UNRAVELING THE LONGITUDINAL RELATIONSHIP BETWEEN ADHD AND ANXIETY DISORDERS: THE CONTRIBUTION OF PARENTING

by

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Abstract

UNRAVELING THE LONGITUDINAL RELATIONSHIP

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Background: The substantially elevated risk for anxiety disorders among children with attention-deficit/hyperactivity disorder (ADHD) is well documented, although a causal explanation for the high comorbidity has yet to be identified. **Objective:** To investigate the extent to which ADHD in young children affects parenting practices, which, in turn, place children at risk for anxiety disorders. **Method:** A sample of 200 children was assessed at ages 3-4 years (Baseline; BL) and at three follow-up time-points (ages 5, 6, and 8 years). Presence or absence of ADHD at BL and presence or absence of anxiety disorder at ages 6 and 8 were determined via semi-structured interview with parents. Parenting behaviors were ascertained at BL, age 5, and age 6, via parental self-report on the Alabama Parenting Questionnaire – Preschool Revision (APQ-PR) and observer-coded video recordings of parent-child interactions (PCI) within the lab. **Results:** (1) Age 3-4 ADHD predicted greater rates of anxiety disorders at age 8. (2) Early ADHD

predicted less parent-rated positive parenting at ages 5-6 and more observer-rated parental negative emotionality and lack of respect for autonomy at ages 3-4 and 5; early ADHD also predicted poorer observer-rated quality of support in parents of 6 year-olds. (3) Lower parent-rated positive parenting between the ages of 3 and 5 years, and higher observer-rated parental negative expressions of emotion at ages 5 and 6, were predictive of child anxiety disorders at age 8, even after controlling for early temperament. (4) Mediation analyses found parenting, particularly that characterized by rejection, low warmth, and poor positive contingency management, to partially mediate the relationship between preschool ADHD and age 8 anxiety disorders. **Conclusions:** Parenting plays an important role in contributing to or alleviating risk for the development of anxiety disorders in children with ADHD. Implications for prevention and intervention are discussed.

Keywords: *ADHD, parenting, anxiety, psychopathology, positive parenting*

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Specific Aims

ADHD is a neurodevelopmental disorder that typically emerges during the preschool years and affects 5-7 percent of children (American Psychiatric Association, 2000). Over the years, it is associated with impairment across various aspects of life. Negative concurrent impairment and poor future prognoses are vast, and include poorer academic achievement, greater difficulty with peers, more family conflict, and increased conduct problems (DeWolfe, Byrne, & Bawden, 2000; G. J. DuPaul, McGoey, Eckert, & VanBrakle, 2001; Hinshaw, 1992a, 1992b), all of which, themselves, can further pave the way for negative outcomes.

ADHD also confers greater risk for other psychiatric comorbidity (American Psychiatric Association, 2000), which may be attributed to a combination of biological and environmental factors. There is an extant literature examining the relationship between ADHD with other externalizing disorders, like oppositional defiant disorder (ODD) and conduct disorder (CD), yet the relationship between ADHD and internalizing psychopathology is less clearly understood (Acosta et al., 2008; Angold, Costello, & Erkanli, 1999; Costello, Egger, & Angold, 2005). Compared to youth without ADHD, children with ADHD present with significantly higher rates of anxiety disorders, with anxiety co-occurring in as many as 50% of these children, depending on the sample (Angold et al., 1999; Jensen et al., 2001; Jensen, Martin, & Cantwell, 1997). Anxiety disorders have a detrimental effect on adaptive functioning, and children with comorbid ADHD+anxiety suffer more from academic, social, and cognitive difficulties and poor self-esteem than children with either disorder alone (Biederman, Newcorn, & Sprich, 1991; Tannock, 2000). However, despite alarmingly high rates of comorbidity and

related suffering, remarkably little is known about why the two co-occur so often, limiting the potential for preventive and early interventions to be put in place.

Parenting and family context have received considerable attention in literature on ADHD and comorbid disruptive behavior disorders (Deault, 2009; Johnston, 1996; Johnston & Jassy, 2007; Johnston & Mash, 2001; Pfiffner & McBurnett, 2006; Pfiffner, McBurnett, Rathouz, & Judice, 2005; Seipp & Johnston, 2005). Despite inconsistencies across studies, which may be attributable to variations in sample characteristics (e.g., age, clinically-referred vs. community sample), measures used, and whether data were collected concurrently or longitudinally, the most supported model to date holds ADHD as a predictor of more negative parent practices, which, in turn, predict more disruptive and defiant behavior in children; this behavior further predicts poorer parenting, giving way to a negative spiral, through which disruptive/defiant symptoms and negative parenting dynamically influence one another (Deault, 2009; Johnston, 1996; Johnston & Jassy, 2007; Johnston & Mash, 2001; Pfiffner & McBurnett, 2006; Pfiffner et al., 2005; Seipp & Johnston, 2005). Thus, child defiance and conduct problems are likely cultivated over time, in part, through a dynamic, bidirectional interplay between early externalizing behavior and parenting (particularly excessive control and rejection), whereas ADHD is more likely a cause than a product of poor parenting. Nevertheless, support for this is limited by studies' correlational or cross-sectional design, reliance on non-objective measures of parenting (i.e., parent or child questionnaires), and/or inception of longitudinal investigation during school age, by which time parent-child interaction patterns have already begun to set in. To truly understand whether ADHD leads to poorer parenting over time (and whether poorer parenting then leads to further

symptomatology) requires unbiased collection of parenting data (e.g., observer ratings during parent-child interactions), across multiple points in time, beginning during early childhood.

With the exception of separation anxiety disorder (SAD) and certain specific phobias (blood, animal, injection, and environmental type), which almost universally emerge before the age of 12 years, the onset of most anxiety disorders peaks in middle-to late-childhood and adolescence (Beesdo, Knappe, & Pine, 2009; Beesdo-Baum & Knappe, 2012). Interestingly, research suggests that comorbid anxiety disorders among children with ADHD are less characterized by phobias, and are, instead, more worry-based (Jensen et al., 2001; March et al., 2000; Molina et al., 2009). The most widely held understanding of the etiology of childhood anxiety disorders implicates a dynamic, transactional interplay of factors, including a predisposition (genetic, temperamental, neurobiological), the development of self-regulatory abilities and cognitive styles, and environmental factors, including parenting and early exposure to feared stimuli and experiences of control (Vasey & Dadds, 2001). In this view, during childhood, the environment, and particularly parenting, is central to the psychopathology.

Specifically, over-controlling and excessively restrictive parenting, poor management of positive contingency, and, to a lesser extent, cold and rejecting parenting, have been linked to an increased risk of childhood anxiety (Ashford, Smit, van Lier, Cuijpers, & Koot, 2008; Chorpita, 2001; Vasey & Dadds, 2001). Unfortunately, much of the research on parenting and childhood anxiety disorders has been limited by the use of cross-sectional and/or retrospective designs, and by the fact that parenting is frequently measured by self- and/or child-report, both of which are subject to biases. However, the

literature that exists provides compelling support for the role of harsh and controlling parenting in provoking and/or maintaining anxiety in children.

Together, these findings suggest one pathway by which anxiety may be borne in children with ADHD. Children with ADHD appear to elicit from parents and caregivers negative caregiving strategies, which are characterized by more rejecting and negative responding and more restrictions upon autonomy (Cunningham & Boyle, 2002; Deault, 2009; Johnston, 1996; Johnston & Jassy, 2007; Johnston & Mash, 2001). These parenting practices are then implicated in the etiology of anxiety disorders (Bogels & Brechman-Toussaint, 2006; Chorpita & Barlow, 1998; Hudson, Comer, & Kendall, 2008; Laskey & Cartwright-Hatton, 2009; McLeod, Wood, & Weisz, 2007; Rapee, 1997; Wood, McLeod, Singman, Hwang, & Chu, 2003). It follows that parenting may serve as a form of intermediary risk factor for, or mediator of, later anxiety among children with ADHD. For simplicity, this hypothesis is termed the Intermediate Parenting Hypothesis (IPH), and refers to the prediction that one route by which children with ADHD are particularly at risk for anxiety disorders is via the negative impact of their behavior on parents, whose practices, in turn, contribute to greater risk for anxiety disorders.

Two studies to date reported on the extent to which negative parenting among parents of children with ADHD may confer risk for childhood anxiety disorders (Kepley & Ostrander, 2007; Pfiffner & McBurnett, 2006). Both identified support for the IPH, reporting that more excessively restrictive and less warm parenting styles were present among families of children with ADHD, particularly where children had comorbid anxiety. However, these studies were limited by a lack of observation-based parenting measures (parenting practices were ascertained by questionnaires completed by parents

and children), as well as by correlational design, such that there was no way to assess temporal relations among factors.

With this in mind, the aims of the present study were as follows. The first aim was to replicate findings of increased incidence of anxiety disorders among children with ADHD, followed longitudinally, from age 3-4 years through age 8. The second objective was to replicate and further describe parenting characteristics in parents of children diagnosed with ADHD at age 3-4, as compared with their non-ADHD peers, using subjective (i.e., self-report scales) as well as objective (i.e., direct observation) measures of parenting, and to determine whether early ADHD predicted these differences over time. Third, we sought to replicate and further describe early parenting characteristics that place children at risk for later anxiety, employing the same measures of parenting within the same sample of children, and assessing the extent to which they predicted the emergence of anxiety disorders over time. The fourth and final aim was to investigate the extent to which parenting might play a mediating role in the relationship between early ADHD and later anxiety disorders. It was predicted that (1) children with early ADHD would be at increased risk for developing anxiety disorders; (2) young children's ADHD would cause parents to develop practices characterized by more control, less warmth, and less positive contingency management than parents of children without ADHD; (3) parenting practices high in control and low in warmth and positive contingency management would be predictive of anxiety disorders at later points in time; and (4) parenting practices would partially account for the development of anxiety disorders in children with ADHD.

CHAPTER 1: Introduction

Background on ADHD

Attention-Deficit/Hyperactivity Disorder (ADHD) is a chronic neurodevelopental disorder affecting roughly one in twenty children (American Psychiatric Association, 2000), or 5-7% of school-aged children in the U.S. and 5.3% worldwide, according to a recent meta-analysis (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). The disorder typically emerges by preschool or early school-age, persists throughout the lifespan in most individuals (American Psychiatric Association, 2000), and is characterized by a constellation of symptoms of inattention, hyperactivity, and/or impulsivity, which are pervasive across contexts and are highly impairing.

ADHD is highly heritable (Ehringer, Rhee, Young, Corley, & Hewitt, 2006; Gizer, Ficks, & Waldman, 2009), with genes estimated to account for up to 76% of the variance in the disorder (Faraone et al., 2005), though ranging from 29% to 98%, depending on subtype and study methodology (Ehringer et al., 2006; Faraone & Doyle, 2001; Rhee & Waldman, 2002). Genes also appear to contribute to whether or not the disorder persists throughout development (Kuntsi, Rijsdijk, Ronald, Asherson, & Plomin, 2005; Price et al., 2005). For instance, a study of preschool children reported heritability estimates of .78 to .81, with 91% of the variance in the disorder's stability across time attributable to genes (Price et al., 2005). Potential genetic markers that have been studied most in ADHD include DAT1, DRD4, DRD5, 5HTT, DBH, ADRA2a, TPH2, MOA, and SNAP25 (Gizer et al., 2009). Thus, while there is no singular gene that clearly produces ADHD, the disorder is highly genetically determined.

The neurobiological correlates of ADHD are heterogeneous as well. Anatomical correlates of the disorder include diverse circuitry, most prominently involving fronto-striato-thalamo-cortical loops and similar circuits involving the cerebellum (Castellanos, 2002; Castellanos et al., 1996; Seidman, Valera, & Makris, 2005). Attempts to pinpoint or isolate specific brain regions responsible for the disorder have largely been unsuccessful, with different studies reporting different structural and functional differences between ADHD and non-ADHD groups. Recent advances in the neurobiology of ADHD have posited the disorder as arising from more primitive, subcortical abnormalities that impact default-mode activity (Uddin et al., 2008). Others have identified delayed, or immature cortical thickness (P. Shaw et al., 2007; P. Shaw et al., 2006). Impaired executive function, characteristic of the disorder, has been related to these structural and functional differences, with improvements in executive function and normalization of cortical thickness hypothesized to underlie relative remission of the disorder (Halperin & Schulz, 2006).

Throughout the lifespan, ADHD is associated with impairment across various aspects of life. In childhood, negative consequences affect academic, social, behavioral, and familial functioning (DeWolfe et al., 2000; G. J. DuPaul et al., 2001; Hinshaw, 1992a, 1992b). Poorer academic achievement has been reported almost universally, with reduced math and reading scores, grade point average, and educational attainment, and many (according to one study, one third of) children failing to complete high school (Barbesi, Katusic, Colligan, Weaver, & al., 2007; Barkley, Fischer, Smallish, & Fletcher, 2006; Breslau et al., 2009; McGee, Prior, Williams, Smart, & Sanson, 2003; Molina et al., 2009; Polderman, Boomsma, Bartels, Verhulst, & Huizink, 2010). Socially, children

with ADHD are significantly more impaired than their typically developing peers (Hinshaw, 2002; Mrug, Hoza, & Gerdes, 2001; Mrug, Hoza, Pelham, Gnagy, & Greiner, 2007). Children with ADHD go on to demonstrate higher rates of conduct problems, delinquency, arrests, early parenthood, and social problems, and poorer career attainment (fired more, have lower job performance) than their typically developing peers (Barkley et al., 2006; Molina et al., 2009). The behavioral dysregulation exhibited by young children with ADHD often makes them difficult to manage, and can be taxing on parents, teachers, and other adults responsible for their care. Higher levels of stress among caregivers and families have been documented virtually universally in studies of children with ADHD and their families (Anastopoulos, Guevremont, Shelton, & DuPaul, 1992; DeWolfe et al., 2000; G. J. DuPaul et al., 2001; Mash & Johnston, 1990; van Oort, Verhulst, Ormel, & Huizink, 2010; Wells et al., 2000). ADHD affects not only the child, but also those around him.

There currently exist two treatments for ADHD that are considered empirically supported: psychopharmacological treatment (methylphenidate and amphetamine, primarily, and atomoxetine and guanfacine, more recently) and behavioral therapy (specifically, a combination of parent training and school-based contingency management)(Johnston, Hommersen, & Seipp, 2008; MTA Cooperative Group, 1999). Behavioral treatments derive from principles of operant conditioning, in that behavior is shaped via reinforcement contingencies (Chronis, Jones, & Raggi, 2006; Pelham & Fabiano, 2008). Behavioral therapy involves, most of all, work with the parent. Specifically, parent training teaches parents to improve their use of praise and practice positive contingency management (positive parenting) to increase the incidence of

desirable behaviors, and to avoid reacting angrily or providing harsh or inconsistent punishments when children misbehave. Behavioral therapy is highly effective at reducing off-task behavior, improving compliance, and improving motivation among children with the disorder, although it is less adept at treating the core, underlying symptoms of the disorder (Abikoff, 2009; Chronis et al., 2006; Knight, Rooney, & Chronis-Tuscano, 2008; Pelham & Fabiano, 2008; Pelham, Wheeler, & Chronis, 1998).

Stimulant and non-stimulant medications act upon the monoamine systems, regulating availability of dopamine and norepinephrine (Minzenberg, 2012). These medications have been shown to be highly efficacious in reducing the core symptoms of ADHD in a majority of cases, across the lifespan (Greenhill, Halperin, & Abikoff, 1999; Johnston et al., 2008; Minzenberg, 2012; J. G. Wood, Crager, Delap, & Heiskell, 2007). Unfortunately, medication is usually discontinued within a short period of time, and for the most part, the beneficial effects of neither medicine nor behavioral treatments persist beyond discontinuation of treatment; as a result, children who have been treated with either or both present with as severe symptoms and associated impairment as their never-treated peers (Molina et al., 2009).

Comorbidity

Not only does ADHD create serious problems for children and those around them, it is also associated with significantly increased risk for psychiatric illness, including both externalizing disorders, such as oppositional defiant disorder (ODD) and conduct disorder (CD), and internalizing disorders, such as anxiety and depression (American Psychiatric Association, 2000; Larson, Russ, Kahn, & Halfon, 2011; Taurines et al., 2010). Despite substantial literature on the comorbidity between ADHD and other

externalizing disorders, far less is known about the co-occurrence of ADHD with internalizing psychopathology (Cosgrove et al., 2010)

An especial paucity of research exists on the comorbidity between ADHD and anxiety. This is despite alarmingly high rates of comorbidity between these disorders. Data from the National Survey of Children's Health indicate that, among a population sample, nearly one in five children with ADHD suffers from a comorbid anxiety disorder (18% compared to 2% of their non-ADHD peers; Kessler et al., 2005). Clinic samples, on the other hand, have shown up to a 50% co-occurrence rate of anxiety disorders among children with ADHD (Angold et al., 1999; Jensen et al., 2001; Jensen et al., 1997). Roughly one third of the children included in the MTA study had at least one anxiety disorder, not including specific phobias (MTA Cooperative Group, 1999a, 1999b).

Data from the MTA study and supported by others indicate that certain types of anxiety disorders are more common than others among children with ADHD; whereas specific phobias are less common, worry-related disorders, like GAD, occur more frequently (Jensen et al., 2001; Schatz & Rostain, 2006; Tannock, 2000). In this way, children with ADHD are prone to worry-related anxiety, as in worrying about competency in school, performance in sports, or interactions with peers, which are understandable given past experiences of failure in these situations (Schatz & Rostain, 2006).

Like ADHD, anxiety disorders lead to distress and impairment across a range of contexts, impacting children socially, behaviorally, academically, and in family relations (Costello et al., 2005; Vasey & Dadds, 2001). Symptoms of anxiety can also mimic

symptoms of ADHD, including concentration difficulty, distractibility, and restlessness, and can lead to avoidance-like behaviors that contribute to avoidance of and difficulty completing mundane tasks. As a consequence, where ADHD and anxiety co-occur, anxiety has the potential to exacerbate ADHD symptomatology, compounding the impairment profile. Compared to children with ADHD alone, children with the comorbidity have more academic, social, and cognitive problems, as well as lower self-esteem (Biederman et al., 1991; Tannock, 2000). Children with anxiety disorders are also at far greater risk for depression (Costello et al., 2005). With some exception, in the absence of treatment anxiety disorders typically run a life-time course, meaning they tend to persist, along with ADHD (Beesdo-Baum & Knappe, 2012; Vasey & Dadds, 2001).

High rates of comorbid anxiety disorders among children with ADHD are therefore of real concern. The cost over time of the comorbidity is significant in terms of impairment and suffering, as well as financial burden to society. The identification of mechanisms by which individuals with ADHD are at risk for anxiety disorders has important potential to inform intervention and prevention efforts, and to reduce these costs. Longitudinal studies are particularly critical for illuminating the contribution of different factors over time (Rutter, 1993).

Anxiety Disorders in Childhood

Anxiety disorders affect a substantial number of children and adolescents without ADHD, as well, with lifetime prevalence estimates between 15 and 20%, and with most anxiety disorders persisting across development (Costello et al., 2005; Sakolsky, McCracken, & Nurmi, 2012). Anxiety disorders are difficult to diagnose during early childhood, and there are limited data on prevalence during preschool (ages 2-5).

However, using standardized interviews, preschool rates range from <1% - 11.5% for separation anxiety, <1% - 6.5% for overanxious (OAD)/GAD, and <1% to 2.3% for social phobia (Costello et al., 2005). According to a recent review, GAD and social phobia tend to emerge first, with median age of onset estimated at 6 and 7 years of age, respectively, and with both showing large variability in range; median age of onset for specific phobias is 7-8, SAD is closer to 9 years of age, and OCD 10 years (Costello et al., 2005). Curiously, data on GAD onset have been inconsistent, and others have reported much later median onset, around adolescence (Beesdo-Baum & Knappe, 2012). By school-age, estimates of anxiety disorders increase to approximately 2.8% for SAD, up to 10% for specific phobias, up to 7% for social phobias, <1% for panic and agoraphobia, and less clear estimates of GAD, with suggestion of less than 3.5% during childhood (Beesdo-Baum & Knappe, 2012).

Vasey and Dadds (2001) summarized nine factors that likely serve as risk or protective factors for anxiety. For simplicity, these are grouped here into: (1) *biological predispositions*, which include genetic factors, neural factors, and temperament; (2) *cognitive factors,* which include emotion regulation skills and cognitive biases and distortions; and (3) *environmental exposure*, including parenting responses, early control experiences, extent of experience with common conditioned stimuli for phobic anxiety, and level of exposure to feared stimuli (Vasey & Dadds, 2001, p. 15). These factors are not independent of one another. Genetics, for instance, may correlate with parenting responses, given that biological parents are influenced by the same genetic predispositions, as well. Parenting response is also affected by infants' temperament; for instance, parents of easy-going infants are likely to react more calmly to them than

parents whose children are of more finicky temperaments, who may be on-edge or overwhelmed as a result. These factors also translate to which environments parents expose their children to; for instance, anxious parents, or parents of more difficult children, may exercise certain precautions, such as not bringing their children to places that are likely to elicit negative responses. Based on these experiences, children develop cognitions about the world around them, causing future behaviors or fears to be more or less likely as a result. These are just a few examples of the many ways in which biological, environmental, cognitive factors can dynamically interact over time, to reinforce anxiety. Further discussion of these three groups of risk factors is included below. As will be emphasized throughout, the various factors are best understood, collectively, as opposed to independently, to protect against or place children at risk for anxiety disorders.

Biological Predispositions

Genetic factors. A recent review estimated moderate heritability of anxiety disorders but with a wide range, from 20 to 65% (Costello et al., 2005; Sakolsky et al., 2012). This is substantial but below heritability estimates of ADHD. Genetic studies have highlighted inheritance that is relatively nonspecific among worry-based disorders, including GAD, agoraphobia, panic disorder, and social phobia, such that individuals with any of these are equally likely to have family members with one of the other worry-based disorders (Hettema, Prescott, Myers, Neale, & Kendler, 2005). Specific phobias appear to be inherited separately. Differences in genetic risk for either of these groups are suggestive of different neurobiological mechanisms behind the two as well.

Neural correlates. Research on the neurobiology of anxiety disorders emphasizes deficient circuitry in two systems, as a function of the specific disorder. In OCD, studies have highlighted functional and structural correlates within some of the same areas as ADHD, including the basal ganglia, orbitofrontal cortex, and anterior cingulate cortex; in contrast, among other, "fear-based" anxiety disorders, the amygdala and prefrontal cortical circuits have been implicated (Blackford & Pine, 2012). An increase in glucocorticoids in these circuits accompanies anatomical differences, and hormone release during fear may contribute to the perpetuation of clinical levels of anxiety (Gunnar, 2001).

Temperament. Temperament is used to refer to a behavioral phenotype of a relatively stable, neurobiological disposition. Three broad temperamental styles have been linked to anxiety: Negative Affect, Effortful Control, and Behavioral Inhibition.

Negative affect refers to negative reactivity, or one's tendency to become easily upset. It is comprised of lower-order behavioral traits that vary depending on the measure used, but generally reflect such traits as being easily angered or difficult to sooth (Rothbart, Ahadi, Hershey, & Fisher, 2001). Effortful control refers to cognitive, behavioral, and emotional regulation, and subsumes impulsivity, inhibitory control, and attentional focusing. Negative affect and effortful control have been widely shown to be associated with symptoms of anxiety, in both concurrent and prospective studies (reviewed by Lonigan & Phillips, 2001; Nigg, 2006).

