiou, Daley, Thompson, & Sonuga-Barke, 2008).

Anxiety Disorders

The current *DSM-IV* defines ten anxiety disorders: Separation Anxiety Disorder (SAD), Specific Phobia, Generalized Anxiety Disorder (GAD), Social Phobia (SP), Panic Disorder with and without agoraphobia, Agoraphobia without panic disorder, Obsessive-Compulsive Disorder (OCD), Post-Traumatic Stress Disorder (PTSD), and Acute Stress Disorder (American Psychological Association, 2000). Separation Anxiety Disorder can only be diagnosed in children; the other anxiety disorders can be diagnosed in children and adults. Generalized Anxiety Disorder (GAD) and Social Phobia (SP) are the most commonly diagnosed anxiety disorders in children with AD/HD; the other anxiety disorders also co-occur with AD/HD, but at lower rates (Elia, Ambrosini, & Berrettini, 2008; Last, Perrin, Hersen, & Kazdin, 1992; Vance et al., 2002). The present review of anxiety disorders in children will therefore focus primarily on GAD and SP.

The hallmark feature of GAD is excessive worry and anxiety about a variety of events not confined to the specific worries present in other anxiety disorders such as SP or OCD. In children, these worries frequently concern health, school, disasters, future events, and personal harm (Weems, Silverman, & La Greca, 2000); however, the fears are not focused on a single specific object or event as in specific phobias. The *DSM-IV-TR* criteria for a diagnosis of GAD require excessive anxiety and worry occurring on

most days over a period of six months and about a variety of topics (American Psychological Association, 2000). The child must find it difficult to control the worry, and in children the worry must be accompanied by at least one of the following physical symptoms: restlessness, being easily fatigued, difficulty concentrating, irritability, muscle tension, or sleep disturbance. The worry must also cause impairment in one or more domains of functioning. The current *DSM-IV-TR* classification recognizes GAD as a disorder that can affect both children and adults.

The hallmark feature of SP is an extreme fear of social or performance situations (American Psychological Association, 2000). In SP, these types of situations are avoided or endured with extreme nervousness or distress, and this anxiety or avoidance must interfere significantly with one's normal functioning. In order to diagnose this disorder in a child, the child must show evidence of having the capacity for peer relationships and the anxiety must occur in the context of interactions with peers, not just in interactions with adults, and the duration of the fear must be at least six months.

As with AD/HD, prevalence estimates for anxiety disorders in children vary widely due to assessment procedures and other methodological variations. Estimates of the prevalence of anxiety disorders in children and adolescents range from 2.8% for a point prevalence to lifetime prevalence estimates of 8.7% to 20.7% (Costello, Egger, & Angold, 2005). GAD , SAD, and SP are the most common anxiety diagnoses in children (Costello et al., 2005). Anxiety disorders commonly co-occur with each other as well. Girls are more likely than boys to report an anxiety disorder of some sort, although sex differences are not usually large (Costello, Egger, & Angold, 2004). However, this effect

has been found to hold even when other factors associated with both sex and anxiety, such as self-esteem, social support, social competence, social support, and physical health, are controlled (Lewinsohn, Gotlib, Lewinsohn, Seeley, & Allen, 1998).

An analysis of the developmental course of anxiety disorders indicated that there are not yet well-defined developmental pathways that illustrate consistency or change in these disorders over time (Warren & Sroufe 2004). The average ages of onset for GAD and SP appear to be in pre-adolescence or early adolescence. The average age of onset for GAD in clinical samples, based on retrospective recall, ranges from 6.5 to 23.6 (Anderson, Noyes, & Crowe, 1984; Hammerness et al., 2008; Last, Hersen, Kazdin, & Finkelstein, 1987; Massion, Warshaw, & Keller, 1993; Noyes, Woodman, Garvey, & Cook, 1992; Woodman, Noyes, Black, Schlosser, & Yagla, 1999). Estimates of the age of onset for SP in clinical samples indicate that the average age of onset for SP is slightly older, around age 11 (Last et al., 1992). The ages of onset for individual participants in these studies extends from early childhood through late adulthood. Some data suggest that onset of GAD peaks between the ages of 5 and 8 as well as between the ages of 16 and 19 (Campbell, Brown, & Grisham, 2003). Several studies have also reported that older children and adolescents tend to have more symptoms of GAD than younger children and that GAD is more likely than other anxiety disorders to have a gradual, rather than sudden, onset (Anderson et al., 1984; Tracey, Chorpita, Douban, & Barlow, 1997). Over the lifespan, the course of both GAD and SP is generally thought to be chronic, although the anxiety may fluctuate based on life demands and stressors (American Psychological Association, 2000).

Several factors have been implicated in the etiology of anxiety disorders in children, including genetics, temperament, parental anxiety, and parenting practices. A study of twins has shown that the heritability of GAD is around .25 (Ehringer, Rhee, Young, Corley, & Hewitt, 2006). There is also a relation between the temperamental feature of behavioral inhibition (BI) and increased risk for anxiety disorders (Flannery-Schroeder, 2004). BI is generally associated with irritability, fearfulness, shyness, and withdrawal in children, and it is believed to be highly heritable (Robinson, Kagan, Reznick, & Corley, 1992). Children who display BI in infancy or toddlerhood are more likely than uninhibited children to develop anxiety disorders in later childhood (Rosenbaum, Biederman, Bolduc-Murphy, & Faraone, 1993). In addition, children that express stable BI across early childhood are more likely to experience anxiety disorders in childhood (Hirshfeld, Rosenbaum, Biederman, & Bolduc, 1992). It is important to note that while BI is a risk factor for the development of anxiety disorders, not all children who are behaviorally inhibited as young children go on to develop clinically-significant anxiety disorders; Manassis, Hudson, Webb, and Albano (2004) note that while BI is a risk factor for the development of anxiety disorders in children, other factors must also be considered in predicting or explaining the development of anxiety disorders in children.

Family factors are also thought to play an important role in the etiology of anxiety disorders. Research has consistently shown that a harsh, controlling, or overprotective parenting style is associated with childhood anxiety disorders across cultures (Bayer, Sanson, & Hemphill, 2006; Heider et al., 2008; Manassis, Hudson, Webb, & Albano, 2004; Silva, Dorso, Azhar, & Renk, 2007). Specifically, a meta-analysis of the literature

10

on parenting style and anxiety disorders suggests that the parenting factors of control and autonomy-granting account for more of the variance in childhood anxiety than parental warmth (McLeod, Wood, & Weisz, 2007). Lastly, robust findings in numerous studies have demonstrated that children with anxiety are also likely to have parents or other relatives with anxiety disorders (Marks, 1986). While this phenomenon may be partly explained by the genetic heritability of a predisposition for anxiety, family influences on anxiety are also thought to occur through processes such as modeling and information transfer (Beidel & Turner, 2005).

Several integrated models of the etiology of anxiety disorders have been proposed. Chorpita and Barlow's (1998) model of anxiety development (see Figure 1) is widely cited in the anxiety literature and integrates biological, temperamental, and environmental factors to show how a child may progress from having a behaviorally inhibited style in early childhood to clinically significant anxiety in later development. The model proposes that the experience of uncontrollable events during early development influences children's perceptions of control. These perceptions of low control lead to increased behavioral inhibition, which is associated with particular somatic correlates including heightened activity of the hypothalamic-pituitary-adrenal (HPA) axis, reticular activating system (RAS), and sympathetic nervous system. During later development, perceptions of low control may intensify the activity of these physiological correlates. Furthermore, perceptions of low control and experiences of low control are learned, so that processing of experiences during later development is biased by earlier memories of uncontrollable experiences. In early development, perceptions of

control are thought to mediate the relationship between stimuli and inhibition or anxiety; in later development, perceptions of control are thought to moderate this relationship. In a study designed to test this model, Choate-Summers (2006) found that an external locus of control (a child's perception that their actions and other events are outside of their own control) was associated with anxiety in children and adolescents; further, locus of control mediated the relation between a behaviorally-controlling family environment and anxiety in children and moderated this relationship in adolescents. This research supports various parts of Chorpita and Barlow's (1998) model.

Comorbid AD/HD and Anxiety: Possible Developmental Pathways

Although the existence of substantial comorbidity between AD/HD and anxiety is well-documented, the reasons for the comorbidity are not well-understood. Tannock (2000) and Lilienfeld (2003) have both outlined several possible reasons for the substantial comorbidity between AD/HD and anxiety in children, many of which are not supported by current research in this area. One of these possibilities is that anxiety may develop first and cause inattention; however, these attention problems do not share the same neuro-developmental etiological process that AD/HD is thought to have. A second possibility is that the substantial comorbidity between AD/HD and anxiety disorders could be caused by shared risk factors for both disorders. Several studies have investigated this possibility; however, no shared genetic or executive functioning risk factors have been identified (Biederman, Faraone, Keenan, Steingard, & Tsuang, 1991; Braaten et al., 2003; Perrin & Last, 1996; Piper, 2007; Rommelse et al., 2009). A third possible explanation is that what is currently conceptualized and diagnosed as comorbid

AD/HD and anxiety could be a distinctive disorder with its own etiology, course, and treatment response. Jensen, Martin, and Cantwell (1997) reviewed the literature on AD/HD and comorbid anxiety and suggested that this presentation may be better characterized as a distinctive disorder; however, this idea does not seem to have gained support in the empirical literature or in reviews published since this time.

One possible explanation for the overlap between AD/HD and anxiety that has gained support is that AD/HD emerges first and makes the development of anxiety more likely. The symptoms of AD/HD and/or the persistent difficulties encountered by a child with AD/HD may contribute to the development of anxiety in some children. Longitudinal research has demonstrated that AD/HD status predicts anxiety in later development, which supports the possibility that having AD/HD makes the development of anxiety more likely. A longitudinal study of a diverse sample found an odds ratio of 10.3 (95% CI=2.7-39.3) for children with AD/HD to have anxiety or depression at eight-year follow-up (Bussing, Mason, Bell, Porter, & Garvan, 2010). Further, five-year and ten-year follow-ups indicate that girls who met DSM-III-R criteria for Attention Deficit Disorder (ADD) experienced anxiety disorders, as well as mood disorders, antisocial disorders, and substance use disorders, at a much greater rate than girls who did not meet criteria for ADD at baseline (Biederman et al., 2006; Biederman et al., 2010).

