This is to certify that the

thesis entitled

A Family Psychiatric Study of
ADHD DSM-IV Subtypes

presented by

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has been accepted towards fulfillment
of the requirements for

M.A. degree in Psychology

Major professor

Date December 11, 2002

A Family Psychiatric Study of ADHD DSM-IV Subtypes

By

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A THESIS

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

MASTER OF ARTS

Department of Psychology

2002

Abstract

A FAMILY PSYCHIATRIC STUDY OF ADHD DSM-IV SUBTYPES

By

JulieAnn Stawicki

The present study is the first to examine parent psychiatric diagnoses identified by structured interview of each parent in relation to prospectively defined DSM-IV ADHD subtypes in children. Information about family psychiatric history in relation to the DSM-IV subtypes of ADHD is relevant to theories of contextual and genetic pathways and to the issue of the validity of the subtype distinctions. Parents of children with (a) ADHD-combined type, (b) ADHD-inattentive type, and (c) control children completed a structured diagnostic interview (Diagnostic Interview Schedule for DSM-IV) and were compared on rates of ADHD and other non-ADHD disorders. Results replicated past findings in that parents of ADHD children were more likely to have ADHD themselves (21.4%) than were parents of control children (4.7%), even when child comorbid disorders were controlled. Also, this study found that DSM-IV ADHD subtypes in parents were not correlated with DSM-IV ADHD subtypes in children, as would be predicted if the subtypes were distinct disorders. Breaking new ground, gender differences in parent prevalence of ADHD were also found. Specifically, girls with ADHD were more likely to have a mother with ADHD (20.8%) compared with control girls (0%) whereas boys with ADHD were more likely to have a father with ADHD (22.6%) compared with control boys (0%). In addition, results suggest possible gender-specific transmission of ADHD.

Acknowledgements

I would like to thank my advisor, Joel Nigg, for all his time and effort spent helping me with this thesis. I would also like to thank the staff of the Child Attention Study for all their help and encouragement in the past 3 years. Also, I thank my committee for their comments on my thesis. Finally, I would like to thank my family and friends for their support throughout my education.

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INTRODUCTION

Overview of ADHD

Characteristics often associated with childhood Attention Deficit Hyperactivity Disorder (ADHD) include constantly moving from task-to-task, lacking concentration for necessary activities such as homework, inability to modify behavior to appropriate context, and interrupting others. The syndrome has a prevalence among school-aged children estimated to be as high as 6.3% in North America (Szatmari, 1992). Children with ADHD often have substantial functional difficulties, including significant problems in school achievement and family and peer relationships, in addition to increased likelihood of other psychiatric disorders.

In a review, Hinshaw (1992) noted that 20% of children with attention disorder showed significant underachievement in school compared to same-aged peers. He noted that they often score below normal IQ, and often have language and speech difficulties. When their academic achievement is related to their performance on IQ tests, nearly half of ADHD children have lower-than-expected achievement in reading, arithmetic, and spelling (Barkley, DuPaul, & McMurray, 1990).

ADHD children also have marked difficulties in social conduct and adjustment. Their impulsive and hyperactive behavior interferes with interactions with parents, siblings, teachers, and peers (Barkley, 1998). Mothers of hyperactive children rate them as more non-compliant and are observed to be more negative in their interactions with their children compared to mothers of controls (Gomez & Sanson, 1993). Hyperactive children have more daily serious quarrels with their siblings compared to controls (Taylor, Sandberg, Thorley, & Giles, 1991). Teachers rate hyperactive children as having severe problems mixing with same-aged peers (Taylor et al., 1991). They are

more often described as having problems in co-operating with others, following in the rules of games and, subsequently, are more often the victims of teasing by other children (Taylor et al., 1991).

ADHD often co-occurs with other disorders, including depression, anxiety, and learning disabilities (Biederman, Newcorn, & Sprich, 1991). Most salient is that many children with ADHD also exhibit other disruptive behavior disorders, specifically, Oppositional defiant disorder (ODD) and Conduct Disorder (CD) (APA, 1994). Both of these syndromes are characterized by disobedience and opposition to authority figures, and angry and resentful behaviors. ODD is often a precursor to CD. Almost all children with CD previously met criteria for ODD (Lahey & Loeber, 1996). Children with CD also demonstrate a pattern of behaviors that violate the basic rights of others or violate age-appropriate norms. Some features of CD include aggression to people and animals, destruction of property, and serious violations of rules (e.g., truancy and running away from home overnight) (APA, 1994). Unsurprisingly, children with ADHD who have comorbid ODD or CD experience even greater problems in social relationships with both family (Gomez & Sanson, 1993) and peers (Taylor et al., 1992) than children with ADHD alone.