Behavioral Inhibition speaks to a broader neurobiological disposition, and as a construct refers to the tendency of an individual to display hypervigilance, withdrawal, and negative affect, particularly in unfamiliar settings (Fox, Henderson, Marshall,

Nichols, & Ghera, 2005). It is considered a more low-level dimension of temperament than more specific traits like negative affect and effortful control. Behavioral inhibition is associated with increased reactivity of the autonomic nervous system, including elevated vigilance and startle response, heart rate and heart rate variability, blood pressure, muscle tension, and cortisol levels (Degnan, Almas, & Fox, 2010; Lonigan & Phillips, 2001). Behavioral inhibition has been widely shown to be a temperamental correlate of anxiety disorders (Calkins & Fox, 2002; Degnan et al., 2010; Fox et al., 2005). For instance, preschool behavioral inhibition is predictive of anxiety disorders five years later (Hirshfeld-Becker, Biederman, Henin, Faraone, Davis, et al., 2007; Hirshfeld-Becker, Biederman, Henin, Faraone, Micco, et al., 2007) as well as symptoms and diagnosis later in development (Biederman et al., 1990; Gladstone, Parker, Mitchell, Wilhelm, & Malhi, 2005; Lonigan & Phillips, 2001). Despite this, and while some consider behavioral inhibition a precursor to anxiety disorders, research shows that only one third or fewer of children who are behaviorally inhibited go on to develop anxiety disorders (Lonigan & Phillips, 2001)

Thus, temperament high in behavioral inhibition, negative affect, and/or effortful control is best conceptualized as predisposing an individual to develop anxiety disorders, when exposed to certain environmental triggers. Potential interactions among temperament, parenting, and environment were discussed earlier. For example, an infant high in behavioral inhibition may lead parents to avoid activating settings altogether, reducing the child's opportunity to learn that certain places need not be feared, and making more likely a fearful response in the future. In a different example, children who are poor at regulating their emotions (low in effortful control) may elicit parental

behaviors that remove the source of distress, but which do not teach effect skills for self-regulation, making children less likely to regulate themselves in the future (Vasey & Dadds, 2001).

Cognitions

Cognition is used here to refer to both emotion regulation and cognitive style (Vasey & Dadds, 2001). In line with temperament research on effortful control, poor ability to regulate one's emotional state is a skill that is likely predictive of anxiety symptoms and disorders, just as adequate emotion regulation skills are protective against them (Thompson, 2001). Similarly, attentional biases have been shown to predate the onset of anxiety disorders and serve to perpetuate anxious feelings (Lonigan, Vasey, Phillips, & Hazen, 2004; Vasey & MacLeod, 2001). Dispositions to different cognitive styles underlie the processes of appraisal and interpretation of situations, and mediate the way in which external events, or triggers, give way to anxious responses (Bogels & Brechman-Toussaint, 2006; Chorpita, 2001).

Environmental Exposure

Aspects of environmental exposure that are particularly relevant to anxiety disorders include parental responses, early control experiences, extent of experience with common conditioned stimuli for phobic anxiety, and level of exposure to feared stimuli (Vasey & Dadds, 2001). During childhood, parenting and environmental variables are often intertwined (Lau, Rijsdijk, Gregory, McGuffin, & Eley, 2007). Particularly in early childhood, as has been illustrated throughout this section, parenting constitutes a substantial majority of a child's experiences. As such, the next section is devoted to parenting practices that contribute to these experiences, and which have been specifically

implicated in the etiology of anxiety. First, a background is included to provide an overview of parenting research and theory. These principles are then applied to the discussion of anxiety disorders.

Parenting and Childhood Anxiety

Much of current parenting theory stems from Baumrind's (1971, 1996) work, which lays out styles of parenting along two dimensions, "responsiveness" and "demandingness." Responsiveness, according to Baumrind (1971), "refers to the extent to which parents intentionally foster individuality, self-regulation, and self-assertion by being attuned, supportive, and acquiescent to the child's special needs and desires" (p.410). Responsiveness subsumes warmth (affection, empathy), reciprocity (being in-tune with one's child), clear communication (about feelings, reasons for rules, and expectations about enforcing them), and [secure] attachment (Baumrind, 1996).

Baumrind (1971) used demandingness to refer to "the claims parents make on children to become integrated into the family and community by their maturity expectations, supervision, disciplinary efforts, and willingness to confront a disputative child" (p. 411). Within this construct are direct confrontations (which, ideally, should be supportive, nonpunitive, authentic, and sensitive), monitoring (clear expectations that are regulated), and consistency and contingency of discipline (predictability of positive and negative reinforcers immediately following children's behavior). Baumrind's demandingness construct may therefore be likened to contingency management, or limit setting. She termed parenting that was high in both responsiveness and demandingness, "Authoritative parenting," and research has consistently shown this style of parenting to predict the highest levels of psychosocial adjustment in children, compared with

Authoritarian parenting (that which is high in demandingness and low in responsiveness), Permissive parenting (in which parents are responsive but not demanding), and Unengaged parenting (in which parents are neither) (Barber, 2001).

The behavioral control referenced by the construct of demandingness is considered important and beneficial for children's development, in promoting emotional growth as well as individuation. Chorpita and Barlow (1998) used the term, "sensitivity-consistency-contingency" to include the practice of warm and responsive parenting alongside [positive] contingency management, which, they argued, promotes not only positive behavior, but positive emotional development, by enabling feelings of control in children. When children are able to predict the outcomes of their behavior, they feel more able to personally influence what occurs in their surroundings, which promotes [their own] feelings of control (a.k.a., locus of control) and reduces feelings of anxiety.

Contingency management, or behavioral control, is not to be confused with excessive, intrusive, and/or psychological control, which are associated with significant risk in children (Barber, 2001). A lack of personal control, Chorpita and Barlow (1998) argue, instead makes children more susceptible to anxiety, in a sense, akin to the paradigm of learned helplessness proposed by Overmier and Seligman (1967). Chorpita and Barlow (1998) contend that parenting that restricts children's opportunities to behave independently or assert their autonomy reduces children's locus of control. Together, they argue, emotional support and respect for autonomy promote children's sense of comfort and control over their environment, which reduces risk for internalizing symptomatology.

Parker (1983) proposed similar constructs, care and protection; care maps closely onto the warmth, contingency dimension of Chorpita and Barlow's (1998) model, whereas protection is likened to the dimension of [over]-control. Others have echoed the importance of contingency control as promoting healthy development, in contrast to psychological control, whereby the restriction of opportunities for autonomous thought and decision-making is considered damaging to children and adolescents, alike (Barber, 2001; Rapee, 1997; Steinberg, Elmen, & Mounts, 1989).

Thus, despite slight variations between models, three factors consistently emerge as relevant to the psychosocial development of children: warmth and positive regard (the opposite of hostility or rejection), contingent reinforcement (demandingness, behavioral control, or positive reinforcement), and respect for autonomy (opposite of over-control or psychological control). Rejecting, hostile, and critical parenting is likely to be internalized by children over time, affecting children's cognitions and beliefs about themselves and those around them: children may develop negative beliefs about themselves (for instance, as incompetent, a failure, or undeserving, etc.), and may begin to view the world as a hostile or dangerous place (Bogels & Brechman-Toussaint, 2006). Over-control in parents reduces children's opportunities to act autonomously, which theoretically limits development of their internal locus of control, impeding a sense of competence or power and instead portraying a sense of being unable to control one's surroundings (Bogels & Brechman-Toussaint, 2006; Chorpita & Barlow, 1998; Rapee, 1997). Inconsistent contingency management operates similarly, by reducing children's sense of control over their surroundings and adding to their view of the world as uncontrollable; consistent responding promotes a sense of predictability, order, and

security in one's surroundings. In sum, warmth, reinforcement, and encouragement of autonomy are viewed as protective against the development of childhood anxiety disorders, and rejection, poor contingency management, and excessive control appear to contribute to risk for anxiety disorders (Ashford et al., 2008; Bogels & Brechman-Toussaint, 2006; Chorpita, 2001; Vasey & Dadds, 2001).

A number of comprehensive reviews highlight empirical support for the relationship between these parenting styles and childhood anxiety (Bogels & Brechman-Toussaint, 2006; McLeod, Wood, et al., 2007; Rapee, 1997; Wood et al., 2003). Unfortunately, methodological differences among studies are numerous, and include differences in children's age (with ranges from age 2 or 4 through age 18), whether parenting measures consisted of non-biased observations or participant rating scales (and if the latter, whether questionnaires were completed by parents, children, or both), which dimensions of parenting were assessed, whether data were gathered cross-sectionally or longitudinally, and whether childhood anxiety was assessed dimensionally (usually within a non-clinical sample) or dichotomously (comparing children with and without diagnoses). It is therefore not surprising that findings are inconsistent. The majority of studies have relied on questionnaires, completed by parents or children, in cross-sectional studies. Overall, despite varying effect sizes, studies have provided empirical support for the theoretical notion that parental rejection and over-control are associated with higher levels of anxiety symptoms and diagnoses in their offspring. There is a particularly strong association between parental over-control and childhood anxiety symptoms and disorders, and a slightly less robust association between anxiety and parental rejection, whereas rejection has been more closely linked to depression (Bogels & Brechman-

Toussaint, 2006; Chorpita & Barlow, 1998; McLeod, Weisz, & Wood, 2007; McLeod, Wood, et al., 2007; Rapee, 1997; Wood et al., 2003). A recent meta-analysis, collapsing across ages and methodology, reported 18% of the variance in dimensional ratings of children's anxiety to be explained by the construct of autonomy-granting (McLeod, Wood, et al., 2007) Another review revealed medium to large effect sizes of maternal control, and concluded that maternal control, particularly in novel situations, is an important predictor of child anxiety (Wood et al., 2003).

Unfortunately, there exists a real dearth of longitudinal studies investigating directionality of the association between child anxiety and parent behavior. Implicit in theory and research on parenting and child anxiety is the notion that parenting practices cause anxiety in children. As discussed earlier, this would be an oversimplification, as parenting most likely does *not* contribute unidirectionally to anxiety. Research suggests that child temperament, anxious symptomatology and parenting can affect each other reciprocally (Lonigan & Phillips, 2001; Thompson, 2001). While parental behaviors may increase or maintain children's anxious symptoms, these symptoms are apt to elicit parenting behaviors as well. For these reasons, longitudinal investigations are needed to better elucidate cause and effect.

One pair of longitudinal studies illustrated this dynamic relationship between children and parents. In the first study, children's shyness at age 2 was predictive of self-rated parental control at age 4, whereas these parent ratings when children were 2 were not predictive of shyness at 4 (Rubin, Nelson, Hastings, & Asendorpf, 1999). A subsequent investigation, however, revealed that the effect of shyness and inhibition at age 2 on children's social reticence at age 4 was moderated by whether or not their

mothers had been observed as being intrusive or derisive toward their children at age 2 (Rubin, Burgess, & Hastings, 2002).

Another longitudinal study suggested that parenting may lead to anxiety by way of instilling negative cognitions in children. Mothers' self-reports of rejection and restrictiveness when children were 5 years old were associated with children's reported self-criticism at age 12, even after controlling for parent-rated earlier temperament (Koestner, Zuroff, & Powers, 1991). In a similar vein, a more recent study found parents of anxious children to possess more negative beliefs about their children (Laskey & Cartwright-Hatton, 2009). The same study reported that parental discipline style (harsh discipline) mediated the relationship between parental anxiety and childhood internalizing symptoms. It may be, therefore, that parental negative cognitions about their children are transferred to children, themselves, via harsh disciplinary practices.

Further studies employing longitudinal designs and observational parenting measures are clearly needed to better understand the relationship between parenting and anxiety. However, parental control and rejection constitute likely mechanisms by which children may be prone to developing anxiety disorders. Studies are also needed to empirically test whether poor contingency management contributes to anxiety in offspring.

Interestingly, in recent years, studies have begun to elucidate neurobiological correlates of the impact of parenting behaviors on children's psychosocial development (Glascoe & Leew, 2010). One study found that observed maternal aggression moderated the relationship between adolescents' amygdala and anterior cingulate volume and depressive symptoms (Yap et al., 2008). Another study by the same researchers reported

volumetric differences in the brains of adolescents whose mothers were observed to respond more punitively during a conflictual problem-solving task. Relative to adolescents whose mothers did not exhibit this punitive response, structural MRI of adolescents with punitive mothers revealed increased brain volume in left anterior cingulate and bilateral orbitofrontal cortex (Whittle et al., 2009). Adolescents whose mothers behaved punitively during a positive event-planning interaction demonstrated larger right amygdala volume, as well. In a longitudinal neuroimaging study following young children from preschool through adolescence, Rao and colleagues found that preschool children whose parents who were observed to be low in "warmth" were characterized by larger left hippocampal volume by the time they reached adolescence than children whose parents were high in warmth (Rao et al., 2010). These studies provide preliminary evidence that the warmth/rejection dimension of parenting may have significant and lasting effects on brain development, providing anatomical evidence of their impact on internalizing pathology. Intriguingly, in Rao et al. (2010)'s investigation, parental warmth at age 8 no longer showed the same association with later hippocampal volume as did parental warmth at age 4. The authors interpreted this as signifying a sensitive period in brain development in early childhood, during which time parenting has an especially potent effect. No studies have yet been published documenting brain differences as a function of parental control. Nevertheless, the concept that normal-range parenting within the general population (i.e., in the absence of physical abuse, trauma, etc.) can affect the neuroanatomical correlates of emotional disorders is intriguing and highlights the impact that parenting behavior can have on children.

Why Do ADHD and Anxiety Co-Occur?

As described above, certain biological and environmental factors are particularly relevant to understanding risk for anxiety disorders. The following section focuses on the ways ADHD may relate to some of these factors. First, an overview of potential biological contributors is considered. In line with the literature on anxiety disorders outlined above, cognitive factors are considered second, and then environmental correlates are reviewed. Finally, the chapter concludes with a discussion of parenting in ADHD, which is again considered with literature on the developmental psychopathology of anxiety disorders in mind. It will be argued that parenting provides an important avenue, directly and indirectly (via interactions with other variables), through which anxiety disorders may be influenced among children with ADHD.

Biological Predispositions for the Comorbidity

Genetics. As reviewed earlier, it is widely accepted that ADHD is a highly heritable disorder, more so than anxiety (Burt, 2009; Faraone, 2000; Faraone et al., 2005; Sakolsky et al., 2012). Genetic research has been somewhat inconclusive with regard to the unique genetic versus environmental origins of ADHD with internalizing symptomatology, although most studies suggest that genetic predispositions to externalizing and internalizing disorders are relatively separate from one another (Cosgrove et al., 2010; Ehringer et al., 2006). A recent meta-analysis focusing on shared environmental variables (i.e., variables that are common across siblings, such as home environment, or family events) identified significant contributions of shared environment to an array of internalizing disorders, as well as ODD and CD; the only disorder studied for which shared environment was not predictive was ADHD (Burt, 2009). This is

consistent with the notion that anxiety disorders are more susceptible to environmental influences than ADHD.

Research on family history of ADHD and anxiety is varied. Maternal depression is a risk factor for child internalizing problems more generally (Humphreys, Mehta, & Lee, 2010), a finding that may have basis in both genes and the environment. Family history of depression is also associated with higher rates of ADHD and externalizing disorders more generally. However, this is not so for the heterotypic comorbidity of anxiety and externalizing disorders (Angold et al., 1999; Cosgrove et al., 2010; Faraone, 2000; Faraone, Biederman, & Mick, 2006; Jarrett & Ollendick, 2008; Sciberras, Ukoumunne, & Efron, 2011). In fact, the family history literature is fairly consistent in reporting that anxiety and ADHD are probably inherited independently (Braaten et al., 2003; Jarrett & Ollendick, 2008). There is some evidence of an association between ADHD+anxiety and maternal substance abuse disorder (Kepley & Ostrander, 2007). There is also an emerging literature that maternal antenatal anxiety may substantially increase risk for anxiety, ADHD, and other externalizing problems in offspring, although the specific timing of the vulnerability (e.g., prior to 32 weeks) and the fact that results hold after controlling for mother's postnatal anxiety suggests that the association may be more due to prenatal environment than to heredity (Clavarino et al., 2010; Van den Bergh & Marcoen, 2004).

Thus, a few statements can be made about the genetic literature. First, ADHD shows higher estimates of heritability than anxiety, which may be inherited but is also highly susceptible to environmental influence. Second, in contrast to depression, which research suggests may be co-inherited with ADHD, anxiety is less prone to such co-

inheritance, and seems to be independently transmitted. Finally, despite some evidence that maternal antenatal anxiety or substance abuse disorder may predict anxiety and ADHD, these factors likely exert their influence via the prenatal environment rather than through genes. Together, these data highlight the importance of the environment in the development of anxiety among children with ADHD.

Neural Correlates. Research on the neural correlates of ADHD has focused largely on catecholamine-rich circuits connecting prefrontal cortex to the striatum, thalamus, and looping back to prefrontal cortex as underlying the attentional and behavioral dysregulation seen in ADHD (Castellanos, 2002; Castellanos & Tannock, 2002; Sagvolden & Sergeant, 1998; Seidman et al., 2005). Dysregulated prefrontal cortical projections to limbic areas have been posited as an overlapping neural mechanism possibly linking ADHD to anxiety. More specifically, disinhibition of the nucleus accumbens, which receives input from prefrontal cortex, would lead to increased activation of the fear circuitry driven by the amygdala (Levy, 2004; Nigg & Casey, 2005).

From a more cognitive/neuropsychological perspective, some researchers have suggested that ADHD+anxiety constitutes its own, distinct disorder (Acosta et al., 2008; Carlson & Mann, 2002; Schatz & Rostain, 2006; Skirbekk, Hansen, Oerbeck, & Kristensen, 2011), characterized by especially poor cognitive regulation and inattention, as opposed to hyperactivity/impulsivity (Acosta et al., 2008; Skirbekk et al., 2011). For instance, a subgroup of children with ADHD that is characterized by "sluggish cognitive tempo" (SCT) displays poor cognitive regulation and is also mostly comprised of inattentive children (Carlson & Mann, 2002; Skirbekk et al., 2011), meeting criteria for

inattentive or combined subtypes. Children with SCT are less at risk for externalizing problems but more at risk for anxiety than their non-sluggish counterparts. However, not all children with the comorbidity are also characterized as having SCT, suggesting that, at best, the SCT model accounts for only a subset those afflicted by both anxiety and ADHD.

Other investigators have examined whether ADHD+anxiety represents its own discrete disorder by investigating whether individuals with ADHD+anxiety demonstrate differential response to stimulants (Schatz & Rostain, 2006). Some research has reported that stimulants are less effective at improving working memory in the comorbid group (Bedard & Tannock, 2008). However, in terms of core symptom improvement, anxious and non-anxious children with ADHD respond similarly to stimulant medication (Abikoff et al., 2005; Diamond, Tannock, & Schachar, 1999; S.R. Pliszka, 2003). Among children with comorbid ADHD+anxiety it is has been suggested that stimulant treatment drives improvement in ADHD-related symptoms, whereas psychosocial treatment drives improvement in anxiety, which suggests an additive quality to the dual diagnosis, each with a distinct etiology (Jensen et al., 2001; S.R. Pliszka, 2003). While it is possible that future studies of the effects of treatment will further elucidate the unique as well as shared substrates in the two co-occurring disorders (Jarrett & Ollendick, 2008; Jensen et al., 2001; Pfiffner & McBurnett, 2006), existing studies of medication response suggest that ADHD+anxiety represents a co-occurrence of two distinct disorders as opposed to a third disorder of its own.

Temperament. Temperament has been used to describe similarities among, as well as differences between, ADHD and internalizing disorders. As reviewed by Nigg

(2006), temperamental styles related to the personality traits of Extraversion (e.g., surgency, positive affect, or approach) and Neuroticism (negative affect, negative temperament, or withdrawal) may be particularly descriptive of ADHD and anxiety, respectively. Temperament high in approach has more specifically been associated with hyperactive-impulsive symptoms. In contrast, negative affect and withdrawal are considered potential precursors to anxiety. Effortful control, encompasses cognitive and behavioral regulation as well as emotional regulation, and may be particularly relevant to symptoms of inattention and anxiety. Nigg (2006) proposed two distinct temperamental routes to ADHD: one characterized by low effortful control, and the other by high approach. Whereas high approach behavior is also linked to antisocial behavior, and may be a shared predisposition to comorbid externalizing disorders, low effortful control may lead to a different, less impulsive-like and more inattentive presentation of ADHD. When coupled with negative affectivity (or withdrawal), low effortful control is linked to anxiety (Lonigan, Vasey, Phillips, & Hazen, 2004; Nigg, 2006). Therefore, where anxiety and ADHD overlap, one would expect to see less approach behavior, and consequently less hyperactivity/impulsivity, but more difficulty with attention. Put differently, low effortful control, and/or high approach, in the presence of normal-range negative affectivity, would predispose a child to ADHD, whereas low effortful control with high negative affectivity would lead to either anxiety or ADHD+anxiety. In this regard, what differentiates ADHD+anxiety from pure ADHD is higher negative affectivity in the former (Nigg, 2006).

Models of anxious temperament based upon behavioral inhibition map less well onto ADHD+anxiety. Whereas anxious children are more likely to have a behaviorally

inhibited temperament, children with ADHD are better characterized by their behavioral disinhibition. Moreover, as reviewed earlier, temperament alone does not account for the presence or absence of anxiety (or any other) disorders. Thus, among children with ADHD, environmental factors may determine the extent to which temperamental predispositions, common or uncommon to ADHD, will lead to anxiety.

In summary, research suggests that ADHD+anxiety is not accounted for by genes, and research on temperament and neurobiological correlates of ADHD and anxiety suggest that the two disorders can be overlapping as well as distinct, with their co-occurrence constituting an "additive" profile rather than one of its own. There may exist a biological predisposition that is common to both, marked by inhibitory control or poor top-down regulation of behavior and affect (and related prefrontal circuitry), which contributes to the comorbidity when interacting with environmental factors.

ADHD-Specific Factors

With some exception, (Wilens et al., 2002), ADHD typically precedes the diagnosis of anxiety disorders in children with ADHD+anxiety by several years (Angold et al., 1999; Daviss, 2008; Jarrett & Ollendick, 2008; Taurines et al., 2010). Time course alone cannot on its own signify cause and effect, although it may provide helpful clues. By DSM-IV diagnostic requirements (American Psychiatric Association, 2000), the onset of ADHD is during early childhood (before the age of 7), whereas internalizing disorders usually do not emerge until later; although the DSM-V extended this age to 12 years, in most cases ADHD emerges well before then (American Psychiatric Association, 2013). This is significant given the extent to which anxiety is susceptible to environmental factors. Therefore, in addition to any potentially overlapping biological predispositions,

ADHD probably also increases vulnerability to developing later anxiety by its effect on external and environmental variables.

One way that ADHD affects the environment is via exposure to ADHD-specific remediations, such as psychostimulant medication or compensatory behaviors. Other environmental considerations are those that result from dysregulated or disruptive behavior in ADHD, and include poor academic performance, social interactions, and family relations, which over time could predict fragile self-concept or demoralization. The following section addresses the potential contributions of these factors to anxiety disorders by reviewing the [limited] respective literature.

ADHD Medication. Given that the first line of treatment for ADHD is stimulant medication, might this treatment make children with ADHD more prone to later anxiety disorders? Findings regarding this question have been mixed. In rodents, early treatment with methylphenidate has been suggested to increase later depressive-like symptoms (Carlezon, Mague, & Andersen, 2003), although such an effect has not been found in humans (Staikova, Marks, Miller, Newcorn, & Halperin, 2010). Some clinicians are hesitant to prescribe stimulants for children with anxiety out of concern for anxious side-effects. However, there is no real evidence that stimulants cause anxiety or depression in humans, and some have argued that, if anything, stimulants may prevent against, and might even improve, anxious and depressive symptoms in children (Daviss, 2008; Gurkan et al., 2010). One recent study of the effects of stimulant medication in children with ADHD and internalizing comorbidity reported significant reductions in symptoms of anxiety and depression, and improvements in quality of life ratings, after only three months of methylphenidate (Gurkan et al., 2010). A longer-running, 10-year follow-up

study tracking children through early adulthood also found stimulant treatment to protect against the development of anxiety and depressive disorders (Biederman, Monuteaux, Spencer, Wilens, & Faraone, 2009). If it is confirmed that medication protects against anxiety and depression, a plausible explanation would be that amelioration of ADHD symptoms reduces risk. Were this to be the case, it would follow that ADHD symptoms are at least partially responsible for increasing risk in the first place.

Anxiety as Compensatory in ADHD. It might be that anxiety serves to protect against, or compensate for, some of the deficits resulting from ADHD. For example, checking behaviors may be considered adaptive among individuals who have routinely misplaced belongings, forgotten items or steps required for a task, or committed careless or impulsive errors. Individuals doing so may be less likely to repeat such mistakes. At the same time, they may also become more prone to negative internalizing symptoms, like checking *too* frequently, or worrying about not having checked enough.