Consistent with this possibility, the average ages of onset and etiologies of AD/HD and GAD suggest that clinically significant symptoms of anxiety may develop after the onset of AD/HD; this sequence has recently been termed a "post-comorbidity" (Taurines, 2010). Epidemiological studies have shown that the average age of onset for

AD/HD is between three and five years old (Barkley, 2006b), whereas the average age of onset for GAD is in early adolescence (Anderson, Noyes, & Crowe, 1984; Hammerness et al., 2008; Last, Hersen, Kazdin, & Finkelstein, 1987; Massion, Warshaw, & Keller, 1993; Noyes, Woodman, Garvey, & Cook, 1992; Woodman, Noyes, Black, Schlosser, & Yagla, 1999). In an epidemiological study of preschool children, 12.8% of children had met criteria for any subtype of AD/HD in the previous three months, whereas less than 1% of children had met criteria for GAD in the previous three months (Lavigne, LeBailly, Hopkins, Gouze, & Binns, 2009). Further, the etiology of AD/HD is thought to be primarily biological and neurological, whereas the etiology of GAD and anxiety more generally is thought to be influenced by a combination of family and environmental factors, genetics, and temperament. Specifically, Chorpita and Barlow's (1998) model of anxiety development theorizes that early and repeated experiences with uncontrollable or unpredictable events contribute to external loci of control in both early and later development; that is, symptoms of anxiety are present in later development due to experiences with uncontrollable or unpredictable events in earlier childhood. Based on these factors, it is plausible that many children may develop anxiety as a result of the persistent difficulties encountered by a child with AD/HD.

Recent studies have begun to examine potential risk and protective factors for anxiety disorders in children with AD/HD. In a cross-sectional study of a community sample, parents of children who had comorbid AD/HD and anxiety were less encouraging of independence and more controlling than parents of children who had AD/HD only and parents of control children (Kepley & Ostrander, 2007). Another cross-

14

sectional study of children with AD/HD found that negative parenting practices, along with special education services, predicted greater anxiety symptoms; that negative cognitive errors predicted a diagnosis of GAD; and that gender, negative cognitive errors, a more external locus of control, and maternal psychopathology predicted a diagnosis of SP (McGee, 2007). Similarly, a cross-sectional study in a large sample of children with AD/HD found that maternal, but not paternal, symptoms of anxiety as well as overprotective parenting and lack of positive parenting predicted anxiety diagnoses in children (Pfiffner & McBurnett, 2006). Further, a study of children with AD/HD found that symptoms of AD/HD and parenting factors (including parenting style, discipline techniques, and attributions regarding child behavior) predicted child symptoms of anxiety, but that the interaction between these two factors did not predict additional variance above and beyond the main effects (Moulton, 2006). Only one longitudinal study has been conducted on this topic; this study found that child temperament and externalizing symptoms at baseline were associated with comorbid AD/HD and anxiety symptoms at one-year follow-up (Baldwin & Dadds, 2008). Notably, several studies have found that parent factors, such as parental psychopathology and negative or controlling parenting styles, are associated with anxiety in children with AD/HD. It is important to interpret findings from these cross-sectional studies cautiously, since these potential risk factors may have preceded the symptoms of anxiety, developed at the same time as the symptoms of anxiety, or appeared after the symptoms of anxiety. However, it appears that further investigation of these parent factors may be warranted, particularly with

regard to identifying mechanisms that may help to explain the connection between parent factors and anxiety in a population of children with AD/HD.

Goals and Hypotheses of the Present Study

In review, AD/HD and anxiety disorders are common disorders in childhood and commonly co-occur (Tannock, 2000); children with AD/HD are three times more likely than children in the general population to develop an anxiety disorder (Angold et al., 1999). Although there are several potential developmental pathways for how a child could develop both AD/HD and an anxiety disorder (Tannock, 2000), current research findings point towards children developing AD/HD first and developing an anxiety disorder in later childhood or adolescence. Previous research in this area has identified controlling and negative parenting practices, maternal psychopathology, child negative cognitive errors, child locus of control, child temperament, and child externalizing disorders as predictors of anxiety in children with AD/HD. Because parenting practices and parental psychopathology have been associated with both AD/HD and anxiety in children, further investigation of these parent-related variables is warranted in order to determine whether they may help to explain the substantial comorbidity between AD/HD and anxiety.

The overall aims of this project, therefore, were to anchor this research on the risk factors for anxiety in children with AD/HD within a theoretical framework and to expand on the existing research. Chorpita and Barlow's (1998) model of anxiety development hypothesizes that exposure to events that are perceived as unpredictable in early development contributes to later anxiety; it is possible that children with AD/HD

experience many unpredictable events in childhood, both as a direct result of their symptoms of inattention, hyperactivity, and impulsivity, and as a result of the other persistent difficulties encountered by a child with AD/HD. In particular, parents of children with AD/HD are more likely to use inconsistent parenting practices (Ellis & Nigg, 2009). Parents of children with AD/HD who also have AD/HD themselves have also been found to use less consistent discipline than parents who do not have AD/HD (Chronis-Tuscano et al., 2008; Murray & Johnston, 2006). Inconsistent parenting may be experienced by children as unpredictable or uncontrollable. Chorpita and Barlow's model of anxiety development also proposes that locus of control moderates children's experiences of unpredictable events in later development. Based on these theoretical considerations and empirical findings, a proposed model for anxiety development in children with AD/HD (see Figure 2) illustrates the hypotheses of this study.

In accordance with the previously reviewed literature and the proposed model, the hypotheses of this study were as follows.

Hypothesis 1: In the domain of parenting practices, it was predicted that inconsistent parenting practices would be associated with increased anxiety in children with AD/HD. Initial evidence suggests that parents of children with AD/HD use more inconsistent parenting practices than parents of healthy control children (Ellis & Nigg, 2009); however, inconsistent parenting has not been examined as a risk factor for anxiety in children with AD/HD. To the extent that inconsistent parenting may expose children to events that they experience as unpredictable, children who experience more inconsistent parenting may experience more anxiety.

17

Hypothesis 2: In the domain of parental psychopathology, it was predicted that parental AD/HD symptoms would account for unique variance in child anxiety when parental anxiety and depression are accounted for. A plethora of past research has found an association between parental anxiety and depression and child anxiety; however, the role of parental AD/HD is of particular interest since many parents of children with AD/HD also have AD/HD. Some research suggests that parents who have AD/HD are less consistent disciplinarians (Chronis-Tuscano et al., 2008; Murray & Johnston, 2006); therefore, parental AD/HD symptoms may impact child anxiety indirectly through an association with inconsistent parenting practices.

Hypothesis 3: In the past, locus of control has been hypothesized to mediate the relationship between parenting and anxiety in early development, and to moderate the relationship between parenting and anxiety in later development (Chorpita & Barlow, 1998). While past research has shown that locus of control is a predictor of anxiety in children with AD/HD, locus of control has not been explored as a mediator or moderator in past research on anxiety in children with AD/HD (McGee, 2007). In this study of children who are in late childhood and early adolescence, locus of control was hypothesized to moderate the relationship between inconsistent parenting practices and child anxiety.

Hypothesis 4: In order to shed further light on the previous mixed findings regarding the timing of the onset of AD/HD and symptoms of anxiety, the present study collected retrospective reports from parents about the ages of onset of AD/HD, GAD, and SP (as applicable for each child). It was hypothesized that parents would report that

18

symptoms of AD/HD were present at an earlier age than symptoms of either anxiety

disorder.

CHAPTER III

METHODS

Participants

The participants for the study included a clinical sample of children with AD/HD. Inclusion criteria for the study were as follows: the child must meet research criteria for AD/HD (any subtype), be between the ages of 8 and 15 (inclusive), and have at least one parent or guardian who is willing to participate. In order to meet research criteria for AD/HD, the child must display at least six of nine symptoms of inattention and/or six of nine symptoms of hyperactivity-impulsivity on the AD/HD module of the Computerized Diagnostic Interview Schedule for Children—IV (C-DISC-IV), the ADHD Rating Scale (ADHD-RS), or the Conners Comprehensive Behavior Rating Scale (CBRS). These symptoms must also be developmentally deviant (t = 65 or 94th percentile) based on parent ratings on the ADHD-RS or the Conners Comprehensive Behavior Rating Scales (CBRS). When the ADHD-RS or CBRS was used to establish the symptom count, the other rating scale was used to establish developmental deviance.

Participants were recruited from the AD/HD Clinic at UNCG and various private practices, schools, and community organizations. A total of 30 participants completed all study procedures. Of these participants, 4 did not meet the research study criteria for AD/HD. The C-DISC-IV, ADHD-RS, and CBRS scores for these four participants were reviewed, and the data for two of these participants were included in the subsequent data

analysis. One of these participants had received a clinical diagnosis of AD/HD at the AD/HD Clinic at UNCG and authorized the researcher to view the ADHD-RS and CBRS ratings from their clinical chart. This participant met the symptom count and developmental deviance criteria based on the ADHD-RS and CBRS ratings provided by the child's father and the child's teachers. The second participant met the symptom count criterion for inattention but was at the 90th percentile for ADHD-RS inattention severity. The remaining two participants were excluded from further analyses. An additional eight families expressed serious interest in participating in the study but did not come to the scheduled research visit; these families were re-contacted and were either unable to be contacted by phone or decided that they were no longer interested in participation.

A summary of the sample can be found in Table 1. Child participants ranged in age from 8.08 years to 15.50 years ($M = 10.56$, $SD = 3.53$) and ranged in grade from 2nd to 9th ($M = 4.64$, $SD = 1.85$). Seventy-one percent ($n = 20$) of the participants were male. Parents were asked to report on their child's race/ethnicity; 71.43% ($n = 20$) of participants were Caucasian, 10.71% ($n = 3$) of participants were African-American, 10.71% ($n = 3$) of participants were multiracial, 3.57% ($n = 1$) were Asian-American or Asian, and 3.57% ($n = 1$) did not report race or ethnicity. Eighty-five-point-seven-one percent ($n = 24$) of the participants lived with both parents, and 14.29% ($n = 4$) of the participants lived with their mother only. According to mother report, 82.14% ($n = 23$) of the participants were taking at least one medication for the management of AD/HD symptoms. Of the total sample, 75.00% ($n = 21$) of participants were taking stimulant medication and 21.43% ($n = 6$) were taking non-stimulant medication for AD/HD;

14.29% ($n = 4$) of the sample were taking both stimulant and non-stimulant medication for AD/HD.