A majority of children with ADHD often experience persistent impairing problems into adolescence and adulthood, with poor long-term outcomes. Approximately 50-80% of children continue to display impairing inattentive or hyperactive symptoms into adolescence (Barkley, 1998). These adolescents are more likely to be in more automobile accidents and to be injured and killed in accidents than same-aged peers (Barkley, et al. 1993). They are also more likely to be suspended or expelled from school

(Barkley, 1998).

Due to the significant social, academic, and family problems associated with ADHD, its chronicity, and frequent poor long-term outcomes, ADHD has garnered much recent attention in the medical and academic communities, as well as in the popular medias. In fact, ADHD has become one of the most extensively studied childhood psychiatric disorders (Cantwell, 1997). In short, it is a syndrome of substantial social and scientific concern. Whereas causal pathways to ADHD are incompletely understood, the field is particularly in need of information about the family environment of children with ADHD. Moreover, the validity of its current definition requires further investigation.

The present study attempts to shed light on the validity of the current definition of ADHD along with taking a first step in providing a more complete understanding of the casual pathways of ADHD, particularly in the area of family context. This study examines the prevalence of psychopathology in the parents of children with ADHD. Family psychiatric studies are a first step in examining the environment of children with ADHD and contribute in establishing the validity of a disorder and resolving heterogeneity. Heterogeneity and changes in definition within ADHD remain major factors impeding interpretation of past studies and necessitating new studies of family environment and family psychiatric history, therefore, important background issues in the definition of ADHD must first be considered.

Heterogeneity

Heterogeneity is an issue from several perspectives: historical differences in definition of ADHD; subtypes defined in DSM-IV; gender; family history; and psychiatric comorbidity. Each is briefly explained.

Recent History of Definitions of ADHD. Past studies of family psychiatric history have used varying definitions of ADHD depending on the edition of the DSM current at the time of the study. The earliest editions, DSM-I (APA, 1952) and DSM-II (APA, 1967) featured a prototypical formulation of Hyperactive Child Syndrome. The diagnosis was based on evidence of hyperactivity and distractibility. However, in the earliest editions of the DSM, hyperactive child syndrome was plagued with the same poor reliability as nearly all the other prototypes. This provoked a return to the Kraeplinian medical model in the DSM-III (Appendix A), which sought to offer a purely operational approach and utilized a polythetic symptom list to operationalize diagnostic criteria. The new criteria were intended to reflect directly observable behaviors. That modification in DSM-III (APA, 1980) greatly improved the reliability of the diagnoses, albeit with unknown effect on validity. The syndrome was renamed Attention Deficit Disorder and three symptom domains were identified: inattention, hyperactivity, and impulsivity. Two subtypes were defined for the first time. A diagnosis of attention deficit disorder [a] with or [b] without hyperactivity depended on the presence or absence of hyperactivity. However it is notable that children had to be both inattentive and impulsive to be diagnosed with either of the two subtypes.

Early behavioral studies of Attention Deficit disorder subtypes based on DSM-III criteria therefore examined children with inattentive and impulsive symptoms and then divided the groups based on the presence or absence of a specified number of hyperactive symptoms. These studies found characteristic behavioral differences between children with hyperactivity and those without hyperactivity, lending support to separating these two subtypes of ADHD (or ADD as it was then called). For example, children with

Attention Deficit with hyperactivity were more disinhibited, impulsive, disruptive, and more socially inappropriate, whereas Attention Deficit children without hyperactivity were more unmotivated, "day-dreamy," and apathetic (Barkley, DuPaul, & McMurray, 1990). Children with hyperactivity and children without hyperactivity also differed with regard to the pattern of comorbid problems exhibited. Children with attention problems and hyperactivity, as defined by the DSM-III criteria, had more instances of comorbid externalizing behavior disorders such as Conduct Disorder and Oppositional Defiant Disorder compared to purely inattentive children (Barkley, DuPaul, & McMurray, 1990). However, although family studies were conducted during that era, as reviewed later, no family studies compared the DSM-III subtypes in relation to frequency of parent psychiatric disorders.

In the DSM-III-Revised (APA, 1987), a diagnosis of attention deficit disorder was based on the total number of symptoms of hyperactivity, impulsivity, and inattention, regardless of which symptom area the child demonstrated most. Subtypes were not operationally defined. As a result, research on the subtypes slowed down substantially. Yet new data continued to support the validity of subtypes. They were therefore reinstated in revised form in the fourth edition of the DSM (APA, 1994).