The inhibitory quality of anxiety could also serve as a buffer against behavioral impulsivity in children with ADHD. For instance, among children who have been rejected due to verbal or physical impulsivity, some anxiety in future interactions might prevent against such acts in the future, therefore serving an adaptive function. On the other hand, too much worry has detrimental effects. Studies employing cognitive tasks to assess the role of anxiety in inhibitory abilities have yielded conflicting results, with some reporting improved performance on inhibitory tests (Manassis, Tannock, & Barbosa, 2000; Oosterlaan, Logan, & Sergeant, 1998; Tannock, 2000), and others reporting greater impulsivity (March et al., 2000).

From an attentional perspective, some have suggested that anxiety aids children with ADHD on tasks of sustained attention (Vloet, Konrad, Herpertz-Dahlmann, Polier, & Günther, 2010), and one might suppose that a child who struggles to remain alert in such settings could benefit from the increased level of arousal that anxiety instills. However, too much anxiety impairs attention, and research demonstrates poorer sustained attention and working memory abilities among children with ADHD+anxiety relative to children with ADHD alone (Tannock, 2000; Tannock, Ickowicz, & Schachar, 1995). Therefore, it is possible that low-level anxiety may serve a compensatory function, but levels beyond that further impair individuals with ADHD.

Demoralization in ADHD. Young children with ADHD are notorious for acting without thinking, behaving recklessly, talking out of turn, performing below their academic potential, and struggling socially to a greater extent than their non-ADHD peers; as a result, they are more likely to be disciplined, to receive poor grades in school, and to be rejected by their peers (DeWolfe et al., 2000; G. J. DuPaul et al., 2001; Hinshaw, 1992a, 1992b). Therefore, one way that ADHD symptoms could conceivably lead to anxiety and depression is through demoralization, resulting from ADHD-related deficits. Little research has been conducted on the potential for demoralization to lead to clinical levels of internalizing pathology. Biederman and colleagues reported that depression was not correlated with ADHD symptom severity and impairment among a sample of depressed children with ADHD, and concluded that therefore demoralization was not responsible for the emergence of depression (Biederman, Mick, & Faraone, 1998). However, demoralization entails far more than ADHD symptom severity, and is more about one's perception of the impairment than about the symptom count, itself. For

instance, whether or not a potentially demoralizing event is experienced as demoralizing may depend on one's cognitive style.

Cognitions in Children with ADHD

Cognitive biases and distortions were discussed earlier as contributing to anxious symptoms and psychopathology. Repeated exposure to demoralizing situations or negative feedback may pave the way for the development and maintenance of negative self-schemas or maladaptive or distorted cognitions. For instance, a child who struggles to focus on studying or to complete schoolwork on time, and who receives poor marks or is reprimanded for it, is prone to developing negative cognitions (e.g., "I can't _____") and beliefs (e.g., "it is impossible for me to do well in school"). Faced with activating events (relevant triggers, such as an upcoming test, social gathering, or long-term project), these thoughts become reactivated, giving way to feelings of worry (e.g., fear of failing an exam, appearing stupid, disappointing parents, etc.), and perpetuating the negative cognitions. Negative or potentially demoralizing events are likely to interact with cognitive style to contribute to or maintain anxiety. Thus, the same disappointing event (for instance, receiving a poor grade) is experienced differently by a child possessing the above cognitive style (hopelessness and worry: "There's no point in trying, I always do badly;" "I'll never get into college") than by another child (disappointment: "that was a really hard test;" "next time I will study harder so I can do well"). In this way, impairment associated with ADHD symptoms may lead to negative cognitive styles, giving way to anxious feelings down the line.

Environmental Exposure in Children with ADHD

As argued earlier, parenting is overarching and inextricably linked to all other aspects of the environments of young children. Therefore, the review of environmental exposure included below is limited to that which directly pertains to parenting, as was done earlier, in reviewing anxiety.

Parenting in Children with ADHD

As Johnston and Mash put it (Johnston & Mash, 2001), "The stressful, demanding, and intrusive nature of the child's ADHD characteristics are likely to evoke negative reactions from other family members and to exert a disruptive influence on family relationships and on the psychological functioning of parents. Thus, in this commonly accepted model, difficulties in families of children with ADHD are seen as driven by, and secondary to, characteristics of the disorder" (p.185).

Young children with ADHD are often disinhibited, impulsive, reckless, overly active, and difficult to parent. They struggle with, and are often resistant to, tasks and activities that are taxing on attention (such as learning-based tasks) or otherwise boring (like chores). They can be messy and disorganized, placing increased burden on those around them. Forgetfulness and difficulty following instructions adds to this, as children may need to be told repeatedly to do things. Routines, like morning routines, are challenging due to distractibility and disorganization, and bedtime routines may be made more challenging by difficulty settling down to sleep. It is not surprising that researchers consistently report higher levels of stress (and distress) among parents of children with ADHD than parents of their typically developing peers (Deault, 2009; Gerdes et al., 2007; Johnston & Jassy, 2007; Johnston & Mash, 2001), as well as increased negative,

directive, and controlling behaviors, decreased social interaction, and decreased positive parenting and contingency setting (Clerkin, Marks, Policaro, & Halperin, 2007; Deault, 2009; Johnston & Mash, 2001).

Questionnaires completed by parents of clinic-referred children and adolescents with ADHD are reflective of poorer parenting practices, including less authoritative and less consistent parenting style, and greater tendency to use excessive control (Cunningham & Boyle, 2002; Deault, 2009; Ellis & Nigg, 2009; Hinshaw, Zupan, Simmel, Nigg, & Melnick, 1997; Johnston & Mash, 2001; Shelton, Frick, & Wooton, 1996). Questionnaires reveal family environments that are high in stress and conflict, relative to families of typically-developing children and adolescents (Biederman et al., 1999; Brown & Pacini, 1989; G. J. DuPaul et al., 2001; Gadow et al., 2000; Scahill et al., 1999).

Observational studies also show that parents of children with ADHD are more directive and less warm than parents of children without the disorder, while children with ADHD are also less compliant and more negative (Barkley, Karlsson, & Pollard, 1985; Cunningham & Barkley, 1979; G. J. DuPaul et al., 2001). This seems to be especially true among younger children (Barkley, Karlsson, & Pollard, 1985; G. J. DuPaul et al., 2001; Mash & Johnston, 1990). High activity levels may contribute more risk to family functioning than inattention, according to some studies (Colder, Lochman, & Wells, 1997; Lewis, 1992), although there is evidence that inattention contributes as well (Fergusson, Lynskey, & Horwood, 1997).

In support of the hypothesis that ADHD symptoms give way to parent behavior and not the other way around, studies have demonstrated that altering children's ADHD-

like behaviors directly predicts changes in parenting. One unique study employed child confederates to complete structured tasks with adult female participants; children were instructed either to engage in on-task behavior or to pretend to be distractible (Ianna, Hallahan, & Bell, 1982). Study participants interacting with the latter group of children were observed by raters to be more demanding and controlling than those interacting with those who were more on-task.

Other studies have compared parent-child interactions when children were on- and off-medication. Humphries, Kinsbourne, and Swanson (1978) and a number of studies by Barkley and colleagues of children at ages 5, 6, 7, 8, and 9 (Barkley, 1988, 1989; Barkley & Cunningham, 1979; Barkley, Karlsson, Pollard, & Murphy, 1985; Barkley, Karlsson, Strzelecki, & Murphy, 1984) reported improved child compliance, along with increased parental positive acknowledgment of compliance and decreased parental directiveness following administration of stimulant medication. Consistent with this, children who were considered responders to stimulant medication were found to demonstrate typical-seeming interactions with their parents following medication, whereas parent-child relations in nonresponders did not improve (Schachar, Taylor, Wieselberg, Thorley, & Rutter, 1987). A much more recent study of methylphenidate and clonidine reported improved quality of life among the families of children with ADHD as a result of treatment (Cannon et al., 2009). Finally, the finding that not only behavioral treatment (parent training), but medication as well, predicted decreases in negative/ineffective discipline in the MTA study (Wells et al., 2000), supports the hypothesis that child dysregulated behavior drives parenting differences, whereas

remission of such behavior disturbances may lead to normalization (or at least improvement) of parenting.

The extent to which these effects are attributable of ADHD, specifically, as opposed to comorbid disruptive behaviors, which are prevalent among children with ADHD, has been disputed. Poor parenting clearly leads to aggression (Deault, 2009; Johnston & Mash, 2001). Many studies that have assessed differences in parenting among school-aged children and adolescents report a stronger association between negative family and parenting factors and conduct problems than ADHD (Deault, 2009; Johnston & Mash, 2001). However, it appears that children with ADHD and comorbid ODD or CD experience more negative parenting and family context than children with ADHD, only, who experience more of this than children without externalizing disorders (Gomez & Sanson, 1994; Johnston, 1996; Johnston, Murray, Hinshaw, William, & Hoza, 2002; Pfiffner et al., 2005; Seipp & Johnston, 2005; Tripp, Schaughency, Langlands, & Mouat, 2007). Nevertheless, even among observational studies, which have the benefit of being unaffected by parent or child rater biases, results are varied. For instance, in one study, parents of children with ADHD, without ODD or CD, were observed to employ more "rejection-coercion" and "inconsistent parenting" than those of typically developing (TD) children, while parents of children with ADHD reported their families were still more "cohesive" than families of children with disruptive behavior (Lindahl, 1998). In contrast, another observational study reported that, among boys with ADHD, child conduct problems were linked to maternal responsiveness and sensitivity, whereas ADHD symptoms were not (Johnston et al., 2002). At present, cross-sectional studies

can be said to provide moderate support for an association between ADHD and parenting, irrespective of disruptive behavior.

Importantly, prospective studies have suggested that poor parenting predicts disruptive behavior, whereas the reverse is true of ADHD (Deault, 2009; Frick et al., 1994; Johnston & Mash, 2001). In one study, Shaw and colleagues found that children who presented with ADHD+ODD/CD at age 6 were more likely than their peers who were typically developing or who had ADHD-only to have had parents at age 2 who were more rejecting (and depressed; D. S. Shaw, Owens, Giovannelli, & Winslow, 2001). Similarly, after controlling for earlier levels of ADHD and ODD, August and colleagues found parents' disciplinary style and family adversity (as well as psychiatric history) to predict persistence of childhood ODD, whereas parenting factors did not predict the course of ADHD (August, Realmuto, Joyce, & Hektner, 1999). However, not all longitudinal studies have reported this pattern; for instance, one study of older children, that commenced at age 7-12 and followed children until age 17, reported influences of ODD on parenting, minimal influences of parenting on ODD, and none of parenting on children's externalizing symptoms, (Burke, Pardini, & Loeber, 2008).

It is research conducted among younger children that particularly lends support for the relationship between parenting and ADHD, with or without comorbidity. First of all, ADHD symptoms appear to be more strongly associated with parenting practices early in development (Cunningham & Boyle, 2002; Mash & Johnston, 1990). Second, in accordance with longitudinal studies reviewed above, it is likely that parenting practices at least partially underlie the development of ODD and CD, particularly among young children. In this sense, disruptive behavior and poor parenting probably reinforce each

other over time. This may also partially account for findings that parenting practices are especially anomalous among older (as opposed to younger) children with ADHD and comorbid ODD or CD (Gomez & Sanson, 1994; Johnston, 1996; Johnston et al., 2002; Pfiffner et al., 2005; Seipp & Johnston, 2005; Tripp et al., 2007).

Together, studies suggest that children with ADHD are more likely to have parents who practice negative discipline styles, and that these parenting practices give way to oppositional and conduct problems later on (Taylor, Chadwick, Heptinstall, & Danckaerts, 1996). Moreover, conduct problems and parenting probably go on to reciprocally influence one another, increasing the strength of the relationship over time (Burke et al., 2008; Deault, 2009; Johnston & Mash, 2001) and potentially accounting for why disruptive behavior disorders are more strongly associated in the literature with parenting than ADHD. In support of this pathway, a longitudinal study by Fergusson, Lynsky, and Horwood (Fergusson et al., 1997) reported that higher levels of inattention in 8 year-old children were associated with higher rates of family problems, and that it was a combination of child conduct and family problems that predicted outcome. Shaw et al. (D. S. Shaw et al., 2001) found more rejecting parenting at an early age to predict later ODD/CD, above and beyond ADHD. Other data have also indicated that negative parenting leads to child aggression only among children who are high in hyperactivity (Colder et al., 1997), suggesting an interaction of factors that increases risk.

Together, research supports the hypothesis that ADHD influences parenting practices. Studies that have been reviewed in this section also serve to illustrate one mechanism by which ADHD may lead to the development of comorbid psychopathology, in this case, disruptive behavior disorders. One route by which this occurs is via ADHD

triggering harsh and negative discipline in parents (Johnston & Mash, 2001). This model closely resembles the IPH, wherein ADHD affects parenting, which contributes to risk for subsequent psychopathology. A second pathway that has been posited to explain the comorbidity of ADHD with ODD and CD holds that inattention and impulsivity may make children more susceptible to the inconsistent discipline and poor parental monitoring of parents of children with conduct problems (Deault, 2009; Johnston & Mash, 2001).

Parenting in Children with ADHD+Anxiety.

Given that parenting and family correlates of ADHD – increased parental control, rejection, stress, inconsistency, and family conflict – correspond to those that pose risk for anxiety disorders, does this partially account for high rates of comorbidity among the disorders? Little research has been conducted to date to address this question. No studies have examined the relationship between these three variables over time, making it impossible to assess causality. However, cross-sectional studies have recently begun to investigate the interplay among ADHD, parenting, and anxiety.

Kepley and Ostrander (2007) obtained, from a community sample of over 7,000 children, 80 children with ADHD, 18 with ADHD and anxiety (GAD, separation anxiety, or social phobia), and 103 typically developing children, in grades 1-4. Almost half of the originally identified children with ADHD met criteria for either a Diagnostic Interview for Children and Adolescents- Revised -Parent Version (DICA-R-P)-based anxiety disorder diagnosis *or* a Worry scaled score of at least 12 on the Revised Children's Manifest Anxiety Scale (RCMAS). However, only 9% of the ADHD children met criteria for both, which the authors conservatively required for inclusion in the

"anxious ADHD" group. Children who presented with no more than minimal anxiety (absence of DSM-IV diagnosis *and* Worry scaled score below 10) were included in the non-anxious ADHD group; children who fell in between were excluded. Similarly rigorous inclusion criteria were observed in selecting children for the ADHD group, which required positive diagnostic interview as well as severely elevated ratings of hyperactivity (2.5 standard deviations above the mean). Mothers completed psychiatric interviews regarding their own symptomatology and completed the Family Environment Scale (FES) and the BASC-Parent Personality Profile (BASC-PPP).

Families of children with ADHD, with and without anxiety, were characterized by greater family conflict than families of control children. Families of ADHD-only and ADHD+anxiety did not differ from each other on family cohesion or expression as measured by the FES. Parents of anxious ADHD children were insignificantly higher on self-ratings of BASC-PPP parental control than were non-anxious ADHD children, who were not significantly higher than control children, but the difference between anxious ADHD and control children was significant. Anxious children also had significantly lower FES independence scores than did their non-anxious or control counterparts. Parents of both groups of ADHD children reported personally experiencing more depression, communicating less, and feeling less able to cope with their parental role than did mothers of control children. Notably, there were no differences between groups in rates of ADHD, anxiety, or depression among mothers, although mothers of anxious ADHD children were more likely to abuse substances than were mothers of control children. The authors concluded that children with comorbid ADHD and anxiety have families that are insular, dependent, and discouraging of autonomy.

Pfiffner and McBurnett (2006) assessed 143 clinic-referred children with ADHD between the ages of 5 and 11. Forty-five percent of the sample met criteria for an anxiety diagnosis as ascertained using a psychiatric interview (all DSM-IV anxiety disorders were included, with the most prominent being simple or social phobia (38%), separation anxiety (12%), and GAD (11%)). In line with Chorpita and Barlow's (1998) model of anxiety development the authors focused on two dimensions of parenting. "Sensitivity/consistency," was assessed using the Positive Parenting subscale of the Alabama Parenting Questionnaire, completed by mothers. "Control" was addressed using the Possessiveness subscale of the Parent-Child Relationship Questionnaire (PCRQ), completed by mothers and children. To account for the effects of anxiety and depression in mothers, mothers were also interviewed about their own psychiatric symptoms.

Anxiety in children was associated with significantly higher PCRQ Possessiveness ratings, based on parent and child reports, as well as lower scores on APQ Positive Parenting (on parent ratings). This is consistent with the notion that increased parental control and decreased warmth and/or positive contingency management are associated with childhood anxiety. Maternal anxiety disorders were found to be associated with comorbid child anxiety, whereas maternal depression was not related to child anxiety. However, maternal anxiety was not found to moderate the association between parenting and child anxiety, nor did parenting moderate between maternal and child anxiety. These data therefore suggest that parenting correlates of anxiety among children with ADHD are the same as those for children without ADHD, and that the association extends above and beyond the mothers' anxiety.

The Pfiffner and McBurnett (2006) study had a number of strengths. First, it used standard diagnostic procedures within a sample of clinic-referred children with ADHD. It was also the only study to date to empirically test parental over-control as well as warmth/contingency management as they relate to theoretical literature on anxiety. Together, the findings of these two studies offer preliminary evidence for the role of parenting in the relationship between ADHD and anxiety, and provide incentive for further research. However, they both also have two notable limitations. First, the cross-sectional nature of the data collected precludes inferences regarding direction of influence and/or the temporal relations among variables. Second, the data on parenting style collected in both studies relied exclusively on subjective self-reports.

Summary and Objectives of the Current Study

The broad literature reviewed suggests that parenting may play a potent role in the relationship between ADHD and anxiety disorders, and preliminary data support this prediction (Kepley & Ostrander, 2007; Pfiffner & McBurnett, 2006).

In summary, children with ADHD are more likely to have parents who use negative parenting styles that are high in rejection, low in positive contingency, and high in control. However, to better determine the potential direction of causality (i.e., does ADHD lead to differences in parenting, the reverse, or both?), systematic longitudinal investigation is needed. Such investigation must also commence early enough in development to detect patterns that develop over time. Finally, more objective measures of parenting are preferable to questionnaires administered to parents and children, for reasons of rater biases.

The anxiety literature implicates similar parenting styles. However, research in this area relies on methodology marked by similar limitations, including retrospective self-report and/or cross-sectional design. Within the anxiety literature as well, longitudinal analyses, beginning in early childhood and using observational measures of parenting, will be necessary to more rigorously assess temporal patterns and potential causality.

Therefore, despite clear limitations, existing literature provides some support for the "intermediate parenting hypothesis" (IPH), which posits that early ADHD provokes negative parenting behaviors (high in rejection and overly restrictive or intrusive control, low in positive contingency management), which, in turn, contributes to risk for anxious psychopathology in children. In line with a transactional model of anxiety development over time (Vasey & Dadds, 2001), these factors may interact and reinforce one another over time, thereby making increasingly pronounced a child's anxious features, until they surpass the threshold for clinical cutoff, and hence, the comorbidity.

The objectives of the present longitudinal investigation were as follows: (1) replicate findings of increased incidence over time of anxiety disorders among children with ADHD, followed longitudinally, from age 3-4 years through age 8; (2) replicate and further describe parenting characteristics in parents of children diagnosed with ADHD at age 3-4, as compared with their non-ADHD peers, using subjective (i.e., self-report scales) as well as objective (i.e., direct observation) measures of parenting, and to see whether early ADHD was predictive of these differences over time; (3) replicate and further describe early parenting characteristics that place children at risk for later anxiety, employing the same measures of parenting within the same sample of children, and

assessing the extent to which they predicted anxiety disorders over time; and (4) investigate the extent to which parenting, as influenced by early ADHD, might play a mediating role in the relationship between early ADHD and later anxiety disorders. It was hypothesized that: (1) children with early ADHD would be at increased risk for developing anxiety disorders by middle childhood; (2) ADHD in early childhood would be predictive of parenting practices characterized by more control, less warmth, and less positive contingency management; (3) parenting characterized by more control and rejection and less positive contingency management would predict anxiety disorders in children, whether or not they also had ADHD; and (4) parenting practices would partially mediate the relationship between early ADHD and later anxiety disorders.

CHAPTER 2: General Methods

Participants

The participants in the present investigation were 200 children who were part of an ongoing longitudinal study examining risk factors for and the developmental course of behavior disorders in young children. Participants were ethnically and racially diverse, reflecting the urban region from which they were recruited. Among them, 60% were Caucasian, 12% were African American, 11% were of Asian descent, and 19% were of mixed racial background. 32% had at least one Hispanic parent. 43% of the Non-ADHD children and 37% of the ADHD sample were non-white ($\chi^2 = 0.69$, p = .471).

Children were initially recruited both from the community and via clinical referrals to achieve a target goal of an approximately 2:1 ratio of preschool children "At-Risk" for ADHD (AR group) to Typically-Developing preschoolers (TD group). After obtaining permission from preschool directors/principals in the urban area surrounding the college, parents of children ages 3-4 in local preschools received materials inviting their participation and asking that they complete the ADHD-RS-IV, Home Version (DuPaul, Power, Anastopoulus, & Reid, 1998) and provide consent for the School Version of the scale to be obtained from teachers. In addition, parents of children explicitly referred to the study via schools, pediatricians, or mental health workers for observed hyperactivity and attention problems were likewise asked to complete the scale and gather ratings from teachers. Based on the questionnaires, children were entered into one of two groups: (i) a TD group, indicating low risk for ADHD and defined by fewer than three of nine Hyperactive/Impulsive and fewer than three of nine Inattention symptoms (ratings of either "Often" or "Very Often") on both parent and teacher ADHD-

RS-IV ratings; or (ii) an AR group, defined by a total of at least six different symptoms ("Often" or "Very Often") of either Hyperactivity/Impulsivity or Inattention across the Home and School Versions of the ADHD-RS-IV. Those not meeting criteria for either group (17% of those rated) were excluded from the longitudinal study. Other exclusion criteria included evidence of Post-Traumatic Stress Disorder or an autism spectrum disorder, a diagnosed neurological condition, Full Scale IQ less than 80 (ascertained via WPPSI-III at baseline), or use of medication for any chronic medical or psychiatric disorder, including ADHD.

Parent and teacher rating scales were obtained for 529 children, 400 of whom met screening criteria. However, to meet our enrollment goal of 150 AR and 75 TD children, matching for sex (roughly 2:1 boy:girl ratio), families were enrolled on a rolling basis, after which enrollment was closed. In the end, we enrolled 216 children (140 AR and 76 TD). After baseline evaluations, children who did not return for either of the two subsequent annual assessments were considered lost to attrition (n=16, 7.4%). Those children who were lost to attrition did not differ significantly in age, sex, IQ, minority status, SES, or parent ratings of ADHD symptoms. However, they did have significantly higher levels of teacher-rated ADHD symptomatology. Thus, despite this one difference, the resulting sample was equivalent to that which was seen pre-attrition. In the end, there were 200 children: 96 with BL ADHD (8 inattentive type, 45 hyperactive/impulsive type, 43 combined type) and 104 without BL ADHD. Fifty-one children met criteria for ODD at BL, 43 of whom (84%) also met criteria for ADHD. Table 1 summarizes the sample characteristics.

Table 1

Sample characteristics

	Non-ADHD Mean (SD)	**ADHD** Mean (SD)
	n=104	n=96
Age	4.26 (.53)	4.35 (.47)
Sex (% male)	70	74
BL WPPSI-III IQ**	111 (12)	102 (13)
SES**	67 (17)	60 (19)
Race (% non-White)	43	37
BL ADHD-RS Home Score - IN / HI **	5.4 (4.5) / 6.7 (5.3)	13.8 (4.7) / 16.8 (5.2)
BL ADHD-RS School Score – IN / HI**	4.3 (5.4) / 4.3 (5.5)	15.0 (6.6) / 17.6 (7.0)

Note: BL = Baseline; SES = Socioeconomic status; ADHD-RS = ADHD-Rating Scale; IN = Inattention; HI = Hyperactivity; BASC-2 = Behavioral Assessment Scale for Children – 2nd Edition; ***p*<.01.