When applicable, both parents were invited and encouraged to participate in the study; however, only one parent was required to participate. Mothers participated with each of the child participants. The mothers in the sample ranged in age from 27 years to 62 years ($M = 42.32$ years; $SD = 7.05$ years). Overall, the educational status of the mothers indicated that this was a highly educated parent sample. In terms of educational level, 39.29% ($n = 11$) of the mothers had completed college and 28.57% ($n = 8$) of the mothers had completed a graduate degree; 7.14% ($n = 2$) of the mothers did not complete high school, 7.14% ($n = 2$) completed high school, and 17.86% ($n = 5$) completed some college or an associate's degree. Seven fathers also participated in the project. These fathers ranged in age from 35 to 49 ($M = 43.29$, $SD = 5.22$). In terms of educational level, 42.86% ($n = 3$) of the fathers reported that they had completed college, 28.57% ($n = 2$) of the fathers reported that they had completed graduate degrees, and 28.57% ($n = 2$) of the fathers reported that they had completed some college.

Materials

AD/HD Diagnostic Status

Computerized Diagnostic Interview Schedule for Children-IV (*C-DISC-IV*; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). The C-DISC-IV is a psychometrically sound structured diagnostic interview that assesses *DSM-IV* Axis I disorders in children. It is administered in a computerized format to parents or children, and answers are given in a yes/no format. For the purposes of this study, the clinician-

administered version was conducted with mothers. Each question was aloud to the mother as it was presented on the computer, and the exact response that the mother provided was entered. Parental responses to the AD/HD module were used to assess the presence of AD/HD.

Conners Comprehensive Behavior Rating Scale (*CBRS*; Conners, 2008). The CBRS is a broad-band rating scale system of children's behavior. It includes parent and teacher report forms for children and adolescents ages 6-18, and self-report forms for children and adolescents ages 8-18. The parent report form has 203 items, the teacher report form has 204 items, and the self-report form has 179 items. The measure consists of 28 scales, including several scales that correspond to DSM-IV disorders and symptom counts. Of particular interest to this project were the ADHD hyperactive-impulsive and ADHD inattentive subscales. The generalized anxiety disorder, social phobia, and separation anxiety disorder subscales were also used for post-hoc analyses. The Conners CBRS has been shown to have good reliability and validity. In the present study, the parent form (CBRS-P) was administered to mothers. The symptom counts and t-scores on the parent-rated AD/HD inattentive and AD/HD hyperactive-impulsive subscales were used to establish the symptom count and developmental deviance of these symptoms in relation to the diagnosis of AD/HD.

ADHD Rating Scale (*ADHD-RS*; DuPaul et al., 1998). The ADHD-RS is a narrow-band rating scale of the symptoms of AD/HD that is based on the *DSM-IV* criteria for the disorder. It is appropriate for use with children and adolescents age 5 to 17. Each of the 18 items is rated on a four-point scale ranging from 0 (never or rarely a problem) to

23

3 (very often a problem). The odd-numbered items correspond to the inattention scale, and the even-numbered items correspond to the hyperactive-impulsive scale. Each scale yields both a symptom count (number of items scored at a 2 or 3) ranging from 0 to 9 and a severity scale (with higher numbers indicating higher severity) ranging from 0 to 27. The ADHD-RS has been shown to have good reliability and validity (DuPaul et al., 1998). The ADHD-RS was administered to mothers in this study. The symptom counts and severity percentiles on this measure were used to assess the symptom count and developmental deviance of inattentive and hyperactive-impulsive behaviors in relation to the diagnosis of AD/HD.

Outcome Variables

Multidimensional Anxiety Scale for Children (*MASC*; March, Parker, Sullivan, & Stallings, 1997). The MASC is a measure of a broad range of anxiety symptoms that is designed for use with children ages 8 to 17. The informant rates each of the 39 items on a four-point scale (never, rarely, sometimes, or often). It contains four factors, three of which contain two sub-factors each: physical symptoms (tense/restless and somatic/autonomic), social anxiety (humiliation/rejection and public performance fears), harm avoidance (perfectionism and anxious coping), and separation anxiety. A confirmatory factor analysis using a sample of children with AD/HD replicated these factors, demonstrating that this measure is a valid measure of anxiety in this population (March et al., 1999). It has good reliability and validity (March et al., 1997). Although norms exist only for the child-report version of the MASC, a study of the MASC in a community sample demonstrated that the MASC parent-report (MASC-P) is reliable and

has the same factor structure as the MASC child self-report (MASC-C) (Baldwin &

Dadds, 2007). The MASC-C t-scores and MASC-P raw scores were used in this study to

assess dimensional symptoms of child anxiety.

Computerized Diagnostic Interview Schedule for Children-IV (*C-DISC-IV)*. The

GAD and SP modules were administered to mothers.

Predictor Variables

Inconsistent parenting was assessed with the Parenting Alliance Inventory (PAI)

and the inconsistent discipline subscale of the Alabama Parenting Questionnaire (APQ).

These are commonly used scales in the study of parenting.

Alabama Parenting Questionnaire (*APQ*; Shelton, Frick, & Wootton, 1996). The

APQ is a measure of parenting practices, appropriate for use with parents of children who

are between the ages of six and thirteen. The parent rates each of the 42 items on a five-

point scale (occurs never, occasionally, sometimes, almost always, or always). The APQ

has five scales, including involvement, positive parenting, poor monitoring/supervision,

inconsistent discipline, and corporal punishment. It has been found to have adequate

reliability and validity (Shelton et al., 1996). The APQ was administered to mothers in

this study and the z-score of the inconsistent discipline scale was used as a measure of

inconsistent parenting practices.

Parenting Alliance Inventory (*PAI;* Abidin & Brunner, 1995). The PAI is a

narrow-band rating scale of the degree to which a parent believes that he/she has a sound

working relationship with the child's other parent. Each of the 20 items is rated on a five-

point scale ranging from 1 (strongly disagree) to 5 (strongly agree). The item scores are

summed to calculate a total score ranging from 20 to 100. The PAI has been shown to have good reliability and validity and correlates with other established measures of marital adjustment, parenting style, and parenting stress (Abidin & Brunner, 1995). The PAI was administered to mothers in this study as a measure of inter-parent parenting consistency.

Parental symptoms of anxiety, depression, and AD/HD were assessed via narrow-band measures of psychopathology.

Conners' Adult ADHD Rating Scales (*CAARS*; Conners et al., 1999). The CAARS is a narrow-band measure of adult AD/HD symptoms. It is appropriate for use with adults age 18 to 81 years. Each of the 42 items is rated on a four-point scale from 0 (not at all, never) to 3 (very much, very frequently). A factor analysis showed that the scale has four factors, covering both the core symptoms of AD/HD as well as associated features: inattention/cognitive problems, hyperactivity/restlessness, impulsivity/emotional lability, and problems with self-concept (Conners et al., 1999). The scale has been shown to have good reliability and validity, and high sensitivity and specificity (Erhardt, Epstein, Conners, Parker, & Sitarenios, 1999). The CAARS was administered to mothers and fathers in this study, and the DSM-IV AD/HD Symptoms subscale was used as a measure of parental AD/HD symptoms.

Beck Anxiety Inventory (*BAI*; Beck, Epstein, Brown, & Steer, 1988). The BAI is a narrow-band measure of anxiety designed for use with adults. Each of the 21 items is rated on a four-point scale ranging from 0 (not at all) to 3 (severely), and the items are summed for a total score ranging from 0 to 63. A higher score represents a greater

severity of anxiety symptoms. It has been shown to have good reliability and validity and it is only moderately correlated with measures of depression, showing that it can discriminate between depression and anxiety (Beck et al., 1988). It has also been shown to correlate with physiological measures of anxiety (Borden, Peterson, & Jackson, 1991). The BAI was administered to mothers and fathers in this study as a measure of parental anxiety symptoms.

Beck Depression Inventory (*BDI*; Beck, Steer, & Garbin, 1988). The BDI is a narrow-band measure of depression designed for use with adults. Each of the 21 items is rated on a four-point scale ranging from 0 (not at all) to 3 (severely), and the items are summed for a total score ranging from 0 to 63. A higher score represents a greater severity of depression symptoms. It has been shown to have good reliability and validity, and it is only moderately correlated with measures of anxiety (Beck et al., 1988). The BDI was administered to mothers and fathers in this study as a measure of parental depression symptoms.

As noted previously, locus of control is considered an important construct in understanding child anxiety. Locus of control is commonly assessed in children using the Nowicki-Strickland Locus of Control Scale (NSLOC).

Nowicki-Strickland Locus of Control Scale (*NSLOC*; Nowicki & Strickland, 1973). The NSLOC is a measure of locus of control that is appropriate for children ages 8-17. The responses to each of the 40 items are given in a yes/no format. Responses are scored as a 0 or 1 and these scores are summed for a total score ranging from 0 to 40. Higher scores on the scale represent a more external locus of control. The NSLOC has

27

adequate reliability and validity (Nowicki & Strickland, 1973). The NSLOC was administered to children in this study as a measure of locus of control.

Procedure

The UNCG Institutional Review Board (IRB) approved this study before recruitment began. Participants were recruited in several ways. The student researcher contacted past clients of the AD/HD Clinic at UNCG who met inclusion criteria in order to invite them to participate. Participants were also recruited from community organizations such as the AD/HD Parent Support Group, Greensboro Day School, and the Hill Center. The student researcher also made contact with six clinicians in the community in order to share information about the study and facilitate recruitment. An advertisement for this study as well as three others taking place at the AD/HD Clinic at UNCG appeared in the January 2011 and February 2011 issues of Piedmont Parent magazine. Of the final sample included in the analyses, 35.7% ($n = 10$) of the participants were recruited from the AD/HD Parent Support Group, 32.1% ($n = 9$) were recruited from the AD/HD Clinic at UNCG, 25.0% ($n = 7$) had responded to the Piedmont Parent advertisement, and 7.1% ($n = 2$) were from other or unknown recruitment sources.