ADHD Subtypes in the DSM-IV. Supported by extensive field trial data utilizing factor analytic and cluster analytic methods, the most recent version, DSM-IV (APA, 1994), defined three ADHD subtypes, based on two symptom domains: (1) inattention-disorganization and (2) hyperactivity-impulsivity. Children are classified into one of three subtypes according to the cluster of symptoms they exhibit. Thus, children can be classified as Primarily Inattentive subtype, Primarily Hyperactive subtype, or Combined

subtype, the latter meaning they exceeded thresholds for symptoms of both inattention and hyperactivity. Notably, the inattentive type children must be below threshold on hyperactivity/impulsivity and vice versa. Although early studies of children using DSM-III criteria suggested fundamental differences between hyperactive and non-hyperactive inattentive children, few studies have examined the validity of ADHD subtypes using the newer and revised DSM-IV criteria. None have done so with family psychiatric history of prospectively defined DSM-IV child subtypes, as detailed later. There is considerable but far from perfect agreement between DSM-III and DSM-IV criteria overall; approximately 79% of children diagnosed as ADHD with DSM-IV criteria would have been diagnosed as ADHD by DSM-III criteria (Lahey et al., 1994). However, the DSM-III and DSM-IV subtypes do not correspond as highly. There is 73% agreement between the diagnosis of DSM-III ADHD with hyperactivity and DSM-IV-Combined subtype, but only a 43% agreement between DSM-III ADHD without hyperactivity and DSM-IV-Inattentive subtype (Lahey et al., 1994). The majority of children diagnosed with DSM-III subtypes are also diagnosed with the corresponding DSM-IV subtype, but the DSM-IV Primarily Inattentive type identifies 56% more children than the DSM-III without hyperactive subtype identifies (Lahey et al., 1994). This underscores the need for further validation studies to evaluate the relation between the DSM-IV subtypes.

However, initial behavioral studies of children with the DSM-IV subtypes confirmed the expected behavioral differences between them. These included an earlier age-at-onset for the Combined subtype; gender differences in prevalence rates, with more males being diagnosed as Combined type than Inattentive type; and different rates of comorbid disorders associated with each subtype (reviewed by Milich et al., 2001).

Thus, behavioral studies have generally supported the validity of the DSM-IV subtype distinctions.

However, studies examining neuropsychological features of ADHD subtypes have yielded mixed results. These studies have compared the Combined type and the Primarily Inattentive type[1]. Faraone et al. (1998) found no differences between subtypes on IQ and academic measures: Houghton et al. (1999) concluded that the combined type had larger deficits on several neuropsychological tests. Nigg et al. (2001) found that on measures of response speed, planning, and interference control, both subtypes of ADHD children demonstrated significant deficits compared to the controls, but did not differ from one another. On a measure of behavior inhibition, differences between subtypes were dependent on gender effects: boys with ADHD-combined type performed worse than boys with ADHD-inattentive type, while there were no differences between girls with ADHD-combined type and girls with ADHD-inattentive type. These mixed findings among and within studies highlight the ambiguity of the validity of the current distinction between the subtypes, and underscore the need for additional data to shed light on the extent to which these ADHD subtypes are closely related or distinct.

Thus, although some behavioral differences between subtypes are accepted, it remains unknown whether these differences reflect distinct etiologies or whether they reflect different expressions of the same underlying processes or disorder. Family studies can inform that question. Yet the newest definitions of ADHD subtypes have not eliminated the debate over the heterogeneity of children diagnosed with ADHD (Hinshaw, 2001; Milich, Balentine, & Lynam, 2001), because several other types of heterogeneity remain to be evaluated.

Family history subtypes. Another view of heterogeneity concerns differences in whether children come from families in which relatives, particularly parents, have ADHD as well. Originally, August and Stewart (1983) defined a different set of ADHD subtypes based on such family history of ADHD: Family History-positive (FH+) meant that one biological parent had a history of ADHD, and Family History-negative (FH-) meant that neither biological parent had ADHD. They found that the FH+ children, as well as their siblings, had more conduct problems. The FH- group was more likely to have academic problems and learning disorders.

More recently Seidman and colleagues (1995) examined neuropsychological performance in children with ADHD FH+ versus children with ADHD FH-. Both ADHD groups were impaired on tests of neuropsychological functions compared to control children, but the FH+ group was more impaired than the FH- group. They also found, however, that child psychiatric comorbidity influenced neuropsychological functioning in addition to family history. Therefore, comorbidity is a source of heterogeneity for both the family history subtypes, as well as the DSM-IV subtypes.