Measures

Diagnosis and Symptom Severity

ADHD Rating Scale-IV. The ADHD Rating Scale – IV (ADHD-RS-IV) (G.J. DuPaul, Power, Anastopoulos, & Reid, 1998) Home and School checklists consist of the nine Inattention and nine Hyperactive/Impulsive items that comprise the ADHD symptom criteria in the DSM-IV (American Psychiatric Association, 2000). Parents and

teachers completed the checklist for all children at the time of screening and again at each subsequent evaluation. Each item is rated on a 4-point scale (0=never or rarely; 1 = sometimes; 2 = often; 3=very often). The ADHD-RS-IV has been used extensively with children as young as age 3 and has been shown to have good reliability and validity in preschoolers (McGoey, DuPaul, Haley, & Shelton, 2007). Within our sample, coefficient alpha for the home version was .908 for inattention and .914 for hyperactivity/impulsivity. For the school version, coefficient alpha was .945 for inattention and .949 for hyperactivity/impulsivity.

Kiddie-Schedule of Affective Disorders – Present and Lifetime Version. The Kiddie-Schedule of Affective Disorders – Present and Lifetime Version (K-SADS-PL) (Kaufman, Birmaher, Brent, Rao, & Ryan, 1996) is a widely used, semi-structured interview for diagnosing psychiatric disorders in children. Because this study was a part of a larger, longitudinal study of the developmental trajectory of behavior disorders, the K-SADS-PL was chosen over the preschool-specific tool, the Preschool Age Psychiatric Assessment (PAPA; (Egger & Angold, 2004)), so it could be consistently administered as children got older. The K-SADS-PL has received preliminary support in the literature for its reliability and validity in diagnosing preschoolers (Birmaher et al., 2009). The K-SADS-PL is based upon DSM-IV diagnostic criteria, thus allowing us to accurately screen for a range of symptoms and diagnoses. In the present study it was used to diagnose both ADHD and anxiety disorders.

Symptom endorsements and diagnoses according to DSM-IV criteria were formulated based on the K-SADS-PL interview, supplemented by teacher ADHD-RS ratings and behavioral observations during the child testing (Gopin, Healey, Castelli,

Marks, & Halperin, 2010). Because ADHD symptoms were particularly of interest, data on individual symptom endorsement were collected systematically. A parent-endorsed symptom on the K-SADS-PL was always counted, regardless of teacher and observer ratings; if a parent endorsed subthreshold features of a symptom (i.e., present but not impairing), endorsement by a teacher ("often" or "very often" on the ADHD-RS) or clinician was considered an endorsement of the symptom. Absence of a symptom according to parent report was overruled only when symptoms were strongly believed to be present, as based on clinician observation or a rating of "very often" on the ADHD-RS teacher form (Lahey et al., 1994). As specified in DSM-IV, evidence of cross-situationality of symptoms and impairment, as well as a persistence of symptoms for at least six months, were required for an ADHD diagnosis. All interviews were reviewed in supervision with a licensed psychologist, who provided oversight at an item-by-item level.

Parenting

Alabama Parenting Questionnaire – Preschool Revision. The Alabama Parenting Questionnaire – Preschool Revision (APQ-PR) (Clerkin et al., 2007) is a 32-item revision of the original APQ (Shelton et al., 1996); ten items from the original version were excluded due to their developmental inappropriateness in preschoolers. The psychometric properties of the preschool revision have been assessed elsewhere in preschoolers, ages 3 to 5 (Clerkin et al., 2007), wherein factor analyses revealed a three-factor solution (Positive Parenting, Inconsistent Parenting, Negative Parenting), similar to that which has been reported among older children (Wells et al., 2000). Items are rated on a 5-point scale, ranging from 1 (*never*) to 5 (*always*). Raw scores were used, as norms

were not available for this measure, and also because we continued to use the measure at Age 6 as well. The Positive Parenting index was of particular interest because of its focus on warmth and positive contingency management, and also because it is widely used and was employed in Pfiffner and McBurnett's (2006) study. Of the 12 items comprising this index, six pertain to contingency management (of which, five refer to praise, such as "you praise your child when s/he behaves well") and the remaining six reflect positive interactions (e.g., "you play games or do other fun things with your child") and support (e.g., "you help your child with his/her work"). Alpha values for Positive, Inconsistent, and Negative Parenting within our sample of children at baseline were .789, .709, and .521, respectively.

Parent-Child Interaction. The Parent-child interaction session (PCI) consisted of three five-minute segments, in accordance with an observation program published elsewhere (Healey, Gopin, Grossman, Campbell, & Halperin, 2010). The first five minutes were spent in "free play," in which children and parents were instructed to play with the toys in the room as they would at home (e.g., puzzles, puppets, bowling, cars, coloring, basketball, action figures). Second, the "structured task" consisted of a set of age-appropriate worksheets that parents completed at a table with their children. Finally, a second structured task asked the child and parent to sit as before, at the table, and for children to attempt to use Duplos to build three-dimensional structures shown in a book of photographs. For this task, parents were told that they could help their children verbally if they wished, but they were asked to refrain from manipulating the blocks themselves. Mothers were typically the parent to complete PCI sessions, with a few exceptions (see Procedure, below, for more detail).

Coding was completed separately for each of the three task conditions using a coding system that we have published elsewhere (Healey et al., 2010) and was based on one used by the NICHD Study of Early Child Care (NICHD Early Child Care Research Network, 1999, 2003, 2006) and augmented by Campbell, Pierce, March, Ewing, and Szumowski, (1994) and Eyberg, Bessmer, Newcomb, Edwards, and Robinson (1994). The coding system used has been widely employed as generating reliable and valid measures of individual and dyad behaviors during parent-child interaction (for review, see Healey et al. (2010)). For each task, four dimensions of parenting were rated on a 5-point Likert scale (1 = very low; 5 = very high) by trained coders, who demonstrated minimum inter-rater reliability of .70 (as measured by comparisons of the 15% of cases that overlapped) (Aspland & Gardner, 2003) and who received regular supervision to ensure that scoring did not drift with time. Raters were blind to the diagnostic status of children and were randomly assigned to portions of the videos to code. Between-rater ICCs generally ranged from .75 to .91. Parent codes included the following.

Emotionally Supportive Presence. This code measures warmth, emotional support, positive regard, and, related to contingency, praise. Very High ratings on this scale were reflective of a parent who appeared confident in the child's efforts, who redirected as needed while still maintaining confidence in the child, and who used positive feedback or praise of successes in order to help children through challenges; thus, such a parent was high in both emotional warmth and consistent reinforcement. On the opposite end, a parent who was Very Low was "aloof," either unavailable or hostile and rejecting when the child was in need of help.

Respect for Autonomy. Respect for Autonomy measures the extent to which the parent demonstrated respect for the child's individuality and perspectives. Parents Very High on this measure demonstrated acknowledgment of and respect for children's different perspectives, even when they expressed differing opinions, and even when parents insisted that activities be completed a certain way. Very Low ratings on this measure were reflective of complete denial of the child's individuality, wherein parents were intrusive, including by physical force.

Quality of Assistance. This scale measures the quality of structure and nonintrusive instruction during a joint task. Very High ratings reflected parental efforts to contribute to challenging children to improve mastery or sophistication, by asking stimulating questions or teaching children skills. Very Low ratings indicated either complete uninvolvement or failure to provide stimulating or constructive suggestions or assistance.

Parent Negative Affect. This scale assesses parental expressions of anger or rejection of the child. Very High ratings here were indicative of anger and hostility, which were highly reactive and barely controlled; such ratings also applied to parental rejection where it was used manipulatively, as a control technique. Very Low ratings indicated the absence of rejecting behaviors, although parents needed not demonstrate supportive presence.

Scores for Emotionally Supportive Presence (from here on also called Emotional Support) aligned with parenting dimensions of warmth as well as positive contingency management. In contrast, Negative Affect taps rejecting traits, in addition to manipulative and instrumental negative regard for the purpose of exerting psychological

control (for instance, by withdrawing love, instilling anxiety, or shaming; Barber, 2001). Respect for Autonomy is likened to the opposite of constraining parenting, another potential form of psychological control (Barber, 2001), and is a construct that has been shown to have a particularly strong association with childhood anxiety (McLeod, Wood, et al., 2007). Quality of Assistance is less clearly defined; it likely taps into emotional warmth as well as respect for autonomy, and perhaps even contingency management, through the provision of structure and expectation during the task.

Temperament

Child Behavior Questionnaire (CBQ), Parent Form. The CBQ (Rothbart et al., 2001) was administered to parents at baseline. The CBQ consists of 94 items, rated on a scale of 1 (extremely untrue of your child) to 7 (extremely true of your child). The measure has demonstrated good internal consistency and stability over time (coefficient alpha = .75, r = .63, respectively) (Rothbart et al., 2001), and is among the most widely used measures of temperament in young children. The scale generates 15 dimensions of temperament, with 14 of the 15 loading onto three factors: Surgency/Extraversion (approach and overall positive affect), Negative Affect (negative emotional reactivity), and Effortful Control (inhibitory control, or self-regulation). Surgency is comprised of Approach/Positive Anticipation, High Intensity Pleasure, Impulsivity, Smiling and Laughter, and the reverse score of Shyness. Negative Affect consists of Anger/Frustration, Discomfort, Fear, Sadness, and the reverse score of Soothability. Finally, Effortful Control includes Attentional Focusing, Inhibitory Control, Low Intensity Pleasure, and Perceptual Sensitivity. In line with the prediction that early

negative affect and effortful control would contribute to later anxiety disorders, these two scales were specifically of interest.

Temperament Assessment Battery for Children - Revised, Teacher Form. The teacher version of the Temperament Assessment Battery for Children – Revised (TABC-R, (Martin & Bridger, 1998)) is a 29-item questionnaire in which teachers rate children's behavior in the classroom. The measure yields four dimensions of temperament: Inhibition (tendency to withdraw in novel situations), Negative Emotionality (the tendency to become upset in frustrating situations), Activity Level (the tendency to engage in energetic activities), and Lack of Persistence (the converse of effortful control; the tendency to desist in tasks that are difficult). The measure has been shown to have good internal consistency (coefficient alpha = .86 - .95) and stability over time (r = .47 - .71). In line with the expectation that early behavioral inhibition would be a risk factor for later anxiety, this subscale was of interest. While at face value negative affect and lack of persistence appear to map onto negative affect and effortful control dimensions of temperament, the TABC-R probes specifically for these traits in the face of frustration or challenging cognitive tasks, and as such, represent different constructs from those studied in the anxiety literature and were omitted from the present study.

Socioeconomic Status

The Nakao-Treas Socioeconomic Prestige Index (Nakao & Treas, 1994) was used to assess socio-economic status (SES) at baseline. Both mothers' and fathers' scores were separately coded and the higher of the two scores was adopted as an indicator of family SES. High scores on this index are indicative of higher SES.

Procedure

Parents of children who met screening criteria for either the AR or TD group were invited for evaluation of eligibility. At this time, the nature and details of the longitudinal study was fully described to parents who then provided newly signed informed consent. The study protocol, including the informed consent form, was reviewed and approved by the Institutional Review Board of the College.

An evaluation to confirm eligibility was conducted (Baseline; BL) by a clinician who was blind to the child's initial group status. The K-SADS-PL screener and supplement items for ADHD and ODD modules were administered to parents. During this time, children were tested using the WPPSI-III. The majority of children completed the PCI session with their mother. In five cases, the PCI was completed with someone other than the mother: for three children the primary caretaker was the grandmother, and for two children, fathers' data were used because they accompanied the children at the initial and all subsequent evaluations. ADHD-RS and BASC-2 questionnaires were collected from parents and teachers, and the APQ-PR was collected from mothers (or, in three cases, by grandmothers who acted as the primary caregivers). Temperament ratings were also completed at baseline; the TABC-R by teachers and the CBQ by parents. DSM-IV symptom endorsements and diagnoses were formulated based on the K-SADS-PL interview, as described above. As specified in DSM-IV, evidence of cross-situationality of symptoms and impairment, as well as a persistence of symptoms for at least six months, were required for an ADHD diagnosis. At BL, 96 children met DSM-IV criteria for ADHD and 104 did not (fewer than 6 symptoms in both domains).

When children were five years of age (Age 5), the K-SADS-PL ADHD and ODD modules (screener and supplement items) were administered again. At Age 5 and Age 6, APQ-PR ratings were again collected from mothers, and PCI data were obtained during assessment sessions. We attempted to conduct the PCI with mothers again, in order to maximize consistency; however, in some cases mothers were unable to attend assessment sessions and fathers brought children in. In order to maximize available data over time, when fathers attended both follow-up sessions, their PCI data were used at Age 5 and Age 6, and the mothers' BL data were discarded (n=5). Where no caregiver completed the PCI more than once, mothers' BL scores were included and data were considered missing at Age 5 and Age 6. Parents and teachers again completed the ADHD-RS, and BASC-2. Finally, at Age 6, the full K-SADS-PL interview was administered to parents. Administration of the full K-SADS-PL enabled assessment for the anxiety disorders, as well as other psychiatric disorders.

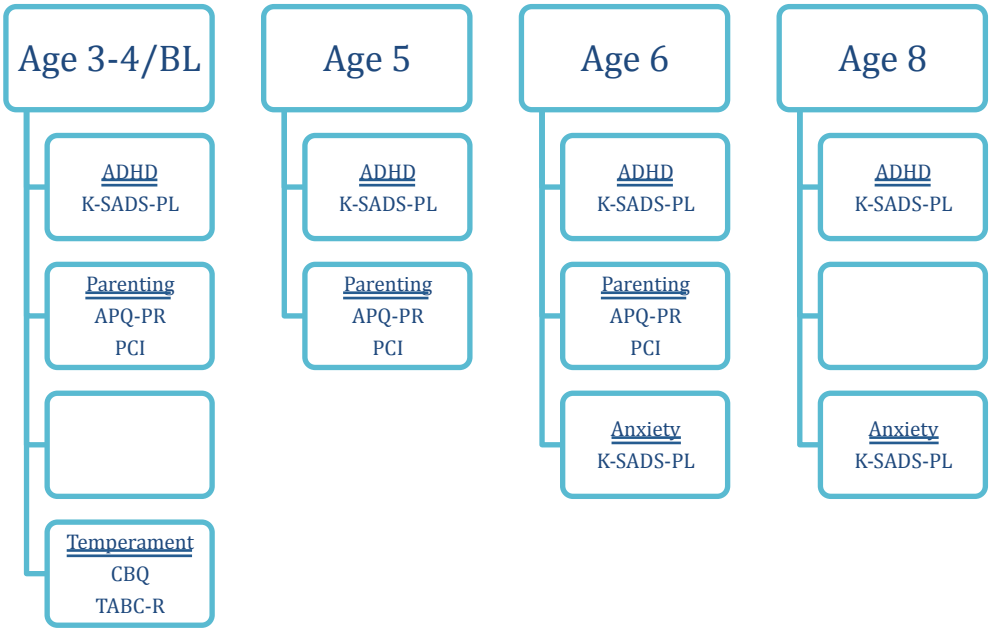
Children were included in the ADHD group if they met strict DSM-IV diagnostic criteria for any of the three subtypes, via ascertainment of at least 6 of 9 symptoms within at least one domain, with cross-setting impairment. An anxiety disorder was considered present if a child received a diagnosis of Generalized Anxiety Disorder (GAD), Separation Anxiety Disorder (SAD), Social Phobia (SP), or Obsessive-Compulsive Disorder (OCD). Anxiety Not Otherwise Specified (ANX-NOS) was also included as an anxiety disorder, in view of the tendency among young children, especially, for anxiety to manifest across a range of domains while not falling neatly into existing DSM-IV categories. In light of research suggesting that comorbid anxiety among children with ADHD is distinct from phobic-like anxiety present in phobias (Jensen et al., 2001; March

et al., 2000), and consistent with what has been done elsewhere (MTA Cooperative Group, 1999a, 1999b), children meeting criteria for a Specific Phobia were not included in the anxiety disordered group, unless they also met criteria for another anxiety disorder.

At Age-8, full K-SADS-PL interviews were again conducted with parents, and parents and teachers completed the ADHD-RS and BASC-2. Parenting measures were no longer collected beyond Age-6. Figure 1 demonstrates the flow of measures used at each year.

Figure 1

Data collected at each of the four assessment points



Note. BL = Baseline; K-SADS-PL = Kiddie-Schedule of Affective Disorders – Present and Lifetime Version; APQ-PR = Alabama Parenting Questionnaire – Preschool Revision; PCI = Parent-Child Interaction; CBQ = Child Behavior Questionnaire, Parent Form; TABC-R = Temperament Assessment Battery for Children – Revised, Teacher Form.

Chapter 3: Study 1: Does Preschool ADHD Predict Anxiety Disorder at Age 6 and/or 8?

Introduction

Study 1 was conducted to replicate findings of increased rates of anxiety disorders among young children with ADHD. It was predicted that ADHD during preschool would be associated with an increased incidence of anxiety disorders in middle childhood. The present dataset did not permit definitively testing whether the association between ADHD and anxiety would increase over time, simply because anxiety was not assessed prior to the age of 6 years. However, assessments of anxiety at Age 6 and again at Age 8 allowed for side by side comparisons of incidence rates at the earlier and later time points.

Method

Variables of Interest

Children were divided into two groups based upon their baseline diagnostic status: ADHD and Non-ADHD. Outcome variables were presence or absence of an anxiety disorder at Age 6 and Age 8.

Data Analysis

To test the hypothesis that early ADHD would predict increased risk for later anxiety disorders, two logistic regression analyses were run between the dichotomous variables: BL ADHD (yes/no) predicting Age 6 Anxiety disorder (yes/no), and likewise, BL ADHD predicting Age 8 Anxiety disorder. A supplementary analysis was conducted to assess the nature of the relationship more dimensionally; ANOVA was run comparing anxious versus non-anxious groups of children at Age 6 and Age 8 on BL ADHD symptomatology (ascertained via the K-SADS-PL, as described earlier).

Results

Table 2 summarizes the rates of anxiety disorders among the sample at Age 6 and 8. Ten children who had ADHD at baseline (13.3%) had at least one anxiety disorder at Age 6 (2 SAD, 2 SP, 3 OCD, 4 ANX-NOS), compared with seven (7.3%) of the Non-ADHD children (3 SAD, 3 SP, 1 ANX-NOS). Logistic regression results indicated that BL ADHD showed some evidence of predicting anxiety disorders at Age 6, but that this did not reach statistical significance (O.R. = 2.0, 95% C.I. = .7-5.4, p = .196).

At Age 8, 20 of the original ADHD children (27.8%) met criteria for an anxiety disorder (4 SAD, 4 SP, 8 GAD, 4 OCD, and 3 ANX-NOS) as compared to nine of the Non-ADHD children (10.2%; 0 SAD, 1 SP, 4 GAD, 0 OCD, 4 ANX-NOS). Logistic regression analyses indicated that BL ADHD was a statistically significant predictor of Age 8 Anxiety (chi-square = 8.281, p = .004), such that children with ADHD were more than three times as likely to have at least one anxiety disorder by Age 8 (O.R. = 3.4, 95% C.I. = 1.4-8.0, p = .006).

Table 2

Rates of anxiety disorders by ADHD status at ages 6 and 8

	Age 6				**Age 8**			
	BL No-ADHD n=96		BL ADHD n=75		BL No-ADHD n=88		BL ADHD n=72	
	n	%	n	%	n	%	n	%
SAD	3	3.1	2	2.7	0	0	4	5.6
SP	3	3.1	2	2.7	1	1.1	4	5.6
GAD	0	0	0	0	4	4.5	8	11.1
OCD	0	0	3	4.0	0	0	4	5.6
ANX-NOS	1	1.0	4	5.5	4	4.5	3	4.2
At least 1 anxiety disorder	7	7.3	10	13.3	9	10.2	20	27.8

Note. SAD = separation anxiety disorder; SP = social phobia; GAD = generalized anxiety disorder; OCD = obsessive-compulsive disorder; ANX-NOS = anxiety disorder, not otherwise specified.

Although not a primary aim, for descriptive purposes, Table 3 reports the rates of anxiety disorders as a function of ADHD status: absence of ADHD or one of the three subtypes. As can be seen, a relatively higher percentage of children with anxiety disorders at each time point had Combined Type ADHD than the other two subtypes.

Table 3

Rates of anxiety disorders by ADHD subtype at ages 6 and 8

	Age 6				**Age 8**			
	No-Anxiety n=154		Anxiety n=17		No-Anxiety n=131		Anxiety n=29	
	n	%	n	%	n	%	n	%
No ADHD	89	57.8	7	41.2	79	60.3	9	31.0
ADHD-PI	6	3.9	1	5.9	4	3.1	3	10.3
ADHD-HI	33	21.4	2	11.8	24	18.3	7	24.1
ADHD-C	26	16.9	7	41.2	24	18.3	10	34.5

Note. ADHD-PI = ADHD, Predominantly Inattentive Type; ADHD-HI = ADHD, Predominantly Hyperactive/Impulsive Type; ADHD-C = ADHD, Combined Type; Anxiety = presence of anxiety disorder; No-Anxiety = absence of anxiety disorder.

In secondary analyses, ANOVA comparing children with and without anxiety disorders at Age 6 on ADHD symptoms at BL revealed significant differences in BL K-SADS-PL inattention symptoms but not hyperactive/impulsive or total ADHD symptoms (see Table 4). Comparisons of children with and without anxiety disorders at Age 8, however, revealed significant differences across all three measures of ADHD symptoms.

Table 4

Differences in BL ADHD symptoms as a function of anxiety disorder status at ages 6 and 8

	No Anxiety Disorder M (SD)	**Anxiety Disorder** M (SD)	**F**	***p***
	Age 6 Anxiety Disorder			
Inattention	2.47 (2.82)	4.00 (3.14)	4.39	.038*
Hyperactivity/Impulsivity	3.51 (3.35)	4.41 (2.96)	1.13	.290
Total Symptoms	5.99 (5.84)	8.41 (5.60)	2.66	.105
	Age 8 Anxiety Disorder			
Inattention	2.44 (2.88)	4.28 (2.70)	9.92	.002**
Hyperactivity/Impulsivity	3.33 (3.32)	5.38 (2.81)	9.54	.002**
Total Symptoms	5.76 (5.87)	9.66 (4.97)	10.99	.001**

Note: ANOVA comparing children with and without anxiety disorders across baseline ADHD symptomatology; BL = Baseline; **p*<.05; ***p*<.01.

Discussion

As expected, diagnosis of ADHD at age 3-4 was associated with a significantly increased risk for later anxiety disorders. There was two-fold increased risk for anxiety disorders at Age 6 among children with BL ADHD, which did not reach statistical significance. At Age 8, children with BL ADHD were 3.5 times as likely to present with

at least one anxiety disorder, relative to children who did not meet criteria for ADHD at BL.

These findings were supported by dimensional comparisons as well. Children who had at least one anxiety disorder at Age 6 were found to present with more BL symptoms of inattention, but not hyperactivity/impulsivity, relative to children without an Age 6 anxiety disorder. Both inattentive symptom count and hyperactive/impulsive symptom count at BL were greater among children with Age 8 anxiety disorders than among their non-anxious counterparts. It is interesting that BL inattention was particularly salient among young children with anxiety disorders early on, particularly given the relatively low levels of inattention that characterize children this age, relative to hyperactivity/impulsivity (American Psychiatric Association, 2000). Given the small size of the sample at Age 6 (only 17 children met criteria for an anxiety disorder), interpretation of this finding is difficult. However, increased inattention is consistent with existing research on anxiety and sluggish cognitive tempo (Carlson & Mann, 2002; Skirbekk et al., 2011).

By Age 8, 28% of children with BL ADHD met criteria for at least one anxiety disorder, compared with 10% of children who did not meet criteria for ADHD at BL. This was more than double the rate of the comorbidity at Age 6 (13% of those with ADHD). Thus, these longitudinal data are consistent with the extant literature documenting a link between ADHD and anxiety disorders, particularly by middle childhood. The 28% rate of anxiety among children with ADHD is consistent with numbers reported elsewhere (e.g., (Jensen et al., 2001; Larson et al., 2011; Newcorn et al., 2001; S. R. Pliszka, 1992, 2000), and fall roughly between rates of community and

clinical samples. The 10% rate of anxiety among non-ADHD children may be somewhat high given the age of the sample, although it must be kept in mind that many of the children in the non-ADHD group entered the study as At-Risk and were therefore probably more likely to present with ADHD symptoms than if they had been screened as typically-developing.