Study participation was completed in one or two visits at the AD/HD Clinic at UNCG or other mutually convenient location. The student researcher explained all study procedures to the child and parents and obtained written child assent and parent consent for participation in the study before completing any other study procedures (see Appendices A-C). Participants who had previously completed an evaluation at the AD/HD Clinic in the six months prior to study participation were asked to sign a release

allowing the student researcher to access the ADHD-RS, CBRS, and C-DISC-IV from the evaluation so that these measures did not need to be repeated. Parents were given a photocopy of the signed assent, consent, and release forms. A summary of the measures that participants completed can be found in Appendix A. Children completed the NSLOC and MASC-C. Mothers completed a demographic information form, the DISC-IV AD/HD and GAD modules, CBRS, ADHD-RS, PAI, APQ, CAARS, BAI, BDI, and MASC-P. As many of the child participants were taking medications for the management of AD/HD, mothers were instructed to complete all ratings of their child's behavior based on the child's behavior when he/she is not taking medication. Participating fathers completed the BAI, BDI, and CAARS. For families who had already undergone an evaluation at the AD/HD Clinic, completion of the study procedures typically took approximately 45 minutes. For participants who had not received an evaluation at the AD/HD Clinic in the six months prior to study participation, completion of the study procedures typically took one-and-a-half to two hours. Upon completion of the study procedures, families received a $10 gift card. The student researcher also provided each family with a feedback letter that summarized the results of some of the measures that they completed as part of participation in the study (see Appendix E).

Administrations of the C-DISC-IV (including those completed during AD/HD Clinic evaluations and as part of this study) were completed by graduate students under the supervision of Dr. Arthur Anastopoulos, a licensed psychologist who is certified in the administration of the DISC-IV. The student researcher received training in the administration of the C-DISC-IV from Dr. Anastopoulos prior to administering this

interview to study participants. The student researcher administered the C-DISC-IV for

all participants who had not previously completed this interview as part of an evaluation

at the AD/HD Clinic.

CHAPTER IV

RESULTS

Preliminary Inspection of the Data

An examination of the data revealed that three variables violated the assumption

of normality: mother BAI score, father BAI score, and father CAARS score. Mother and

father BAI scores were log transformed, resulting in normally distributed variables. The

father CAARS score was unable to be transformed due to the severely non-normal

distribution of this data as well as the small number of fathers participating. Final skew

statistics for all predictor and outcome variables ranged from -.86 to 1.00; final kurtosis

statistics for all variables, except father CAARS score, ranged from -1.08 to .40. All data

to be entered in the regression analyses, except for father CAARS score, fulfilled the

assumption of normality.

Description of the Sample

Descriptive statistics for the AD/HD status, predictor, and outcome variables

appear in Table 2. In terms of number of AD/HD symptoms, parents endorsed an average

of 12.54 total symptoms ($SD = 4.33$), which is consistent with expectations for a clinical

sample as a minimum of six symptoms are required for a diagnosis of AD/HD. On the

ADHD-RS, parents tended to endorse a higher number of symptoms of inattention ($M =$

7.18, $SD = 2.26$) than hyperactivity-impulsivity ($M = 5.36$, $SD = 2.98$); a paired-samples

t-test indicates that this difference is significant ($t = 3.15$, $df = 27$, $p < .01$). Parents also

tended to rate their children as more severely inattentive ($M = 19.43$, $SD = 5.29$) than hyperactive-impulsive ($M = 15.39$, $SD = 7.73$); this difference in the severity ratings was also significant ($t = 3.04$, $df = 27$, $p < .05$). On the CBRS, parents tended to rate inattentive and hyperactive-impulsive symptoms at similar severities ($t = 1.75$, $df = 26$, $p = ns$).

In terms of the predictor variables, the average mother and father BAI scores were within the "not clinically significant" range prior to the log transformations of these variables ($M_{mother} = 8.65$, $SD = 9.24$; $M_{father} = 5.71$, $SD = 6.92$). The average mother and father BDI scores were reflective of mild to moderate depression ($M_{mother} = 11.64$, $SD = 6.63$; $M_{father} = 8.29$, $SD = 7.39$). The mean t-score for mothers on the CAARS was 47.80 ($SD = 11.79$), which is within the normal range; however, the mean t-score for fathers on the CAARS was 65.50 ($SD = 13.25$), which is reflective of a clinically significant elevation in symptoms of AD/HD. The mean APQ inconsistent parenting subscale z-score was -.16 ($SD = .96$), which is reflective of an average amount of inconsistent parenting. The mean PAI raw score was 74.52 ($SD = 19.51$).

In terms of the outcome variables, a wide range of scores was observed on the dimensional measures of anxiety (MASC-C and MASC-P). The mean MASC-C t-score was 52.21 ($SD = 12.60$), which is within the normal range, and the mean MASC-P raw score was 50.25 ($SD = 15.34$). However, less variance was observed on the categorical outcome variables (C-DISC-IV GAD and SP diagnoses); zero percent of the sample met criteria for C-DISC-IV GAD, and only 14.29% (n = 4) of the sample met C-DISC-IV criteria for SP.

Correlations among Variables

Correlations among the variables were conducted and are displayed in Table 3. Several correlations were significant. Higher ADHD-RS inattention severity scores were associated with higher ADHD-RS hyperactivity-impulsivity severity scores ($r = .47$, $n = 28$, $p < .05$). Higher ADHD-RS hyperactivity-impulsivity severity scores were also associated with increased use of inconsistent parenting strategies on the APQ ($r = .60$, $n = 28$, $p < .01$). Maternal BAI and BDI scores were correlated ($r = .67$, $n = 26$, $p < .01$), as were paternal self-reported anxiety and depression ($r = .95$, $n = 7$, $p < .01$). Older child age was associated with higher father BAI scores ($r = .78$, $n = 7$, $p < .05$) and with higher father BDI scores ($r = .80$, $n = 7$, $p < .05$).

Several correlations also approached significance. Increased child age was associated with decreased ADHD-RS inattention severity ($r = -.34$, $n = 28$, $p = .08$). Child sex was associated with MASC-C score ($r = -.33$, $n = 28$, $p = .09$), such that increased child anxiety was associated with being female. Higher father BDI score was associated with MASC-C scores ($r = .672$, $n = 7$, $p = .10$).

Predicting Child Anxiety

Due to the small number of fathers participating in the study and the fact that the paternal psychopathology variables were not significantly correlated with the outcome variables, only mother BAI, BDI, and CAARS scores were used in the following primary analyses.

To test the first hypothesis that inconsistent parenting practices would be associated with anxiety in children with AD/HD, two stepwise regressions were

conducted with MASC-C and MASC-P total scores as outcome variables. Child age and sex were entered in the first step of the regression as covariates, ADHD-RS inattention severity and ADHD-RS hyperactivity-impulsivity severity were entered in the second step, and APQ inconsistent parenting and PAI scores were entered in the third step. No variables entered into the equation in either of these regressions. A logistic regression was also conducted in order to determine whether inconsistent parenting practices predicted C-DISC-IV SP diagnosis. C-DISC-IV SP diagnosis was regressed on APQ inconsistent parenting and PAI scores; this regression was not significant (χ^2 = 3.23, df = 2, p = ns).

To test the second hypothesis that parental symptoms of AD/HD would account for unique variance in child anxiety over and above the variance accounted for by parental anxiety and depression, two stepwise regressions were conducted with MASC-C and MASC-P total scores as outcome variables. Child age and sex were entered in the first step of the regression as covariates, ADHD-RS inattention severity and ADHD-RS hyperactivity-impulsivity severity were entered in the second step, mother BAI and BDI scores were entered in the third step, and mother CAARS scores were entered in the fourth step. No variables entered into the equation in either of these regressions. A logistic regression was also conducted in order to determine whether maternal psychopathology predicted C-DISC-IV SP diagnosis. C-DISC-IV SP diagnosis was regressed on mother BAI, BDI, and CAARS scores; the overall model was significant (χ^2 = 12.59, df = 3, p < .01). The regression coefficients for this model can be found in Table 4. The coefficient for maternal CAARS score approached significance (β = .22, SE = .13,

$p = .08$), indicating that higher maternal symptoms of AD/HD are associated with a child diagnosis of SP.

In order to test the third hypothesis that locus of control would moderate the relation between inconsistent parenting and child anxiety, the APQ, PAI, and NSLOC variables were centered. The centered variables were multiplied to create the APQ x NSLOC and PAI x NSLOC interaction terms. Stepwise multiple regressions were conducted in which the MASC-C and MASC-P variables were regressed on APQ, NSLOC, and APQ x NSLOC and on PAI, NSLOC, and PAI x NSLOC. These regressions were not significant. Logistic regressions were also conducted in which C-DISC-IV SP status was also regressed on these parenting predictors. These regressions were also not significant.

Age of Onset for AD/HD and Anxiety Disorders

As mentioned previously, only four participants met C-DISC-IV criteria for SP, and no participants met C-DISC-IV criteria for GAD. Formal statistical comparisons of the age of onset for AD/HD and SP were not appropriate due to the small number of participants meeting C-DISC-IV criteria for both disorders. Three parents reported that their children's symptoms of AD/HD and SP had emerged at the same age; the fourth parent reported that the symptoms of SP had emerged prior to the symptoms of AD/HD.

Post-Hoc Analyses

Due to the small number of participants that met C-DISC-IV criteria for GAD or SP, the C-DISC-IV intermediate diagnoses and symptom counts were used in post-hoc analyses. Data for 25 participants were included in these analyses, due to missing data for

three participants. Fifty-two percent ($n = 13$) of participants met C-DISC-IV criteria for a full or intermediate diagnosis of SP, and 24.00% ($n = 6$) of participants met C-DISC-IV criteria for a full or intermediate diagnosis of GAD. Logistic regressions were conducted in which C-DISC-IV GAD diagnosis (defined as a full or intermediate diagnosis of GAD) and C-DISC-IV SP diagnosis (defined as a full or intermediate diagnosis of SP) were regressed on the parenting variables and on the maternal psychopathology variables (for a total of four logistic regressions). None of these logistic regressions were significant. The C-DISC-IV symptom counts for GAD and SP were also examined; for GAD, a participant may display between zero and twelve symptoms, and for SP, a participant may display between zero and thirteen symptoms. The symptom counts for GAD and SP were regressed on the parenting variables and on the maternal psychopathology variables (for a total of four logistic regressions). The regression of GAD symptoms on the parental psychopathology variables was significant (F = 5.514, *df* = 3, *p* < .01). None of the other regressions were significant.