These findings also further support the notion that ADHD is usually identified earliest, whereas the incidence of anxiety disorders increases during childhood. Based on our data, Age 8 was the first time at which the relationship between early ADHD and later anxiety was robustly documented, by which point more than one fourth of the children with ADHD displayed comorbid anxiety. However, the lack of diagnostic screening prior to Age 6 limits interpretation, given that it is conceivable (if perhaps unlikely) that the incidence of anxiety disorders early in development was initially higher than at Age 6.

Our sample size was not large enough to statistically test for group differences in *type* of anxiety disorders. However, some observations can be made. First, none of the children met criteria for GAD at Age 6, whereas, by Age 8, 12 children had GAD (4 without BL ADHD, and 8 with BL ADHD, respectively) and 4 had OCD. This is consistent with literature on disorder onset (Costello et al., 2005). Additionally, whereas none of the non-ADHD children met criteria for OCD at either time point, among the children with BL ADHD, 3 met criteria at Age 6 and 4 at Age 8 for OCD. This may be consistent with the notion that, relative to other anxiety disorders, OCD may be more closely associated with ADHD, in terms of neural etiology, inheritance, and relation to impulse-control (Fernando & Robbins, 2011; Sakolsky et al., 2012). Particularly high

rates of GAD among children with ADHD, as compared with other forms of anxiety, is consistent with other literature as well (Jensen et al., 2001; March et al., 2000; Molina et al., 2009). Also notable was that, among children with BL ADHD, the incidence of SAD increased between Age 6 and 8, whereas, among children without ADHD, it decreased to zero. Thus, while non-ADHD children outgrew these relatively normal-range, developmentally typical fears, for children with ADHD, these fears not only persisted, but became more prevalent with time, perhaps suggestive of additional factors perpetuating it. Future studies with larger sample sizes will benefit from addressing differential rates of individual anxiety disorders more systematically.

The present study had several limitations. For one, the method of screening for anxiety disorders was imperfect in two regards. As noted, children were not screened for anxiety prior to Age 6, the evaluation during which full K-SADS-PL interviews were conducted. It is therefore possible that group differences were missed prior to Age 6. This is somewhat unlikely, given that the incidence of anxiety disorders has been shown to increase as children age (Costello et al., 2005; Sakolsky et al., 2012), but that different incidence rates may have been present between the ADHD and Non-ADHD groups at BL cannot be ruled out. Nevertheless, given the finding that higher rates of anxiety emerged later in development, there is incentive for further investigation of environmental factors present in the interim.

Another limitation related to anxiety screening is that symptom information (i.e., on the K-SADS-PL) was obtained through parent- but not child-interviews. Although parents and teachers are typically considered the best informants of externalizing problems in children, it is possible for parents to be unaware of children's experiences of

internalizing troubles, and research suggests that parental and child report of anxiety are not always consistent (March et al., 2000). For this reason, interviews with children are particularly valuable when assessing anxious symptoms. While it might be predicted that parental observation of anxiety is a valid indicator of its presence, lack of observed anxiety cannot be assumed to mean its absence. Following this prediction, our reliance on parent report may have led to a higher rate of false negatives, missing some cases of anxiety. Similarly, more dimensional measures of anxiety might offer richer insight into the nature and severity of the disorders. Future study would benefit from more rigorous assessment of anxiety.

Altogether, the present study replicates findings of increased rates of anxiety disorders among children with very early diagnoses of ADHD, at ages 3-4. Whereas the association was not significant between ADHD at Age 3-4 and anxiety disorders at Age 6, Age 3-4 ADHD significantly predicted the presence of anxiety disorders at Age 8. Regardless of the etiology of this comorbidity, the period between age 3 and age 8 may be a particularly valuable time for children with ADHD, during which interventions may have the capacity to prevent against the emergence of anxiety disorders.

Chapter 4: Study 2: Does ADHD Predict Differences in Parenting?

Introduction

The aims of Study 2 were to: (a) replicate existing data demonstrating differing parenting practices among parents of preschoolers with and without ADHD, (b) assess, prospectively, whether preschool ADHD predicted any such differences over time, and (c) investigate whether comorbid ODD accounted for differences in parenting. It was predicted, first, that ADHD would be associated with more negative parenting practices, including rejection, poor use of positive contingency management, and excessive control. Second, it was predicted that differences in parenting between children with and without early ADHD would become especially pronounced over time, as the effects of children's behavior took an increasing toll on caregivers. Finally, it was predicted that ADHD would predict parenting even after accounting for ODD, which might become more strongly linked to parenting through a negative cycle of interaction over time.

Method

Variables of Interest

As described, children were divided into two groups based upon their BL diagnostic status of ADHD or Non-ADHD. Outcome variables for Study 2 were the subscales of the APQ-PR (Positive Parenting, Inconsistent Parenting, and Negative Parenting) and PCI (Emotional Support, Respect for Autonomy, Quality of Assistance, and Negative Affect) parenting measures, each of which was collected at BL (Age 3-4), Age 5, and Age 6.

In line with others using versions of the same PCI coding system, and in order to maximize reliability, composite scores were formed by summing individual dimensions

(Healey et al., 2010; NICHD Early Child Care Research Network, 1999). One previous study utilizing the same sample of children (Gopin, Healey, Grossman, Campbell, & Halperin, 2012) merged the four parenting scales into a single composite; however, because Emotional Support was not associated with any of our other key variables, (see Tables 5 and 14), the decision was made post-hoc to generate a composite score consisting of Respect for Autonomy + Quality of Assistance + [the inverse of] Negative Affect at each time point.

Data Analysis

To test the hypothesis that early ADHD would be associated with more negative parenting practices, three one-way MANOVAs were run, comparing BL ADHD versus Non-ADHD children across the seven parenting measures, as well as the PCI composite measure (Healey et al., 2010), at each time point (BL, Age 5, and Age 6). This relatively nonspecific design was selected given the somewhat exploratory nature of our research question: to investigate differences in parenting styles associated with ADHD. Unfortunately, the MANOVAs were insufficiently powered to detect differences, and as a consequence, multiple ANOVAs were used instead.

To investigate whether parenting practices would be related to ADHD symptoms dimensionally, correlational analyses were run. Specifically, Pearson product moment correlations were used to test the prediction that greater K-SADS-PL ADHD symptoms would be correlated with more negative parenting.

Previously-cited work within the same longitudinal sample of preschoolers with ADHD indicated a reduction in parent-rated Positive Parenting on the APQ-PR between the ages of 3-4 and when they were assessed one year later, at 4-5 (Clerkin et al., 2007);

thus, we expected this trend to continue at later follow-up. Reduced APQ Positive Parenting was also reported among parents of children with ADHD, albeit within a slightly older sample and using the school-age version of the scale (Pfiffner & McBurnett, 2006). Therefore, we expected to detect similar reductions in parent-rated Positive Parenting in this investigation.

With regard to observational (PCI) data, we predicted that parents of children with early ADHD would display higher levels of expressed Parent Negative Affect and lower levels of Emotional Support, both consistent with reduced warmth as well as reduced contingency management. In line with the prediction that parents of children with ADHD also exhibit restrictive and intrusive levels of control, we also expected Respect for Autonomy to be reduced among parents of ADHD children. As discussed earlier, high levels of Negative Affect might also reflect this construct as well. Finally, Quality of Assistance, as a less specific measure than the other three parenting subscales, was predicted to be lower among parents of children with BL ADHD.

To assess our secondary hypothesis that early ADHD would contribute, over time, to the parenting differences identified, hierarchical linear regression analyses were run. BL parenting measures were included as covariates, in order to assess whether ADHD predicted changes in parenting, above and beyond any pre-existing differences.

To address the question of whether disruptive behavior disorders might account for variance in parenting between ADHD and non-ADHD groups, ODD was included in regression analyses as an additional covariate. Because of the age of the sample, conduct symptoms were rare, and therefore CD was not included in the model. Finally, other potentially confounding variables (gender and SES) were also included as covariates.

Partial eta squared and R^2 estimates of effect size were interpreted according to commonly accepted standards (small = .01, moderate = .06, large = .14; Cohen, 1988).

Results

APQ-PR: Parent-Rated Parenting

ANOVA comparing APQ-PR dimensions in parents of children with and without BL ADHD revealed significant group differences in Positive Parenting at Age 5 and Age 6 (but not at 3-4). No group differences were found either for Inconsistent or Punitive Parenting. Table 5 shows the results of APQ-PR group-wise comparisons.

Table 6 summarizes correlational analyses between parent characteristics and BL K-SADS-PL symptoms of inattention and hyperactivity/impulsivity. As can be seen, Positive Parenting was again the only scale that was associated with ADHD symptoms (small to moderate effect size). Interestingly, Positive Parenting at all three time points was significantly correlated with symptoms of inattention, but not of hyperactivity/impulsivity.

Table 5

Differences in parenting measures as a function of BL ADHD status

		Non-ADHD M (SD)	ADHD M (SD)	F	*p*	η^2
APQ Positive Parenting	BL	52.82 (4.27)	52.33 (4.58)	.589	.444	.003
	5	53.52 (4.38)	51.71 (4.12)	6.816	.010*	.040
	6	53.06 (4.39)	50.97 (5.08)	6.066	.015*	.047
APQ Inconsistent Parenting	BL	15.63 (3.94)	15.66 (3.96)	.003	.960	<.001
	5	15.16 (3.74)	15.15 (3.55)	<.001	.993	<.001
	6	14.84 (3.30)	15.03 (4.14)	.080	.778	<.001
APQ Punitive Parenting	BL	9.67 (2.42)	9.72 (2.24)	.023	.880	<.001
	5	8.89 (2.17)	9.19 (2.01)	.831	.363	.005
	6	8.72 (2.04)	9.41 (2.77)	2.542	.113	.020
PCI Emotional Support	BL	4.18 (.82)	4.21 (.72)	.038	.845	<.001
	5	4.43 (.56)	4.27 (.55)	3.038	.083	.020
	6	4.53 (.52)	4.54 (.56)	.006	.940	<.001
PCI Respect for Autonomy	BL	3.34 (.62)	3.33 (.62)	5.410	.021*	.029
	5	3.32 (.48)	3.31 (.48)	10.543	.001**	.065
	6	3.58 (.40)	3.33 (.29)	2.462	.120	.024
PCI Quality of Assistance	BL	4.37 (.62)	4.13 (.79)	.010	.922	<.001
	5	4.46 (.62)	4.15 (.56)	.010	.921	<.001
	6	4.37 (.55)	4.18 (.69)	12.368	.001**	.111
	BL	1.17 (.32)	1.36 (.49)	9.412	.002**	.050

PCI	Negative Affect	5	1.14 (.33)	1.29 (.49)	4.771	.030*	.031
		6	1.10 (.22)	1.16 (.42)	.778	.380	.008
PCI	PCI Composite	BL	4.18 (.82)	4.21 (.72)	4.846	.029*	.026
		5	4.43 (.56)	4.27 (.55)	7.039	.009**	.045
		6	4.53 (.52)	4.54 (.56)	6.934	.010**	.065

Note: ANOVA comparing children with and without ADHD at baseline across parenting measures. BL = Baseline; APQ = Alabama Parenting Questionnaire, Preschool Revision; PCI = Parent-Child Interaction; PCI Composite = PCI Respect for Autonomy + Quality of Assistance - Negative Affect; **p*<.05; ***p*<.01.

Table 6

Correlations between parenting measures and BL ADHD symptoms

			HI		**IN**	
			r	***p***	**r**	***p***
APQ	Positive Parenting	BL	-.046	.521	-.157	.028*
		5	-.123	.120	-.190	.015*
		6	-.124	.168	-.238	.008**
APQ	Inconsistent Parenting	BL	-.008	.915	.022	.760
		5	-.026	.737	.056	.475
		6	-.063	.484	-.045	.617
APQ	Punitive Parenting	BL	.040	.571	-.019	.788
		5	.125	.110	.017	.825
		6	.173	.054	.022	.806

PCI Emotional Support	BL	.063	.397	.042	.579
	5	-.167	.039*	-.120	.141
	6	.049	.629	.065	.519
PCI Respect for Autonomy	BL	-.211	.004**	-.175	.019*
	5	-.239	.003**	-.211	.009**
	6	-.171	.087	-.107	.285
PCI Quality of Assistance	BL	-.049	.509	-.091	.224
	5	-.004	.959	.008	.924
	6	-.272	.006**	-.290	.003**
PCI Negative Affect	BL	.311,	<.001**	.218	.003**
	5	.229	.004**	.214	.008**
	6	.106	.292	.077	.447
PCI PCI Composite	BL	-.230	.002**	-.201	.007**
	5	-.221	.006**	-.194	.016*
	6	-.248	.012*	-.204	.040*

Note: Pearson product-moment correlations between symptoms of hyperactivity/impulsivity and inattention and parenting measures. BL = Baseline; APQ = Alabama Parenting Questionnaire, Preschool Revision; PCI = Parent-Child Interaction; PCI Composite = PCI Respect for Autonomy + Quality of Assistance - Negative Affect; HI = Number of hyperactive/impulsive symptoms ascertained on the K-SADS-PL at baseline; IN = Number of inattention symptoms ascertained on the K-SADS-PL at baseline; **p*<.05; ***p*<.01.

Hierarchical linear regression was run to determine the relative contribution of BL ADHD to later Positive Parenting, at Age 5 and Age 6, after controlling for BL Positive Parenting, ODD, gender, and SES. Results indicated that BL ADHD was a significant predictor of Age 5 Positive Parenting after accounting for these variables (see Table 7). Similar results were found at Age 6, such that early ADHD predicted persistently low Positive Parenting 2-3 years later (see Table 8). Thus, BL ADHD accounted for an additional 3.2% of the variance in reduced Positive Parenting scores, despite there being no significant group differences at the BL. Interestingly, ODD did not emerge as contributing significantly to Positive Parenting above beyond the other variables.

Table 7

Hierarchical linear regression of BL ADHD in predicting APQ Positive Parenting at Age 5

Variable	**B**	**S.E.**	**Sig.**	**R^2**	**R^2 Change**
1. BL ODD	.630	.768	.413	.012	-
Gender	-.356	.681	.601		
SES	.016	.017	.336		
2. BL Positive Parenting	.571	.072	<.001**	.296	.285
3. BL ADHD	-1.620	.650	.014*	.324	.027

Note: S.E. = Standard error; Sig. = Significance; BL = Baseline; APQ = Alabama Parenting Questionnaire, Preschool Revision; ODD = Oppositional Defiant Disorder; SES = socioeconomic status; ; *p<.05; **p<.01.

Table 8

Hierarchical linear regression of BL ADHD in predicting APQ Positive Parenting at Age 6

Variable	B	S.E.	Sig.	R^2	R^2 Change
1. BL ODD	1.166	.926	.210	.015	-
Gender	-.053	.779	.946		
SES	.020	.020	.307		
2. BL Positive Parenting	.631	.077	<.001**	.365	.350
3. BL ADHD	-1.909	.770	.015*	.397	.032

Note: S.E. = Standard error; Sig. = Significance; BL = Baseline; APQ = Alabama Parenting Questionnaire, Preschool Revision; ODD = Oppositional Defiant Disorder; SES = socioeconomic status; **p*<.05; ***p*<.01.

PCI: Observer-Rated Parenting

Table 5 includes the results from ANOVA comparisons of PCI data. ANOVA comparing children with and without BL ADHD on PCI scales revealed significant, if somewhat variable, group differences. Emotional Support did not differ at any of the time points between BL ADHD and non-ADHD groups. There was a significant group difference in Negative Affect at BL, such that parents of children with BL ADHD were rated higher on this dimension than parents of children without. This persisted at Age 5, but not at Age 6. A similar pattern was evident in ratings of Respect for Autonomy, such that the groups differed at BL and Age 5 but no longer by Age 6 (again, small to medium effect sizes). Specifically, children with BL ADHD had parents who demonstrated

poorer Respect for Autonomy concurrently and one year later. The opposite pattern occurred for Quality of Assistance, such that no group difference was evident at BL or Age 5, but by Age 6, parents of children with ADHD received significantly lower ratings of Quality of Assistance than parents of those without the disorder.

Correlational analyses between PCI scores and ADHD symptoms (see Table 6) again underscored the association between BL ADHD and early (BL and Age 5) parental Negative Affect and [inversely] Respect for Autonomy, as well as later (Age 6) Quality of Assistance, with both inattentive and hyperactive/impulsive symptoms demonstrating strong correlations. Curiously, Age 5 Emotional Support correlated significantly with BL hyperactive/impulsive but not inattentive symptoms.

As mentioned above, in computing the PCI Composite score, Emotional Support was omitted due to its poor association with ADHD (and anxiety disorders). Not surprisingly, the groups differed significantly on this measure at all time points (see Table 5). Both dimensions of BL ADHD symptoms were significantly correlated with the PCI Composite at all three time points (see Table 6).

Hierarchical linear regression analyses investigated the extent to which BL ADHD contributed to differences in PCI parenting scores, over time, by examining the extent to which ADHD uniquely predicted Respect for Autonomy and Negative Affect at Age 5, and Quality of Assistance at Age 6, after controlling for the aforementioned covariates. BL ADHD was found to account for roughly 3% of the variance in Age 5 Respect for Autonomy, after accounting for BL levels of this measure, BL ODD, gender, and SES (see Table 9). SES emerged as a significant predictor in this model as well;

higher SES was associated with higher levels of Respect for Autonomy. ODD did not emerge as a significant predictor in this model.

Table 9

Hierarchical linear regression of BL ADHD in predicting PCI Respect for Autonomy at Age 5

Variable	**B**	**S.E.**	**Sig.**	**R^2**	**R^2 Change**
1. BL ODD	.244	.125	.053	.128	-
Gender	.185	.101	.070		
SES	.008	.135	.004**		
2. BL Respect for Autonomy	.291	.063	<.001**	.248	.120
3. BL ADHD	-.233	.100	.022*	.277	.029

Note: S.E. = Standard error; Sig. = Significance; BL = Baseline; PCI = Parent-Child Interaction, Preschool Revision; ODD = Oppositional Defiant Disorder; SES = socioeconomic status; **p*<.05; ***p*<.01.

SES was also a significant negative predictor of Age 5 Negative Affect, and in this case, it displaced ADHD from the model. In other words, after accounting for differences in SES, BL ADHD no longer predicted increased levels of parental Negative Affect (see Table 10). ODD did not significantly contribute to this model.

Table 10

Hierarchical linear regression of BL ADHD in predicting PCI Negative Affect at Age 5

Variable	**B**	**S.E.**	**Sig.**	**R^2**	**R^2 Change**
1. BL ODD	.064	.083	.442	.138	-
Gender	-.013	.067	.852		
SES	-.006	.002	.001**		
2. BL Negative Affect	-.377	.076	<.001**	.273	.135
3. BL ADHD	-.001	.066	.990	.273	<.001

Note: S.E. = Standard error; Sig. = Significance; BL = Baseline; PCI = Parent-Child Interaction, Preschool Revision; ODD = Oppositional Defiant Disorder; SES = socioeconomic status; **p*<.05; ***p*<.01.

In contrast, BL ADHD emerged as the *only* significant predictor of Age 6 Quality of Assistance. Whereas none of the other covariates, including SES, ODD, or even BL Quality of Assistance, contributed significantly to the model, BL ADHD accounted for 9% of the variance in this measure (see Table 11).

Table 11

Hierarchical linear regression of BL ADHD in predicting PCI Quality of Assistance at Age 6

Variable	**B**	**S.E.**	**Sig.**	**R^2**	**R^2 Change**
1. BL ODD	-.083	.094	.378	.056	-
Gender	-.027	.083	.746		
SES	-.001	.002	.772		
2. BL Quality of Assistance	.081	.058	.167	.078	.022
3. BL ADHD	-.248	.081	.003**	.166	.088

Note: S.E. = Standard error; Sig. = Significance; BL = Baseline; PCI = Parent-Child Interaction, Preschool Revision; ODD = Oppositional Defiant Disorder; SES = socioeconomic status; **p*<.05; ***p*<.01.

Analyses of PCI Composite scores at Age 5 identified SES as a significant predictor, and after accounting for SES, ADHD was no longer significant in the model (see Table 12). Neither ADHD nor SES was a significant predictor of the Age 6 PCI Composite (see Table 13).

Table 12

Hierarchical linear regression of BL ADHD in predicting PCI Composite at Age 5

Variable	**B**	**S.E.**	**Sig.**	**R^2**	**R^2 Change**
1. BL ODD	.169	.217	.438	.162	-
Gender	.171	.175	.331		
SES	.014	.005	.002**		
2. BL PCI Composite	.357	.061	<.001**	.332	.170
3. BL ADHD	-.216	.174	.216	.340	.008

Note: S.E. = Standard error; Sig. = Significance; BL = Baseline; PCI = Parent-Child Interaction, Preschool Revision; ODD = Oppositional Defiant Disorder; SES = socioeconomic status; **p*<.05; ***p*<.01.

Table 13

Hierarchical linear regression of BL ADHD in predicting PCI Composite at Age 6

Variable	**B**	**S.E.**	**Sig.**	**R^2**	**R^2 Change**
1. BL ODD	-.399	.246	.109	.079	-
Gender	-.034	.214	.874		
SES	-.004	.006	.525		
2. BL PCI Composite	.156	.074	.038*	.127	.048
3. BL ADHD	-.002	.210	.299	.138	.011

Note: S.E. = Standard error; Sig. = Significance; BL = Baseline; PCI = Parent-Child Interaction, Preschool Revision; ODD = Oppositional Defiant Disorder; SES = socioeconomic status; **p*<.05; ***p*<.01.

Discussion

The findings of Study 2 support the hypothesis that preschool ADHD is associated with differences in parenting. As predicted, ADHD at ages 3-4 was associated with reduced APQ-PR Positive Parenting relative to Non-ADHD children by Ages 5 and 6. This developed over time, as the association was not significant at BL, and suggested less warmth and poorer contingency management among parents of children with ADHD. Also consistent with hypotheses, PCI data revealed that parents of children with ADHD at BL exhibited more rejecting/less emotionally warm *and* more over-controlling parenting styles while interacting with their children. Parents of children with ADHD provided less room for independence and were more controlling at ages 3-5, but not Age 6. However, by the time these children were 6, the instruction their parents provided was of lesser quality than that provided by their non-ADHD counterparts. As related to emotional warmth, more negative affect was expressed toward children with BL ADHD during BL and Age 5 assessments, but the difference was not significant by Age 6. Where group differences were significant, effect sizes generally clustered around the moderate range. In addition to comparisons using the dichotomous variable of diagnosis, dimensional analyses also showed that greater numbers of symptoms of both inattention and hyperactivity/impulsivity were correlated with poorer-quality scores parenting measures. A surprising finding was that observed Emotional Support of parents was not associated with children's ADHD. Given that this observational measure and the APQ-PR rating Positive Parenting both tap into parental warmth and contingency management/praise, it is unclear why this was the case.

An interesting pattern emerged in the relative contributions of ADHD, ODD, and SES to parenting data. ADHD at ages 3-4 emerged as the strongest predictor of later parent-rated positive parenting, after accounting for any preexisting differences in the same measure. It also accounted for a substantial 9% of the variance in observer-rated parental quality of support two to three years later (at Age 6). Along with early ADHD, SES contributed significantly to parental control, as measured by observations of parents' Respect for Autonomy at Age 5. Moreover, after accounting for SES, ADHD no longer contributed to the variance. Thus, along with ADHD, SES emerged as closely associated with observed parental rejection and control. On the overall Composite score, essentially a measure of parenting quality, SES again overtook ADHD. These findings are consistent with other research documenting poorer parenting quality among low SES families (Lempers, Clark-Lempers, & Simons, 1989; Newland, Crnic, Cox, & Mills-Koonce, 2013), and suggest that children with ADHD and low SES may be particularly at risk.

Perhaps more surprising were findings that comorbid ODD did not account for a more significant portion of the variance in any of the parenting measures assessed. 44.8% of children with ADHD at BL also met criteria for ODD, and therefore it was most likely not a sample size issue. It is notable that most research on the relationship between externalizing disorders and parenting has focused on the school-age period. Our dataset allowed us to investigate these factors earlier than most. These data therefore add to the limited existing studies of the preschool period, documenting clear associations between ADHD and parenting that is not accounted for by ODD, and supporting the hypothesis that the emergence of certain parenting practices among children with ADHD predate and

pave the way for later disruptive behaviors (Johnston & Jassy, 2007; Johnston & Mash, 2001)

A limitation of this study was that alpha level was not adjusted to offset potentially inflated Type I error during multiple comparisons. Interpretation of group comparisons should therefore be made with caution, and replication is needed among larger samples of children.

Despite general findings of rejection and control in parents of children with ADHD during the preschool years, one must also be cautious in drawing conclusions based on specific PCI data points, as there was substantial variability over time in which scores did and did not significantly differ from one another. Such variability is likely due to a number of factors, including (though not limited to) a small range of possible values in ratings, a limited sample size, and the potentially substantial cultural variation among a highly ethnically and racially diverse sample of families. Future studies would benefit from an increased range of ratings on PCI as well as from larger samples. Moreover, larger samples could allow for more sophisticated statistical procedures, such as random regression analysis, to study the trajectory over time; such a technique would better account for intra-individual differences, resulting in decreased standard error and potentially offering clearer and more robust patterns of results.

Another consideration pertains to the lack of information about parental psychopathology in the present study. Parental ADHD may contribute to negative parenting styles, including inconsistent parenting (Arnold, O'Leary, & Edwards, 1997; Chen & Johnston, 2007; Johnston, Mash, Miller, & Ninowski, 2012), and more generally, parental psychopathology may contribute to risk for more negative parenting (Johnston &

Mash, 2001). Given this, it is possible that parental pathology contributed to our findings. Moreover, because parents of children with ADHD are also more likely to have ADHD and other psychopathology (Humphreys et al., 2010), such contributions could potentially confound the results.

Therefore, the present study replicated findings of poorer parenting among young children with ADHD, and provided partial support for the IPH. Positive parenting was found to decrease from baseline, according to parent reports, which provides support for the idea that ADHD contributes, causally, to poorer parenting techniques. Significantly reduced quality of support, based on observational ratings, was found among children who had ADHD at baseline, relative to their same-aged peers; this also supports the hypothesis that early ADHD drives parenting. Observed parental negativity and constrictive parenting also differed between the ADHD and non-ADHD groups, but they were more associated with concurrent ADHD, and were not predictive of ADHD two to three years later.

As argued, there is a need for further investigation of the role of ADHD in the shaping of parenting behaviors. Longitudinal investigations in this study allowed for the study of these factors over time, to better determine causality. Given the bidirectional, transactional relationship between parenting and childhood externalizing disorders, studies of parenting must begin as early as possible, before patterns have set in. Such an approach is particularly necessary in order to best differentiate between parenting qualities that are attributable to children's psychiatric symptoms and those that contribute to them.

Chapter 5: Study 3: Does Parenting Style Predict Later Anxiety Disorders?

Introduction

Given the sparse literature prospectively studying the potential role of parenting in the development of anxiety disorders over time, there is a real need for longitudinal investigation. As reviewed earlier, most studies of the association between parenting and anxiety disorders are either cross-sectional or retrospective; thus, our longitudinal dataset provided an important opportunity to investigate these factors over time, in order to better assess the contribution of different parenting practices to children's development of anxiety.

Study 3 aimed to examine the relationship between parenting styles and anxiety disorders in children. Moreover, we were interested in which early parenting traits would be *predictive of* later emergence of anxiety disorders. As such, whereas we previously investigated the contribution of ADHD to parenting, in Study 3, parenting was studied for its potential contribution to anxiety disorders.

Method

Variables of Interest

For this study, independent variables included the previously described measures of parenting: the self-reported APQ-PR as well as the observer-rated PCI. As before, parenting at BL, Age 5, and Age 6 were considered. The outcome variable was dichotomous: the presence or absence of a diagnosable anxiety disorder, as determined by K-SADS-PL assessments at Age 6 and Age 8. Age 8 was identified in Study 1 as the point by which there existed a sizeable number of children who presented with anxiety disorders; it was also the age by which the incidence of anxiety disorders was associated

with significant group differences between BL ADHD and Non-ADHD children. Although it would have been desirable to include a continuous anxiety variable as well, symptom count was deemed inappropriate given that different anxiety disorders possess different numbers and types of symptoms, which would invariably skew comparisons.

Data Analysis

First, to investigate which parenting variables, if any, correlated with anxiety disorders, ANOVAs compared children with and without anxiety at Age 6 and Age 8 on each parenting measure across each of the three time points. This included each of the three subscales from the APQ-PR, the four from the PCI, and the PCI Composite score generated in Study 2. As in Study 2, MANOVAs were insufficiently powered, and as a consequence, multiple one-way ANOVAs were run; this was not ideal and may have inflated Type 1 error.

To investigate the extent to which the identified parenting variables contributed to the risk of children's anxiety disorders over time, hierarchical logistic regression was used. Because diagnostic interviews had not screened for anxiety disorders prior to Age 6, it was not feasible to control for earlier levels of anxiety in these models. Instead, measures of temperament were included, in line with theory that temperament represents an underlying disposition that [in concert with certain environmental factors] leads to anxiety disorders (Degnan et al., 2010; Lonigan & Phillips, 2001; Nigg, 2006). In accordance with the anxiety literature, three facets of child temperament were relevant: Negative Affect and Effortful Control, as rated by parents on the CBQ, and Inhibition, rated by teachers on the TABC-R. Because these represent theoretically distinct and independently well-studied predisposing correlates of anxiety disorders, they were

considered, together, the most representative temperamental risk factor for later anxiety. To condense the three temperament factors into one, z-scores were computed for each, and they were then added together, such that the sum of z-scores of Negative Affect, Inhibition, and the inverse (absolute value) of Effortful Control comprised a variable that was labeled, Risky Temperament. In addition to temperament, gender and SES were included in regression models as potential confounding variables.

Partial eta squared and R^2 estimates of effect size were interpreted according to commonly accepted standards (small = .01, moderate = .06, large = .14; Cohen, 1988).

It was hypothesized that parenting that was high in rejection and over-control, and low in warmth and contingency management, as evidenced by lower PCI Respect for Autonomy, Quality of Assistance, and Emotional Support, and increased Parental Negative Affect, would predict increased risk for anxiety disorders at Age 8, and that these associations would not have emerged by Age 6. Lower levels of APQ-PR Positive Parenting were also expected to be predictive of Age 8 anxiety disorders, by providing reduced warmth and positive contingency management.

Results

APQ-PR: Parent-Rated Parenting

ANOVA comparing APQ-PR data in parents of children with and without Age 6 Anxiety revealed no significant group differences. Table 14 depicts parenting as a function of group and time.

Table 14

Comparisons of parenting measures among children with and without Age 6 Anxiety Disorders

			No Anxiety M (SD)	**Anxiety** M (SD)	**F**	***p***	**η²**
APQ	Positive Parenting	BL	52.48 (4.39)	51.35 (4.89)	.991	.321	.006
		5	42.69 (4.44)	52.50 (5.24)	.024	.876	<.001
		6	52.06 (5.00)	51.36 (3.84)	.263	.609	.002
APQ	Inconsistent Parenting	BL	15.44 (4.00)	16.94 (4.19)	2.164	.143	.013
		5	15.08 (3.60)	15.25 (3.34)	.033	.856	<.001
		6	14.93 (3.69(	15.73 (3.45)	.627	.430	.005
APQ	Punitive Parenting	BL	9.61 (2.29)	10.59 (2.81)	2.651	.105	.016
		5	8.93 (2.20)	9.38 (2.68)	.643	.424	.004
		6	9.11 (2.42)	8.93 (2.69)	.069	.793	<.001
PCI	Emotional Support	BL	4.18 (.75)	4.52 (.76)	2.649	.106	.017
		5	4.34 (.58)	4.52 (.48)	1.315	.254	.010
		6	4.52 (.53)	4.53 (.59)	.002	.960	<.001
PCI	Respect for Autonomy	BL	3.35 (.62)	3.49 (.67)	.619	.433	.008
		5	3.32 (.49)	3.26 (.44)	.163	.687	<.001
		6	3.47 (.38)	3.50 (.28)	.065	.799	<.001
PCI	Quality of	BL	4.28 (.67)	4.49 (.60)	1.266	.262	.004
		5	4.33 (.62)	4.36 (.62)	.030	.862	.001

	Assistance	6	4.27 (.63)	4.30 (.53)	.027	.871	<.001
		BL	1.25 (.40)	1.05 (.12)	3.437	.066	.022
PCI	Negative	5	1.19 (.37)	1.19 (.55)	.002	.969	<.001
	Affect	6	1.13 (.34)	1.13 (.23)	<.001	.989	<.001
		BL	12.39 (1.28)	12.93 (1.04)	2.346	.128	.015
PCI	PCI	5	12.46 (1.07)	12.43 (1.19)	.009	.926	<.001
	Composite	6	12.60 (1.01)	12.67 (.75)	.042	.839	<.001

Note: BL = Baseline; APQ = Alabama Parenting Questionnaire, Preschool Revision; PCI = Parent-Child Interaction; PCI Composite = PCI Respect for Autonomy + Quality of Assistance - Negative Affect; **p*<.05; ***p*<.01.

ANOVA comparing APQ-PR data in parents of children with and without Age 8 Anxiety revealed significant group differences in Positive Parenting at BL and Age 5, such that parents of anxious children rated themselves significantly lower on items related to Positive Parenting (small to moderate effect sizes). This difference was no longer significant at Age 6. Table 15 depicts parenting as a function of group and time.

Table 15

Comparisons of parenting measures among children with and without Age 8 Anxiety Disorders

		No Anxiety M (SD)	**Anxiety** M (SD)	**F**	***p***	**η²**
APQ Positive Parenting	BL	53.00 (4.39)	51.14 (4.54)	4.073	.045*	.025
	5	53.32 (4.27)	50.88 (4.91)	6.284	.013*	.045
	6	52.63 (4.66)	50.94 (4.24)	2.014	.159	.019
APQ Inconsistent Parenting	BL	15.46 (3.92)	15.75 (4.33)	.123	.726	<.001
	5	14.80 (3.63)	15.72 (3.86)	1.274	.261	.009
	6	14.61 (3.72)	15.444 (4.46)	.701	.404	.007
APQ Punitive Parenting	BL	9.55 (2.05)	9.46 (2.05)	.037	.848	<.001
	5	8.99 (1.97)	8.80 (2.06)	.082	.776	.001
	6	9.13 (2.55)	8.67 (1.85)	.522	.472	.005
PCI Emotional Support	BL	4.23 (.77)	4.20 (.71)	.029	.866	<.001
	5	4.41 (.53)	4.33 (.59)	.390	.533	.003
	6	4.54 (.54)	4.53 (.62)	.004	.952	<.001
PCI Respect for Autonomy	BL	3.37 (.65)	3.31 (.64)	.136	.713	.007
	5	3.35 (.47)	3.21 (.48)	1.56	.214	.002
	6	3.46 (.39)	3.47 (.35)	.008	.930	.019
PCI Quality of	BL	4.32 (.69)	4.17 (.62)	1.08	.300	<.001
	5	4.33 (.61)	4.26 (.63)	.264	.608	.012

Assistance	6	4.34 (.54)	4.11 (.93)	1.65	.203	<.001
	BL	1.24 (.43)	1.25 (.37)	.010	.921	<.001
PCI Negative	5	1.15 (.34)	1.36 (.49)	5.99	.016*	.045
Affect	6	1.09 (.19)	1.13 (.67)	7.13	.009**	.079
	BL	12.45 (1.36)	12.24 (1.23)	.534	.466	.004
PCI PCI	5	12.53 (1.00)	12.11 (1.17)	3.077	.082	.024
Composite	6	12.71 (.76)	12.24 (1.67)	2.808	.098	.033

Note: BL = Baseline; APQ = Alabama Parenting Questionnaire, Preschool Revision; PCI = Parent-Child Interaction; PCI Composite = PCI Respect for Autonomy + Quality of Assistance - Negative Affect; **p*<.05; ***p*<.01.

Logistic regression was used to investigate whether Positive Parenting contributed to Age 8 anxiety disorders. To maximize the robustness of the variable, a composite, Overall Positive Parenting, was computed, which was the average of Positive Parenting at BL, Age 5, and Age 6. After controlling for Risky Temperament, which was, itself, predictive of Age 8 Anxiety, Overall Positive Parenting emerged as a small but statistically significant predictor of Age 8 anxiety disorders (see Table 16). Neither gender nor SES contributed significantly to the model.

Table 16

Logistic regression of Overall APQ Positive Parenting in predicting Age 8 anxiety disorders

Variable	**Odds Ratio**	**95% C.I.**	***p*-value**
Gender	1.55	.49-4.87	.458
SES	1.01	.98-1.03	.628
Risky Temperament	1.37	1.06 – 1.77	.017*
Overall APQ Positive Parenting	.86	.76 - .97	.012*

Note: BL = Baseline; APQ = Alabama Parenting Questionnaire, Preschool Revision; C.I. = confidence interval; SES = socioeconomic status; **p*<.05; ***p*<.01

PCI: Observer-Rated Parenting

ANOVA comparisons of PCI scores (four subscales and the composite) among children with and without Age 6 anxiety disorders revealed no significant group differences (see Table 14), and as a result, Age 6 anxiety disorders were subsequently excluded from further analyses.

Among the four PCI subscales, as well as the composite, the only factor that ANOVA identified as differing between children with and without Age 8 anxiety disorders was Parent Negative Affect. Parents of children with Age 8 anxiety disorders were rated as having significantly higher scores on Parent Negative Affect at Age 5 and

Age 6 than parents of non-anxious children (moderate effect sizes; see Table 14). The groups did not differ at BL. Comparisons of PCI variables are included in Table 15.

Logistic regression was run to investigate whether Parent Negative Affect contributed to Age 8 anxiety disorders. Again, to maximize the robustness of the variable, Overall Negative Affect was computed, averaging the scores at each of the three time points.

Table 17

Logistic regression of Overall PCI Parent Negative Affect in predicting Age 8 anxiety disorders

Variable	**Odds Ratio**	**95% C.I.**	***p*-value**
Gender	1.17	.35-3.95	.803
SES	1.01	.99-1.04	.411
Risky Temperament	1.43	1.10 – 1.85	.007**
Overall PCI Parent Negative Affect	3.96	1.14 – 13.76	.030*

Note: BL = Baseline; APQ = Alabama Parenting Questionnaire, Preschool Revision; C.I. = confidence interval; SES = socioeconomic status; *****p<.05; ******p<.01

Regression analyses including Risky Temperament, gender, and SES as covariates found Risky Temperament as well as Overall Parent Negative Affect to contribute significantly to Age 8 Anxiety Disorder. Specifically, Overall Negative Affect accounted for a four-

fold increased risk for Age 8 Anxiety, after controlling for the other variables. Gender did not contribute significantly, nor did SES (see Table 17).

Discussion

This study, conducted within a sample that was enriched for children at risk for ADHD, confirmed previous findings of reduced warmth and increased expression of negative emotionality among parents of anxious children, while failing to find an association with control, as measured by observations of parent-child interactions. As expected, parenting styles of parents of children with and without anxiety disorders at Age 8 differed in certain ways. However, contrary to predictions, parenting low in warmth (low self-rated Positive Parenting and high observer-rated Negative Affect) was associated with increased risk of Age 8 Anxiety, whereas measures of control (Respect for Autonomy and Quality of Assistance) were found not to differ significantly between the anxious and non-anxious groups.

This was a surprising finding given that studies have shown the reverse to be true (McLeod, Weisz, et al., 2007; Wood et al., 2003). This merits further investigation and replication, to better understand the aberrant finding that control was not related to anxiety. A larger sample and a broader score range on the PCI might yield group differences that reached significance within the Control domain. It is also possible, as described earlier, that Negative Affect during parent-child interactions may have tapped control-related factors (for instance, using rejection as a means of manipulation). Future investigation might also, therefore, benefit from more well-defined measures of control.

On the other hand, it is conceivable that within our sample, which was enriched for children at risk for ADHD and consequently included many children with

externalizing symptomatology, different factors may have been at play. Thus, in contrast to non-externalizing children, for whom parental control predicts anxiety, it is conceivable that among children with symptoms of ADHD, parental rejection plays a more prominent role in conferring risk for anxiety.

Related to this, different parenting practices may confer different risk, depending on the anxiety disorder in question. Whereas historically anxiety research has focused on parental over-control as exerting the greatest influence on child anxiety, more recent research indicates that parenting risk may vary by disorder (McLeod, Wood, & Avny, 2011). Emerging studies suggest that over-control confers nonspecific risk for anxiety disorders, but parental rejection and authoritarianism, as opposed to over-control, may be more predictive of GAD (McLeod et al., 2011; Shanahan, Copeland, Costello, & Angold, 2008). Given that GAD was the most common anxiety disorder in our sample (11% of children with ADHD met criteria for GAD at Age 8, which was twice the number meeting for any of the other anxiety diagnoses), this explanation is plausible. The predominance of worry-like anxiety disorders relative to phobic-related disorders among children with ADHD+anxiety is documented elsewhere (Jensen et al., 2001; March et al., 2000; Molina et al., 2009). Therefore, the evocation of parental negativity and rejection may confer risk among children with ADHD for developing GAD, in particular. Further investigation is needed to better understand differential risk of parenting practices among anxiety disorders, in children both with and without ADHD.

The Age 8 groups with and without anxiety disorders differed in Positive Parenting at BL and age 5, but not at Age 6. Although these data require replication, they

suggest that such parenting practices are particularly impactful during early childhood, a finding that has also been reported in neuroimaging (Rao et al., 2010).

A similar delay was observed in that parents of children with anxiety at Age 6 did not differ in parenting strategies at any point leading up to or concurrent with that time point. This may be attributable to the fact that there were few children with anxiety disorders at this time point. If replicated, however, this could be indicative of the nature in which parenting, over time, gradually exerts an increasingly negative risk on children for internalizing symptoms.

Because the longitudinal study from which this dataset was extracted was designed to investigate externalizing, and not internalizing disorders, the study design presented certain limitations. Perhaps the greatest limitation pertains to the less-than-ideal means of ascertaining information about children's anxiety. On one hand, the K-SADS-PL is a widely used and trusted means of collecting diagnostic information. However, while parents and teachers have been shown to be the most reliable informants when it comes to information about externalizing disorders, when it comes to internalizing disorders, parents may be less reliable informants (March et al., 2000). For purposes of anxiety disorders, it would have been preferable to have children's reports as well. As it was, clinicians' observations of anxiety during children's portions of annual assessments were incorporated into K-SADS-PL diagnoses, although generally speaking, the presence of worry during testing increased symptom endorsement, whereas its absence was unlikely to "cancel out" parents' report.

Relatedly, the absence of early diagnostic interviews (pre- Age 6) limits the extent to which parenting is conclusively regarded as causal in this study. To assert causality

would require that parenting contributed to anxiety at age 8 over and above pre-existing anxiety. In place of data on prior diagnostic status, temperament was used as a covariate. Although this decision was informed by the literature on temperament in anxiety, diagnostic status in preschool would have been preferable.

The nature of this sample, which includes a disproportionately larger number of children with behavior disorders than occurs in the general population, limits the generalizeability of the present results to a broader population. It is therefore possible that a more population-based (i.e., less-externalizing) sample would present with different parenting styles as linked and not linked to anxiety. As mentioned also in Study 2, a final limitation of Study 3 pertains to the statistical approach employed during multiple comparisons, in that alpha was not adjusted to offset potentially inflated Type I error.

In summary, the present study provided support for the IPH's prediction that negative parenting strategies contribute to later anxiety disorders among children. Parenting when children were 6 years of age was predictive of child anxiety disorders at Age 8, but not at 6, suggesting a causal process that occurs over time. However, the nature of the study design (i.e., the lack of diagnostic screening at baseline) precludes the ability to draw firm inferences regarding causality.

CHAPTER 6: Study 4: Does Parenting Mediate the Relationship Between ADHD and Anxiety?

Introduction

The IPH posits that parenting serves as an intermediary risk factor for anxiety disorders among children with ADHD. It proposes that one route by which children with ADHD are at risk is via the negative impact of their behavior on parents, whose practices, in turn, contribute to greater risk for anxiety disorders. Empirical data were outlined in Chapter 1, providing preliminary support for the hypothesis. Chapters 3-5 described a series of three studies providing further support for the pathway of development portrayed by the model.

To summarize, Study 1 showed that children with ADHD at age 3-4 were more likely to develop at least one anxiety disorder by age 8 than their non-ADHD peers. Study 2 provided evidence that more negative caregiving strategies are practiced by parents of children with ADHD at 3-4, and are characterized by more rejection (expressed negative affect), less positive contingency management, and more control (placement of restrictions upon autonomy). For two of the four parenting measures predicted by ADHD (positive parenting and quality of assistance), ADHD predicted continuing deterioration of parenting through age 6. Study 3 partially replicated research on negative parenting styles associated with anxiety disorders. Observations of greater negative and rejecting parenting, and parent-ratings of less positive parenting when children were 3 to 6 were predictive of anxiety disorders two to five years later, at age 8. Contrary to findings of others (McLeod, Wood, et al., 2007), over-controlling parenting practices were *not* found to predict later anxiety disorders. Rather, parental rejection and

poor contingency management were significant predictors, even after accounting for children's temperament.

To test the full IPH model, Study 4 investigated whether parental rejection and poor contingency management at ages 5-6 would play a mediating role in the relationship between ADHD at age 3-4 and anxiety disorders when children were 8 years of age.

Method

Variables of Interest

Baseline ADHD, Age 8 anxiety disorder, and the two parenting variables identified in Studies 2 and 3 (Positive Parenting and Parent Negative Affect) were the focus of the present study. In accordance with the time sequence proposed by the IPH, parenting scores collected at times in between, and not including, BL (the initial assessment of ADHD at ages 3-4) and Age 8 (the outcome measure of anxiety disorder) were specifically of interest. The average of Positive Parenting at ages 5 and 6 was calculated to generate Age 5-6 Positive Parenting, and the average of Parent Negative Affect at ages 5 and 6 was similarly computed and termed, Age 5-6 Parent Negative Affect. The latter was reverse-scored for ease of interpretation, so that the direction of the expected effect was consistent for both parenting variables. Therefore, for both Age 5-6 Positive Parenting and Age 5-6 Parent Negative Affect, high scores were reflective of desirable and low scores were reflective of undesirable parenting.