Post-hoc analyses were conducted in order to determine whether there were significant differences between the participants who met C-DISC-IV criteria for both AD/HD and SP (AD/HD + SP group) and the other participants (AD/HD only group) with regard to the parenting or paternal psychopathology variables. First, the groups were compared on child age, sex, ADHD-RS inattention severity, and ADHD-RS hyperactivity-impulsivity severity. Participants in these groups did not differ with regard to age or ADHD-RS hyperactivity-impulsivity severity. For sex, Levene's Test for equality of variances indicated that the two groups did not have equal variances (*F* =

29.71, $p < .01$); a t-test for equality of means when equal variances was not assumed revealed a significant difference with regard to the sex composition of these two groups ($t = 3.39$, $df = 23.00$, $p < .01$). The AD/HD + SP group contained 100% ($N = 4$) female participants, whereas the AD/HD only group contained 33.33% ($N = 8$) female participants. For ADHD-RS inattention severity, Levene's Test for equality of variances indicated that the two groups did not have equal variances ($F = 5.76$, $p < .05$); a t-test for equality of means when equal variances was not assumed revealed a trend for significance with regard to the difference in ADHD-RS inattention severity scores for these two groups ($t = -1.83$, $df = 22.59$, $p < .05$). The AD/HD + SP group had more severe symptoms of inattention ($M = 21.50$, $SD = 1.29$) than the AD/HD only group ($M = 19.08$, $SD = 5.63$), although both of these mean scores are within the clinical range.

Next, in order to determine whether the AD/HD + SP group and the AD/HD only group differed on the parenting and maternal psychopathology variables, t-tests were conducted to compare these groups on maternal BAI, BDI and CAARS scores and on PAI and APQ inconsistent parenting practices scores. The t-test for maternal BAI score approached significance ($T = -1.99$, $df = 24$, $p = .06$) and indicated that mothers of children in the AD/HD + SP group reported significantly more symptoms of anxiety than mothers of children in the AD/HD only group. The t-test for maternal BDI score was not significant. The t-test for maternal CAARS score was significant ($T = -3.90$, $df = 23$, $p < .01$) and indicated that mothers of children in the AD/HD + SP group reported significantly more symptoms of AD/HD than mothers of children in the AD/HD only

group. The t-tests for the APQ inconsistent parenting practices subscale and the PAI were not significant.

Two multivariate multiple regressions were conducted in order to determine whether the parenting variables or maternal psychopathology variables were associated with MASC-C subscale scores (physical symptoms, harm avoidance, social phobia, and separation anxiety/panic). In the first multivariate multiple regression, the MASC-C subscale scores were regressed on the PAI and APQ inconsistent parenting practices scores; this multivariate multiple regression was not significant. In the second multivariate multiple regression, the MASC-C subscale scores were regressed on the maternal BAI, BDI, and CAARS scores. There was a trend for significance for maternal BAI score to predict the outcome variables ($\Lambda = .653$, $F = 2.261$, $df = 4$, $p = .11$). Neither maternal BDI score nor maternal CAARS score predicted the MASC-C subscale scores. Because these multivariate multiple regressions were not significant, univariate follow-up tests were not conducted.

Because the CBRS provides scores similar to the primary outcome variables of this study, additional analyses were conducted in order to determine the utility of these scores. In order to explore the association between the parenting variables, the maternal psychopathology variables, and the CBRS anxiety scores, correlations between the PAI, APQ inconsistent parenting scale, maternal BAI, maternal BDI, and maternal CAARS scores, and CBRS GAD, SP, and SAD subscale scores were calculated. There were no significant correlations between the parenting variables and the CBRS anxiety subscales. Increased maternal anxiety was associated with increased CBRS GAD score ($r = .409$, n

= 26, $p < .05$) and with increased CBRS SAD score ($r = .444$, $n = 26$, $p < .05$). There was a trend for significance for the association between CBRS SAD score and maternal BDI score ($r = .334$, $n = 27$, $p = .09$).

In order to determine whether the parenting variables or maternal psychopathology variables were associated with MASC-C scores or CBRS anxiety scores indicative of clinically significant anxiety, a series of logistic regressions was conducted. First, a new variable was created that indicated whether the MASC-C t-score was greater than or equal to 60 (coded as a 1) or less than 60 (coded as a 0). New variables were also created that indicated whether the CBRS GAD, SP, and SAD subscale t-scores were equal or greater than 65. The creation of these new variables indicated that 26.67% ($n = 8$) of the sample had a t-score of greater than 60 on the MASC-C, 81.48% ($n = 22$) had a t-score of greater than 65 on the CBRS GAD subscale, 62.96% ($n = 17$) had a t-score of greater than 65 on the CBRS SP subscale, and 51.85% ($n = 14$) had a t-score of greater than 65 on the CBRS SAD subscale. The logistic regression of the new CBRS SP variable on maternal BAI, BDI, and CAARS scores was significant ($\chi^2 = 10.22$, $df = 3$, $p < .05$). The regression coefficients for this regression analysis can be found in Table 5. The coefficient for maternal CAARS score approached significance ($\beta = .22$, $SE = .12$, $p = .06$) and indicates that increased maternal symptoms of AD/HD are associated with higher parent-reported ratings of child social phobia. None of the other logistic regressions were significant.

In order to explore whether other aspects of parenting were associated with anxiety in this sample, the APQ subscales and total score were correlated with the

MASC-C and MASC-P total scores. The APQ monitoring subscale was significantly

correlated with the MASC-C total t-score ($r = .527$, $n = 28$, $p < .01$), indicating that poor

monitoring/supervision was associated with child anxiety. The APQ total score was also

significantly correlated with the MASC-C total t-score ($r = .416$, $n = 28$, $p < .05$).

Multiple regressions, multivariate multiple regressions, and logistic regressions were

conducted in order to determine whether the APQ monitoring subscale or the APQ total

score, in combination with the PAI, predicted MASC-C total score, MASC-P total score,

MASC-C subscale scores, or C-DISC-IV SP diagnosis; none of these regressions were

significant.

CHAPTER V

DISCUSSION

It is well-documented that children with AD/HD are at increased risk for the

development of anxiety. Some have estimated that around 25% of children with AD/HD

have an anxiety disorder (Tannock, 2000), and a large meta-analysis found that children

with AD/HD have a three times greater risk of developing an anxiety disorder than

children in the general population (Angold et al., 1999). Although this comorbidity is

well-documented, the reasons for this overlap are not as well-understood. With regard to

the timing of AD/HD and anxiety, AD/HD frequently develops first and is followed by

the onset of anxiety in later development; some have referred to this sequence as a "post-

morbidity" (Taurines, 2010). This developmental sequence suggests that having AD/HD

may put children at increased risk for having anxiety. Some research has begun to

identify potential risk factors that may increase the likelihood of developing anxiety in

children with AD/HD; these potential risk factors have included negative or controlling

parenting practices, parental psychopathology, child externalizing symptoms, and child

educational difficulties (Acosta et al., 2008; Baldwin & Dadds, 2008; Kepley &

Ostrander, 2007; McGee, 2007; Moulton, 2006).

The purpose of this study was to further examine the role of parenting practices

and parental psychopathology in predicting anxiety in a sample of children with AD/HD.

Study hypotheses were guided by a model of anxiety development in children with

AD/HD that was informed by Chorpita and Barlow's (1998) model of anxiety development. Specifically, this study hypothesized that inconsistent parenting practices and parental psychopathology would predict symptoms of anxiety in children with AD/HD. The study also hypothesized that locus of control would moderate the relation between parenting practices and anxiety, and that parents would report that their children's symptoms of AD/HD emerged before their symptoms of anxiety.

Summary of Results

Planned analyses revealed initial and tentative support for the role of maternal psychopathology as a predictor of child anxiety in this sample of children with AD/HD. A logistic regression supported the hypothesis that maternal symptoms of anxiety, depression, and AD/HD would predict C-DISC-IV SP diagnosis; the regression coefficient for maternal symptoms of AD/HD approached significance, indicating that maternal symptoms of AD/HD were most highly associated with C-DISC-IV SP status. However, when the C-DISC-IV SP diagnosis was expanded to include a full or intermediate C-DISC-IV diagnosis of SP for a post-hoc analysis, the regression of C-DISC-IV SP on the maternal psychopathology variables was no longer significant. This finding points to the instability of these results in this small sample. These maternal psychopathology variables did not significantly predict dimensional measures of child anxiety. Measures of inter-parent consistency and inconsistent discipline did not significantly predict C-DISC-IV SP diagnosis or symptoms of child anxiety. Further, the hypothesis regarding the moderating role of child locus of control in the relation between inconsistent parenting and child anxiety was not supported. With regard to the hypothesis

regarding the ages of onset for AD/HD and anxiety, only a small number ($n = 4$) met full C-DISC-IV criteria for both AD/HD and an anxiety disorder. Of these participants, four parents reported that their child's symptoms of AD/HD and anxiety had emerged at the same age, and one parent reported that her child's symptoms of anxiety had emerged before symptoms of AD/HD.

A number of post-hoc analyses were conducted in order to further test the a priori hypotheses as well as several other possible predictors of child anxiety in this sample. Several of these analyses provided additional initial support for the hypothesis that maternal psychopathology would predict child anxiety in this sample. A comparison of the subset of the sample that met criteria for both AD/HD and SP and those who met criteria for AD/HD only indicated that mothers of children in the AD/HD + SP group had significantly more symptoms of AD/HD. The maternal psychopathology variables also significantly predicted clinically significant symptoms of SP on the CBRS, and the regression coefficient for maternal symptoms of AD/HD approached significance. Increased maternal anxiety was also significantly correlated with the CBRS GAD score and the CBRS SAD score. There was also a trend for significance for maternal depression to predict the MASC-C subscale scores (physical symptoms, harm avoidance, social anxiety, and separation/panic) in a multivariate multiple regression. There was also a trend for maternal depression to predict the CBRS SAD score. The hypothesis that inter-parent inconsistency and inconsistent discipline would predict child anxiety in this sample was not supported in the post-hoc analyses. However, a different dimension of

parenting, decreased parental monitoring and supervision, was found to correlate significantly with child-reported symptoms of anxiety.

In summary, the findings of this study did not support Hypothesis 1 that inconsistent parenting practices would predict child anxiety in children with AD/HD. The findings of this study tentatively supported Hypothesis 2 that maternal psychopathology would predict child anxiety, and maternal symptoms of AD/HD were significantly associated with child anxiety in some, but not all, planned and post-hoc analyses. Hypothesis 3, that child locus of control would moderate the relation between inconsistent parenting and child anxiety, was not supported. Lastly, Hypothesis 4 that parents would report that symptoms of AD/HD emerged before symptoms of anxiety was not supported; however, very few participants in this sample met criteria for both AD/HD and an anxiety disorder.

Implications

The findings of this study have implications for theory, research, and clinical practice. With regard to the theoretical model proposed by this study, the findings provided initial support for Hypothesis 2. Overall, the finding that maternal psychopathology predicts anxiety in children with AD/HD is consistent with past research (Kepley & Ostrander, 2007; McGee, 2007; Moulton, 2006). However, the specific finding that maternal symptoms of AD/HD are associated with child anxiety is a new contribution to this literature. Parental anxiety and depression have been firmly established as correlates of child anxiety in children with and without AD/HD (Flannery-Schroeder, 2004), but parental AD/HD has not been previously examined as a risk factor

44

for anxiety in children. Considering that AD/HD is conceptualized as having a genetic basis (Barkley, 2006a), parental AD/HD may be a particularly salient potential risk factor for anxiety in families of children with AD/HD. Inconsistent parenting was not supported as a predictor of anxiety in this sample, and this finding has several possible explanations. It is possible that children do not perceive inconsistent parenting to be an uncontrollable stimulus, that other dimensions of parenting that have been established as correlates of child anxiety (such as overprotectiveness or overcontrolling parenting) explain more variance in child anxiety, or that this analysis did not have sufficient power to detect the effect of inconsistent parenting.

The results of this study suggest several areas for continued research. First, continued research on the role of maternal psychopathology as a predictor of anxiety in children with AD/HD is needed; in particular, this research should utilize a larger sample of children and attempt to recruit larger numbers of children with clinically-significant anxiety. The role of paternal anxiety, depression, and AD/HD as predictors of anxiety in children with AD/HD is still unclear; future research should examine whether parental psychopathology contributes to an understanding of anxiety in children with AD/HD. It is also important to examine the mechanisms by which parental psychopathology may influence the development of anxiety in children with AD/HD and to determine whether these mechanisms are unique to children with AD/HD and their families or whether these mechanisms may generalize to a broader sample of children. In this study, parental psychopathology was hypothesized to predict child anxiety through an association with inconsistent parenting; however, there was no association between inconsistent parenting

and child anxiety in this study. Longitudinal research on the risk factors for anxiety in children with AD/HD may be useful in establishing causal links between parental psychopathology and anxiety in children with AD/HD, and may also be useful for examining potential mechanisms that help to explain the relation between parental psychopathology and anxiety in children with AD/HD.

The finding that maternal AD/HD predicts anxiety in children with AD/HD, if replicated in future research, may have implications for the assessment and treatment of children with AD/HD and their families. This finding suggests a more important role for parental psychopathology in the assessment, case conceptualization, and treatment planning for children with AD/HD; for example, assessment of parental psychopathology may be an important part of a comprehensive psychological assessment of a child suspected of having AD/HD. Psychosocial treatment of children with AD/HD frequently involves parent training; this finding suggests that acknowledging or addressing parental psychopathology during such parent training may enhance the ability of parent training to help protect children with AD/HD against anxiety or possibly other comorbid disorders. Family interventions for children and parents that both have anxiety have been shown to be effective (Cobham, Dadds, & Spence, 1998), but similar interventions have yet to be developed for children and parents that both have AD/HD. If further research establishes a mechanism by which parental psychopathology influences the development of anxiety in children with AD/HD, it may also be important to address those factors clinically.

Lastly, this study used several measures of child anxiety and raises issues about the utility of the MASC, the CBRS anxiety subscales, and the C-DISC-IV in this

population. For example, a comparison of the rates of clinically significant anxiety obtained by using each measure varied widely: 26.67% ($n = 8$) of the sample had a t-score of greater than 60 on the MASC-C, 81.48% ($n = 22$) had a t-score of greater than 65 on the CBRS GAD subscale, 62.96% ($n = 17$) had a t-score of greater than 65 on the CBRS SP subscale, 51.85% ($n = 14$) had a t-score of greater than 65 on the CBRS SAD subscale, and 14.29% ($n = 4$) met C-DISC-IV SP criteria. Past research has demonstrated that there is only moderate agreement between parents and children regarding children's symptoms of anxiety (Greco & Morris, 2004), and the varying rates of clinically-significant anxiety obtained by each measure in this study is consistent with this past finding. For example, the child self-reported anxiety on the MASC-C was lower than the parent-reported child anxiety on the CBRS. However, even with regard to the two parent-reported measures, the CBRS and the C-DISC-IV, the rates of clinically significant anxiety reported varied widely. This finding suggests the importance of future research on the utility of the MASC (particularly the MASC-P), CBRS, and C-DISC-IV in the assessment of anxiety and also underscores the importance of a multi-informant, multi-measure assessment of child psychopathology.

Limitations

Although the results of this study are useful in beginning to understand the role of parental psychopathology as a predictor of anxiety in children with AD/HD, these findings must be interpreted in the context of the limitations of the study. One important limitation of this study was the cross-sectional design. While a longitudinal design would have been useful in establishing the temporal relationship between symptoms of AD/HD,

parental psychopathology and parenting practices, and child anxiety, a cross-sectional

design was most practical for the purposes of the current study. The correlational and

regression analyses employed in this study do not establish causality, but do help to

elucidate the associations between parental psychopathology, parenting practices, and

anxiety symptoms in children with AD/HD.

A second limitation of this study was the small sample size, which impacted

several of the analyses. The small sample size decreased the power of the study and may

have impacted the ability to detect small or medium effects in this sample; it is possible

that the hypotheses regarding inconsistent parenting and age of onset were not supported

because there was not sufficient power to test these hypotheses. Further, the hypothesis

that maternal psychopathology predicted child anxiety was supported in some but not all

of the analyses. For instance, the logistic regression of C-DISC-IV SP full diagnosis on

maternal psychopathology was significant, but the logistic regression of C-DISC-IV SP

diagnosis (expanded to also include intermediate diagnoses) was not significant. This

inconsistency calls the significant result into question, since the first logistic regression

described had a very uneven distribution of participants in the two outcome

classifications and the second logistic regression had a more even distribution of

participants in the two outcome classifications. Further research using a larger sample

size and in samples containing adequate numbers of participants with clinically-

significant anxiety is needed.

A third limitation of this study was that the rate of anxiety reported in this sample,

both on the MASC-C and the C-DISC-IV, were lower than would be expected based on

the reports in the literature (Tannock, 2000). It is uncertain why the rates of anxiety reported by participants in this study were lower than was expected. One possible explanation is that the majority (82.1%; $n = 23$) of participants in the project were taking at least one medication for the management of AD/HD symptoms. Some initial research has found that treatment with stimulant medication may serve as a protective factor against anxiety, depression, and disruptive behavior over time in children with AD/HD (Biederman, Monuteaux, Spencer, Wilens, & Faraone, 2009). Although it was not tracked formally, several parents also indicated on the C-DISC-IV that their child was receiving some form of psychosocial treatment for AD/HD or other difficulties. The high rate of medication treatment and the utilization of psychosocial treatment by some participants may have contributed to the lower-than-expected rates of anxiety.

Conclusion

Overall, this study provided initial support for the role of maternal psychopathology as a predictor of anxiety in children with AD/HD. In particular, higher maternal symptoms of AD/HD appear to be associated with child anxiety in children with AD/HD; parental symptoms of AD/HD were previously unexplored as a possible risk factor for anxiety. This finding has implications for the assessment and treatment of children with AD/HD. Further research should examine the possible mechanism by which maternal psychopathology may influence the development of anxiety in children with AD/HD.

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APPENDIX A: CONSENT TO ACT AS A HUMAN PARTICIPANT

UNIVERSITY OF NORTH CAROLINA AT GREENSBORO

CONSENT TO ACT AS A HUMAN PARTICIPANT

Project Title: Risk Factors for Anxiety in Children with AD/HD

Project Director: Sarah O'Rourke

Participant's Name: _____

What is the study about?

This is a research project. The aim of this research project is to learn why some children with Attention-Deficit/Hyperactivity Disorder (AD/HD) also have symptoms of anxiety.

Why are you asking me?

You are invited to participate in this project because you have a child who is between the ages of 8 and 15 years old and has AD/HD.

What will you ask me to do if I agree to be in the study?

If you agree to be in this study, you will be asked to complete several questionnaires about your child's symptoms of AD/HD and anxiety. You will also be asked to complete questionnaires about being a parent and about your own symptoms of AD/HD, anxiety, and depression. You will be asked to complete an interview about your child's symptoms of AD/HD and anxiety. It will take one to two hours to complete the study visit. Completion of the interview or questionnaires may make you feel uncomfortable. If you have any questions about the study procedures, you may contact the Project Director, Sarah O'Rourke, at (336) 346-3196 x. 704.

What are the dangers to me?

There is minimal risk associated with being in this study. The questionnaires and diagnostic interview ask about personal information, such as symptoms of AD/HD and anxiety. Answering these questions may make you feel uncomfortable. You may skip questions that you do not want to answer. Participation is completely voluntary.

If you have any concerns about your rights, how you are being treated or if you have questions, want more information or have suggestions, please contact Eric Allen in the Office of Research Compliance at UNCG at (336) 256-1482.Questions, concerns or complaints about this project or benefits or risks associated with being in this study can

be answered by Sarah O'Rourke who may be contacted at (336) 346-3196 x. 704 or Dr. Arthur D. Anastopoulos who may be contacted at (336) 346-3196 x. 303.

Are there any benefits to me for taking part in this research study?

You will receive a summary of the measures that you and your child completed. This summary is intended as a summary of research data. It is not a tool for making decisions about your or your child's mental health care. However, this summary information may be of some use to clinicians working with you or your child.

Are there any benefits to society as a result of me taking part in this research?

Increased knowledge about why some children with AD/HD also have anxiety may lead to improved treatments for children with AD/HD and their families.

Will I get paid for being in the study? Will it cost me anything?

If you and your child participate in this study, you will receive a $10 gift card as payment for your time. You will receive a $10 gift card even if you or your child discontinue your participation in the study or do not answer certain questions on the questionnaires or interview. There are no costs to you or your child for being in this study.