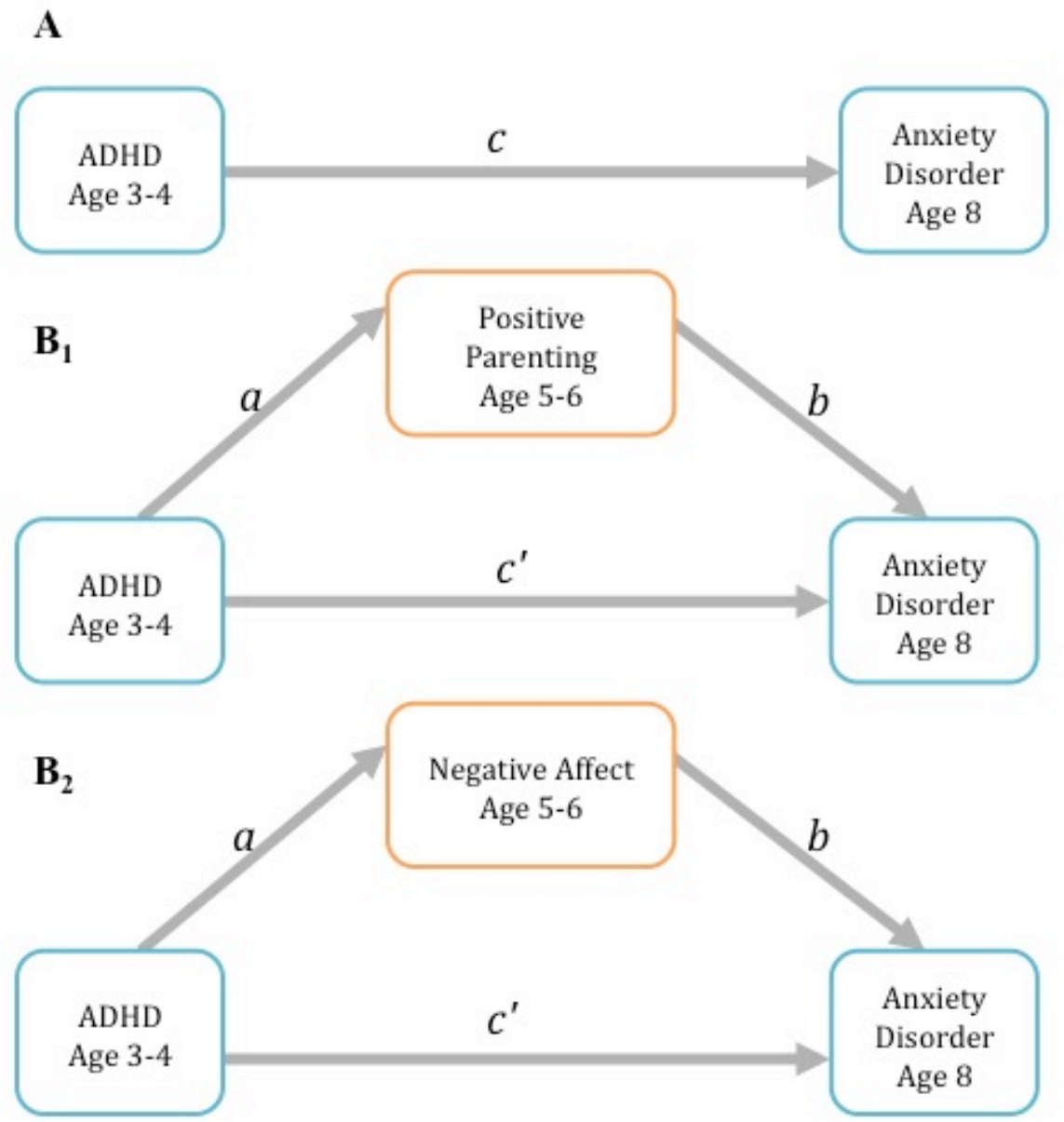
Data Analysis

Two independent sets of analyses were used to test the mediation model posited by the IPH. First, *simple mediation* was assessed (*Models 1* and *2*) with the commonly-used, causal-steps strategy (Baron & Kenny, 1986), employing hierarchical logistic

regression that was then followed by bootstrapped tests of the significance of the effects. In the second method, *multiple mediation* was tested using bootstrapping, a technique popularized only recently (and described more below (Preacher & Hayes, 2008)), in order to assess the mediating effects of both parenting variables, together (*Model 3*). Although the latter was considered more powerful and preferable (for reasons explained below), Baron and Kenny's (1986) approach was included as well due to its widespread familiarity and acceptance within the field.

Causal-steps approach to assess simple mediation. The widely-used, causal-steps approach recommended by Baron and Kenny (Baron & Kenny, 1986) assessed simple mediation by each of the two variables of interest (Positive Parenting and Parent Negative Affect). In simple mediation, only one mediator is studied and assessed in a given model. Thus, two separate models were tested, the first with Positive Parenting as the mediator (*Model 1*), and the second with Parent Negative Affect (*Model 2*). Figure 2 illustrates both of these models. The independent variable was BL ADHD and the dependent variable was Age 8 anxiety disorder.

Figure 2
Simple Mediation Models 1 and 2



Note: Adapted from Preacher and Hayes (2008; p.881). (A) shows the total effect, *c*, through which ADHD at Ages 3-4 is presumed to affect Anxiety Disorder at Age 8; (B_1) shows *Model 1*, in which Age 5-6 Positive Parenting was predicted to partially mediate the effect of ADHD on anxiety disorder; (B_2) shows *Model 2*, in which Age 5-6 Parent

Negative Affect was predicted to partially mediate the effect of ADHD on anxiety disorder; path *a* represents the effect of ADHD on the mediator; path *b* represents the effect of the mediator on Anxiety Disorder after accounting for ADHD; the *ab* pathway signifies the indirect effect; path *c'* shows the difference between the total effect and the indirect effect (*c – ab)*.

Baron and Kenny (1986) outlined four steps to test for mediation. Step 1 requires that the independent variable be correlated with the dependent variable. Step 2 requires that the independent variable also be correlated with the mediator variable. Step 3 requires that the mediator be significantly correlated with the outcome variable after controlling for the independent variable. Finally, to be considered mediation, Step 4 requires that the effect of the independent upon the dependent variable be removed (in the case of a full mediation) or attenuated (in the case of a partial mediation) after controlling for the mediator.

In accordance with the four steps, partial correlations were first run among the three variables in each of the two models, controlling for SES, which was found to correlate with parenting scores in Studies 2 and 3. Gender was not included as a covariate because it was not found to be associated with Age 8 anxiety disorder. Where Steps 1-3 were satisfied (i.e., partial correlations were significant), regression analyses were run, also covarying for SES. Specifically, hierarchical linear regression provided unstandardized (B) and standardized (β) coefficients for the *a* path; hierarchical logistic regression analyses generated unstandardized coefficients (B) as well as odds-ratios for paths *c* (the effect of ADHD on anxiety disorder)*, b* (the effect of Positive Parenting on

anxiety disorder, controlling for ADHD), and *c'* (the effect of ADHD on anxiety disorder, controlling for Positive Parenting). The causal-steps approach was then considered complete; accordingly, if the odds-ratio of *c'* was found to be smaller than *c*, evidence of a mediation was said to have occurred.

To test whether the indirect effect was significant, bootstrapping was performed (Mackinnon, Lockwood, & Williams, 2004; Preacher & Hayes, 2004, 2008). Bootstrapping is a nonparametric procedure that selects and reselects subsamples at random (using a "replacement" procedure in which individuals are removed and replaced with other individuals from one subsample to the next) from within the full sample and measures the path coefficients within each subsample. Each of the path coefficients (*a*, *b*, *c*, and *c'*) is averaged across subsamples to produce an overall coefficient for each path within the model. Path coefficients for the indirect effect, *ab*, are sorted by size, from smallest to largest, and a confidence interval is then generated. Confidence intervals (C.I.s) that do not include zero suggest that the effect of the mediation is significant. Preacher and Hayes (2004) recommend the use of 5,000 samples, with bias-corrected, 95% confidence intervals of effect size.

While the significance of the indirect effect can also be tested using the Sobel test, this test requires normal distribution of data, and generally possesses low power unless a very large sample size is used (Preacher & Hayes, 2004). As reviewed by Preacher and Hayes (2004), the assumption of normality is especially problematic in mediation analysis because the product of two coefficients that yields the indirect effect (i.e., the *a* path multiplied by the *b* path) is likely to be skewed. In contrast, they reason,

bootstrapping offers a superior significance test for the indirect effect that is robust in samples of all sizes and is unaffected by non-normality of data.

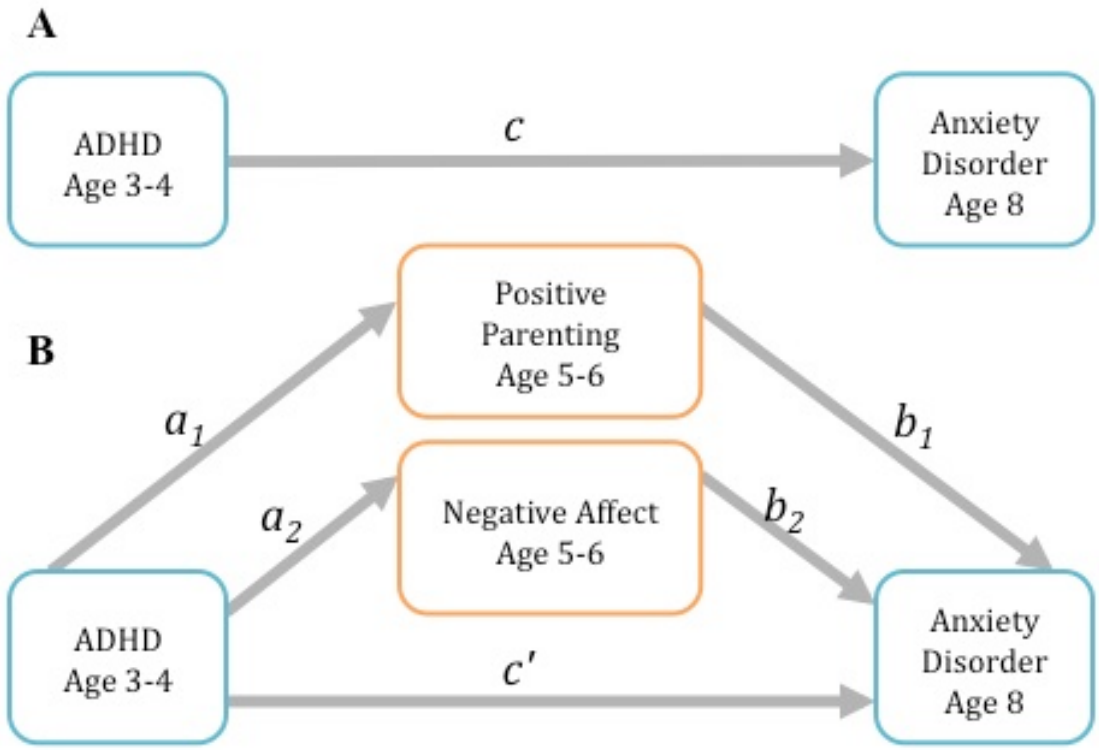
Bootstrapping to assess multiple mediation. Beyond its use as a follow-up test of significance of the indirect effect, bootstrapping has also been shown to be a more powerful alternative to using Baron and Kenny's (1986) approach (Mackinnon et al., 2004; Preacher & Hayes, 2004). This is due, at least in part, to its emphasis on the indirect effect, *ab*, instead of on individual pathways. Thus, whereas the causal-steps approach requires that *a, b,* and *c* all be significant for mediation to be considered, bootstrapping requires only that the indirect effect be significant (Preacher & Hayes, 2004, 2008). The former is more prone to Type II error, and therefore less powerful, because of the multiple significance tests that must be satisfied in order for the null hypothesis to be rejected. With this in mind, *Model 3* was run in accordance with bootstrapping procedures, without regard for whether partial correlations described above were found to be significant.

Bootstrapping analyses were conducted using a macro designed for SPSS, described by Preacher and Hayes (2008), and which is particularly well suited to handle *multiple* mediation by looking at overall mediation as well as by contrasting individual mediators. As the authors assert, "When multiple mediators are entertained, it is often more convenient, precise, and parsimonious to include all of them in the same model" (pp.886-887). As such, *Model 3* tested whether parental warmth/lack of rejection and positive contingency management (as measured by Positive Parenting and Parent Negative Affect) partially mediated the relationship between early ADHD and later anxiety disorder.

Bootstrapping procedures also identified the extent to which each of the two measures individually contributed to the mediation. Although collinearity between the two parenting measures would attenuate the specific indirect effects of each, multiple regression assessed the *unique* contributions of each (Preacher & Hayes, 2008). Investigation of the potential collinearity of the two variables (Positive Parenting and Parent Negative Affect) showed they were insignificantly correlated with one another (Pearson correlation = .056, p = .471). *Model 3* is depicted in Figure 3.

Figure 3

Model 3: Multiple Mediation Model to Test the IPH



Note: Adapted from Preacher and Hayes (2008; p.881). (A) shows the total effect, c, in which ADHD at Ages 3-4 is presumed to affect Anxiety Disorder at Age 8; (B) shows the indirect effects of ADHD on Anxiety Disorder through the two mediators, Negative Affect and Positive Parenting; (a_1) and (a_2) show the effect of ADHD on the respective parenting variables; (b_1) and (b_2) show the effects of the two mediators on Anxiety Disorder after accounting for ADHD; the *ab* pathway signifies the indirect effect; (*c'*) shows the direct effect, which is equal to the difference between the total effect and the indirect effect (*c – ab)*.

Results

Causal-steps approach to assess simple mediation.

***Model 1*: Positive Parenting.** Pearson product moment and point biserial correlational analyses, run between the independent (BL ADHD), mediating (Positive Parenting at age 5-6), and dependent (Age 8 Anxiety Disorder) variables, and controlling for SES, were indeed significant (see Table 18). In line with the causal steps approach, mediation analyses could therefore proceed.

Table 18

Partial correlations among BL ADHD, Age 5-6 Positive Parenting, and Age 8 Anxiety Disorder

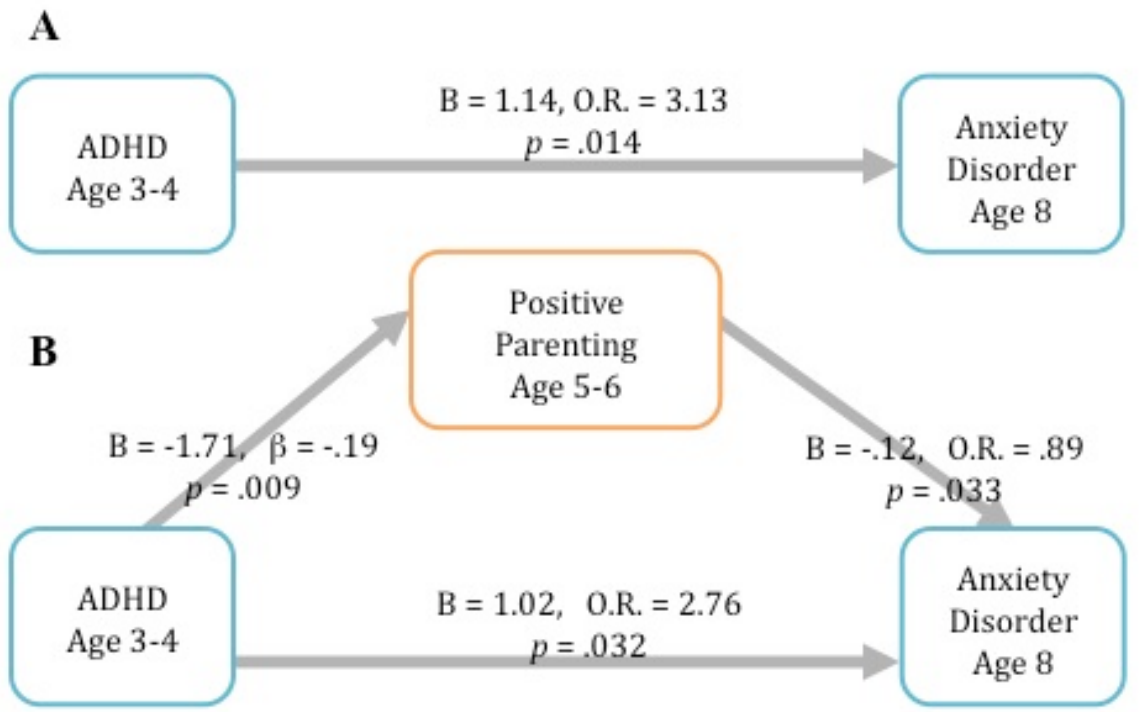
	Positive Parenting	**Age 8 Anxiety Disorder (-/+)**
BL ADHD (-/+)		
Correlation Coefficient	-.185*[i]	.206*[ii]
Sig (2-tailed)	.023	.011
Positive Parenting		
Correlation Coefficient	-	-.205*[i]
Sig (2-tailed)		.012

Note: All correlations controlled for socioeconomic status; BL = Baseline; APQ = Alabama Parenting Questionnaire, Preschool Revision; Positive Parenting = mean of APQ-PR Positive Parenting at Age 5 and Age 6; [i] point-biserial correlation; [ii] phi coefficient; **p*<.05; ***p*<.01

The simple mediation model was assessed for the parenting variable, Age 5-6 Positive Parenting, using hierarchical regression and controlling for SES. Results are depicted in Figure 4. They indicate that, after accounting for Age 5-6 Positive Parenting, ADHD was still a significant predictor of Age 8 Anxiety, but its effect size decreased slightly, therefore signifying a partial mediation.

Figure 4

Model 1: Analysis of the mediating effect of Age 5-6 APQ-PR Positive Parenting upon the relationship between Age 3-4 ADHD and Age 8 Anxiety Disorder



*Note: (*A) shows the total effect of ADHD on later anxiety disorder, without accounting for Positive Parenting; (B) shows unstandardized and standardized coefficients for individual pathways of the mediation model, and the direct effect, after accounting for the indirect effect; attenuation of the total effect after accounting for the indirect effect is suggestive of a partial mediation; both (A) and (B) are controlling for SES; O.R. = odds ratio.

To test the significance of the mediation, bootstrapping was run with 5,000 samples and 95% bias-corrected confidence estimates. Bootstrapping revealed

confidence intervals for the indirect effect that did not cross zero (indirect effect = .191; C.I. = .014 to .554). Therefore, the partial mediation of *Model 1* was, in fact, significant.

***Model 2*: Parent Negative Affect.** Partial correlations among the independent (BL ADHD), mediating (Parent Negative Affect), and dependent (Age 8 Anxiety Disorder) variables were not all significant (see Table 19). Because the IV and MV were not significantly correlated, this simple mediation *Model 2* was not pursued.

Table 19

Partial correlations among BL ADHD, Age 5-6 Parent Negative Affect, and Age 8 Anxiety Disorder

	Age 5-6 Parent Negative Affect	**Age 8 Anxiety Disorder (-/+)**
BL ADHD (-/+)		
Correlation Coefficient	-.140 [i]	.229** [ii]
Sig (2-tailed)	.097	.006
Age 5-6 Parent Negative Affect❖		
Correlation Coefficient	-	-.327** [i]
Sig (2-tailed)		<.001

Note: All correlations controlled for socioeconomic status; BL = Baseline; PCI = parent-child interaction; Age 5-6 Parent Negative Affect = mean of Parent Negative Affect at Age 5 and Age 6; ❖Negative Affect is reverse scored, such that higher values are

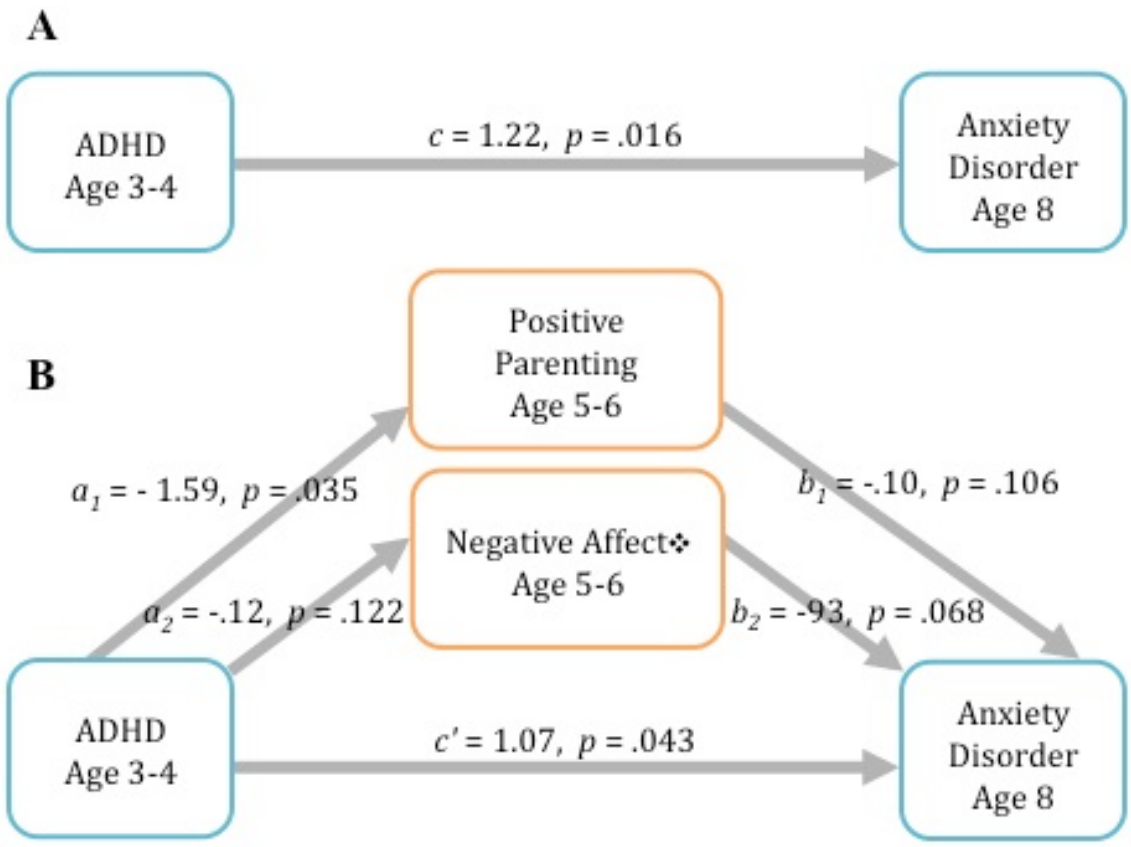
reflective of less Negative Affect; [i] point-biserial correlation; [ii] phi coefficient; **p*<.05; ***p*<.01.

Bootstrapping to Assess Multiple Mediation

Multiple logistic regression using bootstrapping included Positive Parenting and Parent Negative Affect, together (*Model 3*), as mediators and assessed each component of the mediation model proposed. Preacher and Hayes (2008) advise against the use of standardized coefficients; thus, only unstandardized coefficients are reported. As shown in Figure 5, the total effect of BL ADHD on Age 8 anxiety disorders was significant (B = 1.218, $z(132) = 2.402$, $p = .016$). BL ADHD was also predictive of low Positive Parenting at 5-6, via the *a* path, (B = -1.595, $t(132) = -2.130$, $p = .035$). Significance was not achieved for the *b* paths, (Positive Parenting B = -.101, $z(132) = -1.618$, $p = .106$; Negative Affect B = -.930, $z(132) = -1.823$, $p = .068$). However, as this is not a requirement for mediation using bootstrapping (Preacher and Hayes, 2008), it did not prevent analyses from proceeding anyway; rather, the lack of significant individual paths speaks more to collinearity between the variables such that the effect of each mediator was attenuated by the inclusion of the other, as is the case in multiple regression (Preacher & Hayes, 2008). Results are depicted in Figure 5 and summarized in Table 20.

Figure 5

Model 3: Bootstrapped, multiple mediation analysis of Positive Parenting and Parent Negative Affect at Age 5-6 on the relationship between Age 3-4 ADHD and Age 8 anxiety disorder



*Note: (*A) shows the total effect of ADHD on Anxiety Disorder, without accounting for the mediators; (B) shows unstandardized coefficients for individual pathways of the mediation model, and the direct effect, after accounting for the indirect effect; ❖Negative Affect is reverse scored, such that higher values are reflective of less Negative Affect.

Table 20

Model 3: Multiple mediation path coefficients of the effect of BL ADHD on age 8 anxiety disorder through age 5-6 Positive Parenting and Parent Negative Affect

	Unstandardized Coefficients	**S.E.**	**z (t)**	***p***	**Wald**
a					
Positive Parenting*	-1.595	. 749	(-2.13)	.035	-
Negative Affect❖	-.120	.077	(-1.56)	.122	-
b					
Positive Parenting	-.101	.062	-1.62	.106	2.62
Negative Affect❖	-.930	.510	-1.82	.068	3.32
c					
BL ADHD*	1.218	.507	2.40	.016	5.77
c'					
BL ADHD*	1.067	.527	2.02	.043	4.09
Control variable					
SES	.005	.014	.368	.713	.13

Note: Results of bootstrapping, unstandardized path coefficients with 5,000 samples using bias-corrected, 95% confidence intervals (C.I.); S.E. = standard error; SES = socioeconomic status; ❖Negative Affect is reverse scored, such that higher values are reflective of less Negative Affect; *p<.05.

Bootstrapping obtained bias-corrected confidence estimates (Mackinnon et al., 2004; Preacher & Hayes, 2008), computing 5,000 samples, with 95% confidence intervals of indirect effect size (Preacher & Hayes, 2008). Results indicated that *Model 3*'s partial mediation was, in fact, significant, as demonstrated by confidence intervals for the total mediation that did not include zero (B = .2723, C.I. = .016 to .681).

Neither Positive Parenting nor Parent Negative Affect emerged as a unique mediator on its own (see Table 21). There was not a significant difference between the indirect effect offered by Positive Parenting and that of Parent Negative Affect, as indicated by the contrast in Table 21.