How will you keep my information confidential?

The information that you provide as part of this research study will be kept confidential. Your name will not be written on any of the questionnaires that you and your child complete. You and your child will be assigned a unique code, and this code will be used to identify you. Only the principal investigator and project director will have access to the key that links your name with your unique code. The questionnaires that you and your child complete will be stored in a locked file cabinet. Passwords will protect information that has been entered on the computer. You will not be identified by name when data from this project is published. All information obtained in this study is strictly confidential unless disclosure is required by law. The investigators are legally required to report incidence of child abuse, disabled adult abuse, gunshot/knife wounds, communicable diseases, and if you present an imminent danger to the health or safety of another or yourself.

During or after your involvement in this research project, you may become aware of other research projects being conducted in the AD/HD Clinic that may be of interest to you. One such project currently underway is *Assessing Problematic Behaviors and Emotional Reactions in Adolescents*. This study uses some of the same behavioral data collection procedures. Should you decide to participate in this other project, behavioral data collected from this project can be shared with the other research project in order to spare you the trouble of having to repeat the same data gathering procedures. Only the

behavioral data common to each project will be shared, and data will only be shared with projects for which you have given written consent.

What if I want to leave the study?

You have the right to refuse to participate or to withdraw at any time, without penalty. If you do withdraw, it will not affect you in any way. If you choose to withdraw, you may request that any of your data that has been collected be destroyed unless it is in a de-identifiable state.

What about new information/changes in the study?

If significant new information relating to the study becomes available which may relate to your willingness to continue to participate, this information will be provided to you.

Voluntary Consent by Participant:

By signing this consent form you are agreeing that you read, or it has been read to you, and you fully understand the contents of this document and are openly willing consent to take part in this study. All of your questions concerning this study have been answered. By signing this form, you are agreeing that you are 18 years of age or older and are agreeing to participate, or have the individual specified above as a participant participate, in this study described to you bySarah O'Rourke.

Signature: _____ Date: _____

It would be ok to contact me in the future about similar research projects (please check one):

☐ Yes ☐ No

APPENDIX B: CONSENT FOR A MINOR TO ACT AS A HUMAN PARTICIPANT

UNIVERSITY OF NORTH CAROLINA AT GREENSBORO

CONSENT FOR A MINOR TO ACT AS A HUMAN PARTICIPANT

Project Title: Risk Factors for Anxiety in Children with AD/HD

Project Director: Sarah O'Rourke

Participant's Name: _____

What is the study about?

This is a research project. The aim of this research project is to learn why some children with Attention-Deficit/Hyperactivity Disorder (AD/HD) also have symptoms of anxiety.

Why are you asking my child?

Your child is invited to participate in this project because he or she is between the ages of 8 and 15 years old and has AD/HD.

What will you ask my child to do if I agree to let him or her be in the study?

If you agree to allow your child to participate in the study, he or she will be asked to complete two questionnaires. It will take less than one hour for your child to complete these questionnaires. Completion of the study procedures may cause mild discomfort. If you have any questions about the study procedures, you may contact the Project Director, Sarah O'Rourke, at (336) 346-3196 x. 704.

What are the dangers to my child?

There is minimal risk associated with being in this study. The questionnaires and diagnostic interview ask about personal information, such as symptoms of AD/HD and anxiety. Answering these questions may make you or your child feel uncomfortable. You or your child may skip questions that you do not want to answer. Participation is completely voluntary. Even if you participate and give permission for your child to participate, your child will still be asked if they want to participate in the project and his or her choice will be respected.

If you have any concerns about your rights, how you are being treated or if you have questions, want more information or have suggestions, please contact Eric Allen in the Office of Research Compliance at UNCG at (336) 256-1482.Questions, concerns or complaints about this project or benefits or risks associated with being in this study can

be answered by Sarah O'Rourke who may be contacted at (336) 346-3196 x. 704 or Dr. Arthur D. Anastopoulos who may be contacted at (336) 346-3196 x. 303.

Are there any benefits to my child as a result of participation in this research study?

You will receive a summary of the measures that you and your child completed. This summary is intended as a summary of research data. It is not a tool for making decisions about your or your child's mental health care. However, this summary information may be of some use to clinicians working with you or your child.

Are there any benefits to society as a result of my child taking part in this research?

Increased knowledge about why some children with AD/HD also have anxiety may lead to improved treatments for children with AD/HD and their families.

Will my child get paid for being in the study? Will it cost me anything for my kid to be in this study?

If you and your child participate in this study, you will receive a $10 gift card as payment for your time. You will receive a $10 gift card even if you or your child discontinue your participation in the study or do not answer certain questions on the questionnaires or interview. There are no costs to you or your child for being in this study.

How will my child's information be kept confidential?

The information that you and your child provide as part of this research study will be kept confidential. Your name will not be written on any of the questionnaires that you and your child complete. You and your child will be assigned a unique code, and this code will be used to identify you. Only the principal investigator and project director will have access to the key that links your name with your unique code. The questionnaires that you and your child complete will be stored in a locked file cabinet. Passwords will protect information that has been entered on the computer. You will not be identified by name when data from this project is published. All information obtained in this study is strictly confidential unless disclosure is required by law. The investigator is legally required to report incidence of child abuse, disabled adult abuse, gunshot/knife wounds, communicable diseases, and if your child presents an imminent danger to the health or safety of another or himself/herself.

During or after your involvement in this research project, you may become aware of other research projects being conducted in the AD/HD Clinic that may be of interest to you. One such project currently underway is *Assessing Problematic Behaviors and Emotional Reactions in Adolescents*. This study uses some of the same behavioral data collection procedures. Should you decide to participate in this other project, behavioral data collected from this project can be shared with the other research project in order to

spare you the trouble of having to repeat the same data gathering procedures. Only the behavioral data common to each project will be shared, and data will only be shared with projects for which you have given written consent.

What if my child wants to leave the study or I want him/her to leave the study?

You have the right to refuse to allow your child to participate or to withdraw him or her at any time, without penalty. If your child does withdraw, it will not affect you or your child in any way. If you or your child chooses to withdraw, you may request that any data that has been collected be destroyed unless it is in a de-identifiable state.

What about new information/changes in the study?

If significant new information relating to the study becomes available which may relate to your willingness allow your child to continue to participate, this information will be provided to you.

Voluntary Consent by Participant:

By signing this consent form, you are agreeing that you have read it or it has been read to you. You fully understand the contents of this document and consent to your child taking part in this study. All of your questions concerning this study have been answered. By signing this form, you are agreeing that you are the legal parent or guardian of the child who wishes to participate in this study described to you bySarah O'Rourke.

_____   Date: _____
Participant's Parent/Legal Guardian's Signature

_____   Date: _____
Participant's Parent/Legal Guardian's Signature

It would be ok to contact me in the future about similar research projects (please check one):

☐ Yes ☐ No

APPENDIX C: CHILD ASSENT FORM

UNIVERSITY OF NORTH CAROLINA AT GREENSBORO

CHILD ASSENT FORM

Study Title: Risk Factors for Anxiety in Children with AD/HD

My name is Sarah O'Rourke.

What is this about?

I would like to talk to you about why some kids with AD/HD also have symptoms of anxiety. I want to learn about your feelings and thoughts that have to do with anxiety.

Did my parents say it was ok?

Your parent(s) said it was ok for you to be in this study and have signed a form like this one. Your parent will be in the same building as you while you are participating in the study.

Why me?

We would like you to take part because you are between the ages of 8 and 15 and have AD/HD.

What if I want to stop?

You do not have to say "yes", if you do not want to take part. We will not punish you if you say "no". Even if you say "yes" now and change your mind after you start doing this study, you can stop and no one will be mad at you.

What will I have to do?

You will answer two different questionnaires that ask questions about your thoughts and feelings.

Will anything bad happen to me?

The questionnaires that you will fill out ask questions about your thoughts and feelings. Some kids feel uncomfortable answering these questions. You may skip any questions that you do not want to answer, and you may stop participating in the study at any time if you feel uncomfortable.

Will anything good happen to me?

By being in this study, you may help other kids who also have AD/HD by helping us to learn about why some kids with AD/HD also have anxiety.

Do I get anything for being in this study?
Your family will receive a $10 gift card for being in this study.

What if I have questions?
You are free to ask questions at any time.

If you understand this study and want to be in it, please write your name below.

_____   _____
Signature of child   Date

APPENDIX D: SUMMARY OF PARTICIPANT MEASURES

Summary of Participant Measures

Participant	Measure
Child	Multidimensional Anxiety Scale for Children—Child Report (MASC-C)
	Nowicki-Strickland Locus of Control Scale for Children (NSLOC)
Mother	ADHD Rating Scale (ADHD-RS)*
	Conners Comprehensive Behavior Rating Scale (CBRS)*
	Alabama Parenting Questionnaire (APQ)
	Parenting Alliance Inventory (PAI)
	Beck Anxiety Inventory (BAI)
	Beck Depression Inventory (BDI)
	Conners' Adult ADHD Rating Scale (CAARS)
	Computerized Diagnostic Interview Schedule for Children—IV (C-DISC-IV) AD/HD, GAD, and SP modules*
	Multidimensional Anxiety Scale for Children—Parent Report
Father	BAI
	BDI
	CAARS

* These measures were not completed by parents of children who had not received a diagnostic evaluation at the AD/HD Clinic in the six months prior to their participation in the study.

APPENDIX E: SAMPLE FEEDBACK LETTER

<date>

Dear *<Parent name>*,

Thank you very much for your recent participation in our research project examining risk factors for anxiety in children with AD/HD. Your participation has helped bring us closer to a better understanding of the relationship between AD/HD and anxiety in children.

Attached is a summary of the information that we collected about you. Because this information was collected as part of a research study and not a clinical evaluation, we are not able to offer formal clinical diagnoses or treatment recommendations. We can, however, tell you that *<child name>* met **research criteria** for *<diagnosis>*.

Again, I must emphasize that this is a research diagnosis and should not be used for making decisions about treatment. If you would like to obtain a formal clinical diagnosis for your child, this should be done by an experienced clinician after information from multiple sources has been taken into consideration. For this reason, I would recommend that you share the attached summary with any health care professional who may be evaluating your child in the future.

I very much appreciate your time and participation in this study. If you have any questions, please feel free to contact me at (336) 346-3196 x. 704.

Sincerely,

Sarah O'Rourke   Arthur D. Anastopoulos, Ph.D.
Project Director   Principal Investigator

SUMMARY OF CHILD ASSESSMENT RESULTS

Date of participation:

Computerized Diagnostic Interview Schedule for Children IV (C-DISC-IV):
<Child name> met ***research criteria*** for *<diagnosis>*. *<Child name>* did not meet ***research criteria*** for *<diagnosis>*.

Conners' Comprehensive Behavior Rating Scales (CBRS):

Subscales	Percentile
DSM-IV: AD/HD—Predominantly Inattentive Type	
DSM-IV: AD/HD—Predominantly Hyperactive-Impulsive Type	

AD/HD Rating Scale (ADHD-RS):

Subscales	Percentile
Inattention	
Hyperactive-Impulsive	

Multidimensional Anxiety Scale for Children (MASC):

Subscales	Percentile
Physical Symptoms	
Harm Avoidance	
Social Anxiety	
Separation Anxiety	
Total	

Description of Assessment Procedures

Computerized Diagnostic Interview Schedule for Children IV (C-DISC-IV)

The C-DISC-IV is a structured diagnostic interview that evaluates the presence or absence of various behavioral, emotional, and psychiatric disorders in childhood. In this study the C-DISC-IV was administered to a parent to determine if AD/HD, Generalized Anxiety Disorder, or Social Phobia are present in the participant.

Conners' Comprehensive Behavior Rating Scales (CBRS)

The CBRS is a behavior checklist that assesses AD/HD and a number of other common childhood conditions. The parent version of this scale was used in the current study to determine the degree to which child AD/HD symptoms deviated from expectations based upon comparisons with children of the same age and gender.

ADHD Rating Scale (ADHD RS)

The ADHD Rating Scale is an 18 item checklist that directly assesses AD/HD symptoms as defined by the Diagnostic & Statistical Manual of Mental Disorders - Fourth Edition (DSM-IV). The parent version of this scale was used in the current study to determine the degree to which child AD/HD symptoms deviated from expectations based upon comparisons with children of the same age and gender.

Multidimensional Anxiety Scale for Children (MASC)

The MASC is a self-report measure that assesses anxiety symptoms in children. The child version of this scale was used in the current study to determine the presence and severity of child anxiety symptoms.

Table 1. *Sample Characteristics*

Characteristic	Child/Adolescent (N = 28)		Mother (N = 28)	
	%	**(*n*)**	**%**	**(n)**
Sex				
Male	71.43	(20)	--	
Female	28.57	(8)	100	(28)
Race/Ethnicity				
Asian	3.57	(1)	--	
Black/African American	10.71	(3)	--	
White	71.43	(20)	--	
Multiracial	10.71	(3)	--	
Did not report	3.57	(1)	--	
Maternal Education Level				
Some High School	--		7.14	(2)
Completed High School	--		7.14	(2)
Some College or Associate's Degree	--		17.86	(5)
Four year degree or higher	--		39.29	(11)
Graduate Degree	--		28.57	(8)
Child Resides With				
Both Parents	85.71	(24)	--	
Mother Only	14.29	(4)	--	
Father Only	--		--	
Medication Status				
Any AD/HD Medication	82.14	(23)	--	
Stimulant Medication for AD/HD	75.00	(21)	--	
Non-Stimulant medication for AD/HD	21.43	(6)	--	
Other Psychoactive Medication	3.57	(1)	--	

[a]The number of participants taking stimulant medication and non-stimulant medication is not equal to the total number of participants taking any medication for AD/HD because some participants were taking both stimulant medication and non-stimulant medication for AD/HD.

Table 2. *Descriptive Statistics*

	M	*SD*	Minimum	Maximum	Skew	Kurtosis
AD/HD Status						
ADHD-RS IA Severity ($n = 28$)	19.43	5.29	7	27	-.59	-.24
ADHD-RS HI Severity ($n = 28$)	15.39	7.73	2	27	-.26	-.66
CBRS IA Severity ($n = 27$)	77.85	11.27	53	90	-.75	-.51
CBRS HI Severity ($n = 27$)	72.48	17.12	39	90	-.53	-1.16
Predictors						
BAI mother transform. ($n = 26$)	.79	.44	0	1.58	-.22	-.52
BDI mother ($n = 28$)	11.64	6.63	1	29	.47	.25
CAARS mother ($n=25$)	47.80	11.79	32	71	.68	-.78
BAI father transform. ($n = 7$)	.67	.39	.30	1.32	.87	-.41
BDI father ($n = 7$)	8.29	7.39	1	20	.71	-.97
CAARS father ($n = 6$)	65.50	13.25	53	84	.35	-2.07
APQ Inconsist. Parenting ($n = 28$)	-.16	.96	-2.04	1.71	-.03	-.26
PAI ($n = 27$)	74.52	19.51	23	97	-.86	.08
Outcome						
MASC-C ($n = 28$)	52.21	12.60	25	76	-.36	.34
MASC-P ($n = 28$)	50.25	15.34	23	78	.09	.76

Note. ADHD-RS = AD/HD Rating Scale; CBRS = Conners Comprehensive Behavior Rating Scale; IA = Inattention; HI = Hyperactivity-Impulsivity; BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory; CAARS = Conners Adult ADHD Rating Scale; APQ Inconsist. Parenting = Alabama Parenting Questionnaire Inconsistent Parenting subscale; PAI = Parenting Alliance Inventory; MASC-C = Multidimensional Anxiety Scale for Children – Child Version; MASC-P = Multidimensional Anxiety Scale for Children – Parent Version.

Table 3. *Correlations among Predictor and Outcome Variables*

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Child age	--														
2. Child sex	.01	--													
3. ADHD-RS IA Severity	.06	.22	--												
4. ADHD-RS HI Severity	-.34	.08	.47*	--											
5. BAI mother transform.	-.08	-.11	.18	.21	--										
6. BAI father transform.	.78*	-.18	.21	-.18	.34	--									
7. BDI mother	.10	-.05	-.03	-.01	.67**	.27	--								
8. BDI father	.80	-.12	.21	-.10	.21	.95**	.13	--							
9. CAARS mother	-.11	.12	.13	.03	.31	.42	.37	.57	--						
10. CAARS father	.17	-.64	.03	-.39	.43	.28	.53	-.01	-.52	--					
11. PAI	.01	.03	.08	.01	-.09	-.05	-.07	-.07	.28	.31	--				
12. APQ	-.02	-.24	-.01	.60**	.16	.40	.15	.46	.20	-.19	-.07	--			
13. NSLOC	.18	-.02	-.02	-.02	.03	-.04	-.15	-.11	.06	-.20	-.01	-.02	--		
14. MASC-C	.04	-.33	.14	.13	.01	.64	.06	.67	.12	.27	.14	.26	.27	--	
15. MASC-P	-.23	-.26	-.25	-.02	.27	-.13	.30	-.18	.20	.45	-.03	.04	.11	.27	--

Note. ADHD-RS = AD/HD Rating Scale; CBRS = Conners Comprehensive Behavior Rating Scale; IA = Inattention; HI = Hyperactivity-Impulsivity; BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory; CAARS = Conners Adult ADHD Rating Scale; APQ = Alabama Parenting Questionnaire Inconsistent Parenting Scale; PAI = Parenting Alliance Inventory; MASC-C = Multidimensional Anxiety Scale for Children – Child Version; MASC-P = Multidimensional Anxiety Scale for Children – Parent Version.
* $p < .05$, ** $p < .01$

Table 4. *Logistic Regression Examining Maternal Psychopathology as a Predictor of C-DISC-IV SP Diagnosis*

Predictor	β	*SE*	*Significance*
BAI mother transform.	5.16	4.44	.25
BDI mother	-.22	.30	.46
CAARS mother	.22	.13	.08

Note. BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory; CAARS = Conners Adult ADHD Rating Scale

Model $\chi^2 = 12.59$, $df = 3$, $p < .01$

Table 5. *Post-Hoc Logistic Regression Examining Maternal Psychopathology as a Predictor of CBRS SP T-score ≥ 65*

Predictor	β	*SE*	*Significance*
BAI mother transform.	.05	1.50	.97
BDI mother	-.21	.13	.12
CAARS mother	.22	.12	.06

Note. BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory; CAARS = Conners Adult ADHD Rating Scale

Model $\chi^2 = 10.22$, $df = 3$, $p < .05$

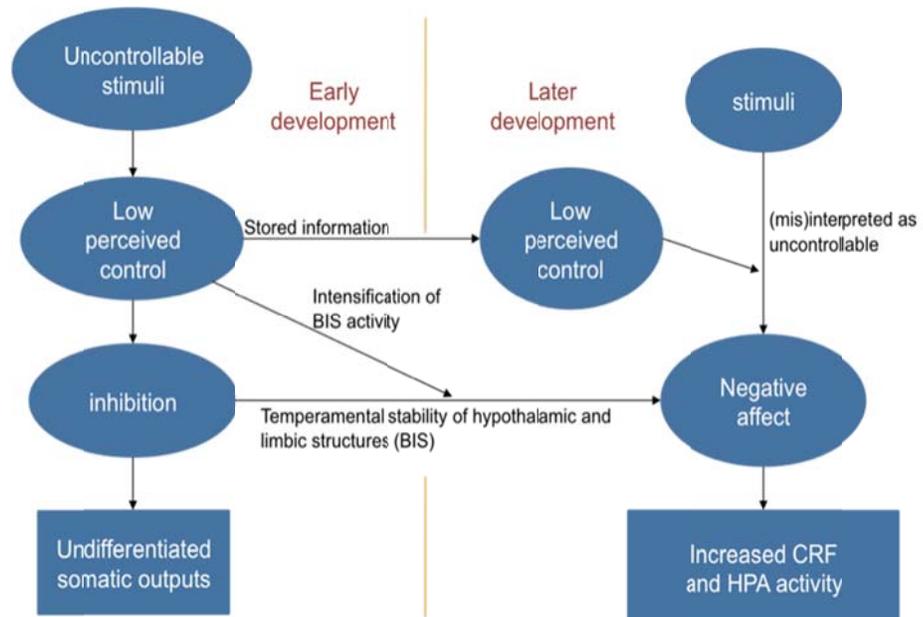Figure 1: Chorpita and Barlow's (1998) Model of Anxiety Development

Figure 2: Proposed Model of Anxiety Development in Children with AD/HD