Table 21

Model 3: Multiple mediation effect size and significance testing

	Point Est.	**Bootstrapped Point Est.**	**S.E.**	**B.C. 95% C.I.**	
				Lower	**Upper**
Total*	.272	.285	.166	.010	.665
Positive Parenting	.160	.159	.143	-.040	.555
Negative Affect❖	.112	.126	.117	-.027	.447
Contrast	.049	.034	.202	-.343	.465

Note: Results of bootstrapping using standardized coefficients of parenting with 5,000 samples using bias-corrected, 95% confidence intervals (C.I.); Point Est. = normal regression coefficient of indirect effect *ab*; Bootstrapped Point Est. = bootstrapped

coefficient of indirect effect *ab*; S.E. = standard error; B.C. = bias-corrected; Contrast = comparison of the indirect effect of Positive Parenting vs. Parent Negative Affect; ❖Negative Affect is reverse scored, such that higher values are reflective of less Negative Affect; *statistically significant mediation effect.

Discussion

This study investigated the hypothesis that parenting at ages 5-6 would partially mediate the relationship between ADHD at ages 3-4 and anxiety disorders when children were 8. Two analytic procedures, one a more traditional technique and another more powerful, modern approach, both provided support for this hypothesis. To summarize, simple mediation analysis using the causal-steps approach found support for *Model 1*: parent-rated positive parenting was a small but significant (according to subsequent bootstrapping tests) partial mediator. The same approach did not provide support for *Model 2*; observed parent negative affect was not identified as a mediator. However, *Model 3,* which included both measures of parenting as mediators, found there to be a significant partial mediation of the relationship between early ADHD and later anxiety disorder that was attributed to the two, together. Neither positive parenting nor parent negative affect emerged as unique mediators in the model.

Therefore, this study provided support for the hypothesis that one pathway by which ADHD indirectly confers risk for anxiety disorders is via parenting. It is also the first of its kind to document the direction of the effect, longitudinally, among childhood ADHD, parenting, and childhood anxiety disorders. The particular style of parenting identified was that high in rejection and low in warmth and use of positive contingency management.

Models 1 and *3* were the two supported by the data. The first was of positive parenting, which was based upon parents' ratings. The second was comprised of this factor as well as observer-rated negative affect. Therefore, a limitation of the present study was that both mediating variables relied upon parent ratings, meaning that parent

ratings were the primary source of information, comprising the independent, mediating, and dependent variables (diagnostic status, which represented the IV and the DV, was based on largely parent interview). It would have been preferable for the variables to have been independent of one another, as utilizing the same rater for all three presents potential confounds related to systematic biases in responding. This was a design issue that PCI data had been included to address. Future replication, incorporating observational and other-rated measures of parenting, is needed to confirm the present findings.

The extent to which the direct effect was mediated by parenting was fairly small. This serves as a reminder that parenting is probably one of many factors that contribute to the comorbidity of ADHD+anxiety; critical though it may be, it is only one small slice of the pie. Other factors that likely contribute include those reviewed in Chapter 1, and which were not studied in the present investigation. Moreover, given that risk for anxiety probably ensues from an interaction of numerous factors, the present model, which looks at only one of these, is highly oversimplified. Future studies will benefit from using more advanced modeling techniques, like structural equation modeling, which enables the researcher to investigate the effects of a multitude of variables and their interactions over time.

A more extensive consideration of the present study is reserved for the following chapter, within the context of the larger discussion.

Chapter 7: Overall Discussion

Summary of Findings

The aim of the present investigation was to empirically test the Intermediate Parenting Hypothesis (IPH) longitudinally, in a sample enriched for ADHD symptoms at the baseline age of 3-4 years. Children were followed until age 8. The IPH predicts that one mechanism by which children with ADHD are at elevated risk for anxiety disorders is via parents' negative parenting practices, which are elicited or magnified by children's ADHD-related behaviors. The four-part series of studies presented herein offers support for this hypothesis, as the first study to date to examine the relationship between ADHD, parenting, and anxiety disorders over time.

Prior research provided support as well. Thus, parents of children with ADHD have been shown to be more over-controlling, rejecting, and harsh, and to provide less positive reinforcement, relative to parents of typically developing children (Deault, 2009; Johnston, 1996; Johnston & Jassy, 2007). These negative parenting characteristics are also implicated in research on the developmental psychopathology of childhood anxiety disorders (Bogels & Brechman-Toussaint, 2006; Chorpita & Barlow, 1998; McLeod, Wood, et al., 2007; McLeod et al., 2011; Rapee, 1997). However, prior studies have been limited, first and foremost, by lack of longitudinal data collection and overreliance on self-report questionnaires, and secondly, by varying metrics, ages, and comorbidities. As a result, findings published to date have left important questions unanswered. One remaining question in both ADHD and anxiety research has to do with temporal order of parenting styles and childhood psychopathology. The extent to which parenting is affected by a child's ADHD has been debated, with some arguing that ADHD leads to

negative parenting styles (Johnston & Jassy, 2007; Johnston & Mash, 2001) and others attributing parenting differences among children with ADHD to comorbid disruptive behavior disorders (Burke et al., 2008). Although theoretical literature implicates parenting as a risk factor in the development of childhood anxiety disorders, little research has been conducted demonstrating, prospectively, a temporal linkage between early parenting practices and later anxiety disorders (Bogels & Brechman-Toussaint, 2006; Chorpita & Barlow, 1998; McLeod, Wood, et al., 2007; McLeod et al., 2011; Wood et al., 2003). A second remaining question has been which parenting practices are *consistently* implicated in the two disorders. In particular, whereas parents of children with anxiety disorders have been found to be overly controlling across studies, whether parental rejection is also elevated among these children has been disputed (Bogels & Brechman-Toussaint, 2006; Chorpita & Barlow, 1998; McLeod, Wood, et al., 2007; McLeod et al., 2011; Wood et al., 2003).

We hypothesized that early childhood ADHD would impact parenting practices over time, leading parents to exercise more excessive restriction and over-control, and exhibit more critical and rejecting behavior, less warmth, and reduced positive contingency management. In turn, parenting practices high in rejection and over-control and low in positive contingency would then make children more prone to developing anxiety disorders over time. Finally, we hypothesized that parenting would partially mediate the relationship between ADHD at ages 3-4 and anxiety disorders two to five years later, at ages 6 and 8. Utilizing an existing longitudinal dataset that was enriched for ADHD symptoms at baseline, the relationship between early ADHD, parenting practices, and later anxiety disorders was systematically assessed over time.

Study 1 confirmed the increased risk for anxiety disorders among children with ADHD. ADHD at age 3-4 did not significantly predict anxiety disorders at age 6, but it did by age 8; a small number of children presented with anxiety disorders at six years of age, but by age 8, a substantial proportion (28%) of the sample of children with early ADHD also met criteria for one or more anxiety disorders. Therefore, Study 1 replicated findings of increased risk for anxiety disorders in children with ADHD, while additionally documenting the emergence of this relationship subsequent to the onset of ADHD.

Study 2 indicated that parents of children with ADHD at age 3-4 years emitted more negative affect and less respect for children's autonomy both concurrently and again at Age 5, as based on observed interactions. ADHD at ages 3-4 also predicted poorer parenting at later assessment points, including lower self-reported positive parenting at ages 5 and 6, reduced quality of assistance during observations at age 6, and lower scores overall parenting quality composite based on observations during parent-child interactions at ages 5 and 6 years. Importantly, these associations remained significant after accounting for parenting at BL ADHD, as well as gender, SES, and comorbid ODD. Therefore, the findings provide evidence that early ADHD actually led to reduced parental warmth, positive reinforcement, and constructive and stimulating support within the context of learning opportunities. There was some evidence that early ADHD perpetuated parents' already lower respect for their child's autonomy, as well as their already greater expression of parental negative affect, although this pattern did not continue to be significant by the time children were 6 years old.

Study 3 found that low self-rated positive parenting between the ages of 3 and 5 was predictive of anxiety disorders in children at age 8 (but not age 6). The same was true of observer ratings of parent negative affect at ages 3-4 and 5. Moreover, lower positive parenting and greater negative affectivity remained significant predictors of anxiety even after controlling for children's earlier-measured temperament. This suggests that negative and rejecting parenting contributed unique risk for childhood anxiety, above and beyond children's predisposition to anxiety disorders. Contrary to our predictions, however, observations of neither respect for autonomy nor quality of assistance differentiated between children with and without later anxiety disorders. Therefore, within these data, rejection, lack of warmth, and poor positive contingency management were predictors of anxiety disorders, whereas measures of parent over-control were not.

Finally, Study 4 found age 5-6 parenting, particularly self-rated warmth and contingency management, to have a significant mediating effect on the relationship between ADHD at 3-4 and anxiety disorders at age 8. Observed lack of parental negative affect (rejection) at age 5-6 was not a significant mediator on its own, but combined with parent-rated positive parenting to also be a significant partial mediator. Therefore, mediation analyses confirmed our prediction that parenting would partially mediate the relationship between early ADHD and later anxiety disorders. Specifically, one way that ADHD appeared to indirectly lead to anxiety disorders was by inciting in parents less warmth and provision of positive reinforcement, and more parental expressed negative emotion toward children. Parental over-control did not mediate this relationship.

Two other cross-sectional studies have investigated the role that parents play in the development of anxiety disorders among children with ADHD (Kepley & Ostrander, 2007; Pfiffner & McBurnett, 2006). Both reported increased over-control among parents of children with ADHD+anxiety. The first study relied on parent as well as child ratings of parenting on the PCRQ and parent ratings on the APQ. Among a sample of clinic-referred 5-11 year-olds, higher ratings of PCRQ parental possessiveness (rated by parents and children) were found in an ADHD+anxiety group than in a typically developing comparison group, and children with ADHD-only fell in between. Decreased APQ positive parenting ratings (by parents) were also reported (Pfiffner & McBurnett, 2006). Our findings, using the preschool version of the APQ, replicated these differences in positive parenting, in addition to providing insight into the temporal sequence through which the relationship between ADHD and positive parenting unfolded. However, differences in control were not found in our dataset. The second study investigated a sample of first through fourth graders with ADHD, ADHD+anxiety, and typically developing controls; the authors found parents of children with ADHD+anxiety to be particularly discouraging of autonomy and restrictive over their children, as based on parent reports on the BASC-PPP and the FES (Kepley & Ostrander, 2007). Measures of parental warmth/rejection or positive reinforcement were not collected.

In contrast to findings of these other two studies, and contrary to our hypothesis, parents of anxious children were *not* observed to be more controlling than parents of their non-anxious counterparts at earlier points in time. There are a number of potential reasons for these anomalous findings. One is that it is possible that the brief observational measures we used were insufficiently sensitive to detect differences in the

sorts of control techniques reported elsewhere. Whereas the parent rating scale included a measure of warmth/rejection/positive contingency management, it did not measure over-control. However, observed control (respect for autonomy and quality of assistance) differed between groups of children with and without early ADHD, suggesting that the measure was not insensitive. Another plausible explanation could be that observations of parental negative affect tapped into aspects of psychological (as opposed to behavioral) over-control, and that psychological over-control was partially reflected in high scores on this scale. Notably, findings of parental control were reported on parent- and child-rating scales in the two previously-mentioned studies.

Aside from measurement issues, sample composition in this study may also have contributed to the surprising findings regarding control. Given that our sample was comprised of a substantial proportion of children presenting with symptoms of ADHD at baseline, it is possible that anxiety disorders result from different factors among children with and those without high levels of ADHD symptoms. If both control and rejection each contribute to anxiety within the greater population, it may be that rejection contributes more prominently among children with ADHD. Potentially related to this, recent studies have reported children's experiences of parental rejection and *not* parental over-control to be associated with their risk of having GAD (McLeod et al., 2011; Shanahan et al., 2008). It follows that the present findings of high levels of parental rejection and not control among anxious children may be predictive of particularly high rates of GAD. In fact, 11% percent of children with ADHD at age 3-4 met DSM-IV criteria for GAD when they were 8; this was twice the rate of any of the other anxiety disorders studied. Others have also reported higher levels of worry (compared to phobic-

like anxiety) among children with ADHD+anxiety (Jensen et al., 2001; March et al., 2000; Molina et al., 2009). Therefore, parenting may confer particular risk for GAD among children with ADHD. Unfortunately, the number of children with GAD in the present study was not sufficiently large for this hypothesis to be tested.

It is also possible that age differences in our sample were responsible for the lack of findings of parental over-control; specifically, our sample was younger than those in most studies that have been conducted, and included a narrower and slightly younger age-band than the Kepley and Ostrander (2007) and Pfiffner and McBurnett (2006) studies. Research on depression has underscored the importance of developmental stage in affecting the impact of parenting on children's brain development and internalizing symptomatology. For instance, neuroimaging studies highlighted a sensitive period, ending at age 8, during which time children's brain development in regions relevant for depression and anxiety was particularly susceptible to parental nurturance (or lack thereof; Rao et al., 2010). These findings were echoed by another study, which showed parental behavior management (which was comprised of consistency, positive reinforcement, and monitoring) to be a unique and potent predictor of depression in children under the age of 8 (Ostrander & Herman, 2006). In the same study, children's cognitive style (specifically, locus of control) became increasingly predictive of depression from age 8 onward, whereas parental behavior management became a weaker and, by age 10, an insignificant predictor of depression in children. These two studies corroborate the poignant impact of parental warmth and contingency management in early childhood, in particular, and highlight a need for future research in developmental psychopathology to focus on the role of parenting in early development. Perhaps

consistent with this notion that children are particularly susceptible to parental warmth early in development, in the present study, anxiety disorders at age 8 were predicted by parent-rated positive parenting when children were 3-4 and 5, but not 6 years of age.

There are probably many routes by which children with ADHD become prone to developing anxiety. In addition to parenting practices and the other potential avenues reviewed in Chapter 1, the present data lend some support for other pathways, as well. One account proposes that children with SCT, who are characterized by high levels of inattention and less commonly by hyperactivity/impulsivity, are more prone to comorbid anxiety and ADHD, and that children with SCT reflect a distinct neuropsychological and psychopathological phenotype (Schatz & Rostain, 2006). In Study 1, inattentive, but not hyperactive-impulsive, symptoms at ages 3-4 were predictive of anxiety disorders at six years of age, which might be considered support for the hypothesis that inattention and anxiety are linked from an early age. However, early hyperactive/impulsive symptoms predicted anxiety disorders by age 8. It may be that the risk conferred by SCT among some participants was then eclipsed by the greater incidence of anxiety disorders that emerged for other reasons as well.

Parenting stress is an additional variable, upon which we did not report but which may have contributed to and interacted with parenting practices in driving anxiety. Parents of children with ADHD experience more stress and family conflict than do parents of typically-developing children and adolescents (Biederman et al., 1999; Brown & Pacini, 1989; G. J. DuPaul et al., 2001; Gadow et al., 2000; Scahill et al., 1999). Parent stress has also been found to increase risk for internalizing disorders

among children followed longitudinally (Ashford et al., 2008). It is conceivable that one mechanism by which the increased incidence of anxiety disorders occurs is via the effect of stress on parent practices. In fact, this is precisely the mechanism written about by Johnston over the years (Johnston & Jassy, 2007; Johnston & Mash, 2001). Thus, stress may represent an additional intermediary step, mediating the relationship between ADHD in children and negative responses in their parents.

Strengths, Limitations, and Future Directions

This was the first investigation to utilize a longitudinal design to explicitly and systematically assess the relationship between ADHD, anxiety disorders, and parenting over time. Only by doing so could inferences about causality be tested empirically. Moreover, the early age of commencement of the studies (i.e., tracking of children and parents from the time children were 3-4 years of age), allowed us to evaluate interactions among parent and child variables from an critically early point in development, and perhaps before more ingrained habits had taken hold.

The systematic tracking of these relationships between children's disorders and the parenting practices of their parents is unique and contributes to the respective literature on ADHD as well as that on anxiety disorders in children. In particular, the prospective design is something that few studies of parenting in child anxiety disorders have benefitted from up to this point. Our dataset allowed us to assess which forms of parenting predated the onset of anxiety disorders, something that few have examined to date (Rubin et al., 2002; Rubin et al., 1999). This is far preferable to cross-sectional studies that compare children with and without anxiety disorders, as well as longitudinal studies that track children who are either anxious already or believed at-risk, as it allows

for the observation of anxiety disorders as they unfolded over time. Specifically, the present design allowed for rigorous examination of one potential mechanism for anxiety disorders, parenting, and for preliminary answer to the question of whether parenting partially accounts for increased risk for anxiety disorders in children with ADHD.

Observational measures of parenting were another strength of the study, in that they offered potentially more objective information parent ratings, alone, given that parental self-reports are subject to biases from other factors (such as parental psychopathology, stress, or culture, to name a few). Collecting multi-informant data was methodologically more sound, as well, because parents were relied upon for diagnoses of ADHD and anxiety disorders (the independent and dependent variables); since parents are subject to patterns of and biases in responding, reliance solely on parent report can lead to confounding results.

Despite these strengths, there were a number of limitations that should be considered. First, we lacked child reports of anxious symptoms, and instead relied upon parental report and supplementary information (from teacher rating-scales and from qualitative observations made while children were completing testing and/or in the office). The association between child and parent ratings of anxiety has been shown to be less than ideal (March et al., 2000), suggesting that children's self-ratings offer unique and important perspective on their symptomatology. A related limitation is that we did not conduct diagnostic screenings for anxiety disorders prior to age 6. Doing so would have enabled us to better infer the directionality of effects.

Another limitation was our somewhat exploratory approach to parenting data in Studies 2 and 3. Thus, we were interested in determining which parenting practices

would be associated with the two disorders, as well as which would be shared between the two. A more conservative approach would have been to select, a priori, which parenting variables would be incorporated into regression analyses. A more significant flaw, which resulted from this exploratory approach, was the fact that numerous one-way ANOVAs were conducted in order to identify which variables were relevant, potentially inflating alpha and rate of Type I error. Unfortunately, there was insufficient power to detect group differences using the more conservative and preferable MANOVA.

A final limitation of our study is that we did not include interviews of parental psychiatric status. Thus, we were unable to assess whether parental psychopathology was contributing to outcome variables. This could conceivably occur through different mechanisms, including by affecting parenting practices themselves, via parental modeling of anxiety, and/or inheritance. Notably, both of the existing studies investigating the relationship among ADHD, anxiety, and parenting gathered data on parental anxiety, and one on parental depression and ADHD, as well; neither found parental psychopathology to impact results (Kepley & Ostrander, 2007; Pfiffner & McBurnett, 2006). However, mothers with ADHD have also been shown to use less positive parenting, more negative and inconsistent parenting, and to provide children less opportunity to comply with commands before restating them than mothers without ADHD, as based on APQ as well as observational measures (Chronis-Tuscano et al., 2008). That numerous mothers of children with ADHD may have also had ADHD and that this may have driven parenting practices is a potential confound that cannot be ruled out in the present study.

In light of the dynamic, transactional relationship among factors that is most accountable for anxiety disorders in children with ADHD, future studies will benefit from more advanced statistical techniques, such as structural equation modeling. Such approaches allow for more comprehensive investigation of the way in which multiple variables interact over time to shape protection and risk. Future studies will benefit also from larger sample sizes. As identified earlier, an important empirical question that would be testable within a larger sample of children would be whether children with ADHD are at differential risk for certain anxiety disorders over others as a function of parents' rejecting qualities and/or poor contingency management.

Future studies may also wish to investigate which specific symptom dimensions of ADHD account most for the IPH. It might be predicted, for instance, that symptoms of hyperactivity/impulsivity would be most predictive of negative parenting behaviors, because they are more overtly disruptive than symptoms of inattention. However, Seipp (Seipp & Johnston, 2005) found that subtype did not affect the interactions of mothers and their 7 to 9 year-old sons.

Implications for Prevention and Treatment

The role of parents in treating childhood anxiety is well established. Parental involvement in psychotherapy for childhood anxiety disorders is a crucial aspect of treatment (Barrett, Dadds, Rapee 1996). Whereas standard cognitive-behavioral therapy for childhood anxiety includes parents, virtually universally, interventions have been developed that place special emphasis on family relations. Family-based cognitive behavioral therapy, which targets aspects of parenting and family dynamics, has shown some evidence of improved efficacy at reducing children's anxious symptoms, relative to

individual cognitive-behavioral therapy, alone (Bogels & Siqueland, 2006; Wood, Piacentini, Southam-Gerow, Chu, & Sigman, 2006).

Results of the present investigation provide further indication for the practice of parent training among parents of children with ADHD. Parent training, whether parent management training, behavioral parent training, or parent-child interaction therapy, is typically recommended to improve externalizing behavior among children with ADHD. However, principles common to all versions of these programs include expressions of warmth, praise, and consistency of discipline; parents are taught to use positive contingency setting to reinforce desirable behaviors or approximations thereof, and to reduce their reliance on negative, hostile, reactive, or overly punitive consequences for misbehavior. In the latter case, parents are taught to administer consequences (e.g., time-out, removal of privileges) with a neutral, matter-of-fact tone, in such a way that children learn which behaviors to avoid in the future without internalizing a sense of parental rejection or removal of warmth. In essence, parent training teaches parents to do the *opposite* of what was found in the present study to place children at risk for anxiety: to use positive reinforcement and consistency of responding, and to avoid reacting to misbehavior out of anger or frustration. Indirectly, parent training may be helpful not only for behavior problems, but also protective against future anxiety problems as well.

Parent psychoeducation may be especially critical with prevention in mind, to inform parents of the importance of consistent contingency management and expression of warmth and positive regard. It is important that parents learn that yelling or inflicting harsh punishment for failure to complete tasks or perform up to expectations is unhelpful to children and has the potential to lead to emotional issues over time. This is an intuitive

lesson that is often shared with parents in clinical settings, currently; however, the present study offers hard data showing that negative parenting in preschoolers, at least in the context of ADHD, presents very real and damaging risk for children by mid-elementary school.

An intriguing recent study that identified very young children (pre-age 4) with a profile of high withdrawal and poor behavioral inhibition (i.e., whose temperament placed them at risk for anxiety disorders) implemented a six-session, parent training, preventive intervention, in which parents were educated about anxiety, parenting techniques, and the role of over-protection (Cuthbert, 2010). Results indicated that the number and severity of anxiety disorders three years later was significantly reduced compared with the control group. Therefore, there is evidence that preventative interventions, targeting children at risk, have the potential to offset this risk. Future iterations of this and similar programs may benefit from modifying curricula to target children presenting with externalizing disorders, as opposed to internalizing symptoms, as primary.

It is interesting that parent-child interaction therapy (PCIT) has been shown to have strong benefit not only in externalizing symptoms, but internalizing symptoms as well (Chase & Eyberg, 2008). What differentiates PCIT from other parent training programs is its use of in-vivo coaching of parents while they interact with their children (McNeil & Hembree-Kigin, 2011). This allows for modification of parenting not just at a conceptual level but a molecular level, as well, wherein the subtlest parent behaviors are broken down and reconstructed. For instance, within the clinic setting, attention can be called to the slightest negativity in a statement, which could be perceived by a child

differently than the parent intended; conversely, enthusiasm is trained, and use of labeled praise is modeled and drilled, so parents achieve a certain level of mastery before therapy is considered complete. The end result is the rigorous shaping of parenting practices.

Programs that alter parenting practices at their core may therefore offer real benefit down the line by protecting against further psychopathology. Such programs may be particularly worthwhile for parents of children with ADHD, especially where there is evidence of negative parenting practices, or where other risk factors for (e.g., family history of) or subthreshold symptoms of anxiety are present. As recommended earlier, future investigation of the way in which multiple variables interact over time to shape protection and risk will offer a clearer picture of the relative contribution of parenting, both relative to, and in conjunction with other factors. Such studies will be informative in assessing in which cases there is may be a discernable clinical need for such programs and in which cases the risk is more minimal. The present investigation revealed statistically significant associations among ADHD, anxiety, and parenting, with effect sizes generally ranging from small to moderate. Pending future research, parenting interventions in children with ADHD may be advisable for a broader group of families than currently receive this intervention. In the meantime, the present results only bolster the practice of prescribing these interventions for parents of children with ADHD.

Conclusion

Overall, the data from this series of studies provides support for the hypothesis that young children with ADHD elicit negative parenting characteristics, which in turn lead to subsequent anxiety disorders. This does not entirely explain the development of anxiety disorders among these children, but instead provides a partial account for how the

comorbidity occurs. As such, these findings provide incentive for future studies to examine the concurrent and preventative effect of parent training on anxiety disorders in children with ADHD.

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