Comorbidity disorders in children with ADHD. In studies of ADHD using DSM-III defined subtypes, different patterns of comorbidity were found depending on the presence or absence of child hyperactivity. Barkley, DuPaul, and McMurray (1990) found that children with Attention Deficit without Hyperactivity were more likely to have Major Depressive Disorder compared not only to controls but also compared to those children with Attention Deficit with Hyperactivity. Similar patterns of associated comorbidity were also found with DSM-III subtypes in relation to learning disorder (LD). As noted earlier, learning disorders are also commonly diagnosed in children with

ADHD. In a study using the DSM-III definitions of ADHD, children with Attention Deficit Disorder without Hyperactivity were more likely to be diagnosed with LD (Barkley et al. 1990). However more recent studies of differences in LD comorbidity rates for the DSM-IV subtypes have found mixed results. Some studies using the DSM-IV definitions have found similar rates of LD for each ADHD subtype (see Barkley, 1998 for a review).

However, as noted earlier, the most common comorbid disorders diagnosed with ADHD are other externalizing behavior disorders; rates for these comorbid disorders differ according to ADHD subtype. Using the DSM-III definitions of ADHD, children with Attention Deficit with Hyperactivity were more likely to also be diagnosed with CD or ODD compared to those children with Attention Deficit without Hyperactivity (Barkley, DuPaul, & McMurray, 1990).

This pattern has held up in the DSM-IV ADHD subtypes. For example, Lalonde, Turgay, and Hudson (1998) investigated the comorbidity patterns of the three ADHD subtypes with both CD and ODD. The majority of the sample ($N = 100$) consisted of children with ADHD combined type, (78%); inattentive type accounted for 15%; and hyperactive-impulsive type accounted for the remaining 7%. Of the children with combined type, 85% also had comorbid ODD and 8% had comorbid CD. Of those with the primarily inattentive type, 33% also were diagnosed with ODD, but none had CD. Of those children with the primarily hyperactive impulsive type, 100% had comorbid ODD and 57% also had comorbid CD. Across subtypes, 78% of the sample had ODD, 10% had CD. Such results suggest that controlling for such child comorbidity may be

essential when interpreting family history data. Unfortunately, very few family studies of ADHD have controlled for such comorbidity.

Gender Differences. Another source of heterogeneity that has been almost completely overlooked in the area of family prevalence studies is potential gender differences in subtype expression and in ADHD expression generally. Although boys are more frequently diagnosed with ADHD, with gender ratios of 6:1 (Barkley, 1998), many girls are affected. In addition, boys and girls apparently differ in the expression of the disorder. Girls with ADHD typically are more socially withdrawn and have more internalizing symptoms than boys with ADHD (Breen, 1989). Boys and girls have different ADHD subtype prevalence rates as well as different symptom manifestation; boys are more likely to be diagnosed with ADHD-Combined subtype, while girls are more likely to be diagnosed with ADHD-Inattentive subtype (Lahey et al., 1994). Also, boys are up to nine times more likely to be referred to a clinic than are girls (Barkley, 1998). Therefore, the use of clinic samples, as is the case for most studies of ADHD, may be misrepresenting differences between boys and girls. Accordingly, applying results found with primarily male samples to female samples may be overlooking important gender effects. As reviewed later, virtually no family psychiatric studies of ADHD have looked at results for girls.

Summary. Problems with heterogeneity, along with the evolving definition of ADHD, compound the difficulties in establishing an etiological pathway for ADHD. It is important that these issues be considered in family psychiatric studies. The present study therefore focused on DSM-IV ADHD subtypes, and included analyses of psychiatric comorbidity, family history subtypes, and gender effects in children and parents.

Consideration of developmental context in terms of family history can address possible causal factors as well as inform the validity of the current definition of ADHD subtypes.

Why a Family Prevalence Study?

Despite the extensive literature on ADHD, family studies of ADHD are relatively few (Cantwell, 1997). Yet family studies, especially family psychiatric studies, are an important first step in examining the environment of children with ADHD. In addition to providing clues to the developmental environment, family psychiatric studies also become relevant in their contribution to establishing the validity of a disorder and resolving heterogeneity.

The family environment is undoubtedly one of the most important factors in the development of a child. A substantial literature establishes the important role of family environment on children's development and psychopathology. For example, problems in the family ecology, such as low socioeconomic status (SES), marital discord, and family conflict are linked with psychopathology in children (Biederman et al., 1995). Frick (1994) reviewed dysfunction in families in relation to childhood ADHD. He concluded that ADHD was more prevalent in families where there was high marital conflict, one parent had antisocial or aggressive behavior, and one or both parents had a history of psychiatric disorder. Biederman et al. (1995) found that parental mental disorder was the most predictive of child problems in ADHD families.

Parental psychopathology in particular has ramifications throughout the family environment. For example, parental psychiatric problems may degrade parenting ability, rendering parents less consistent, warm, or attentive to children (Barkley, 1998). SES may be lowered by parent inability to perform at work, a frequent problem for adults with

ADHD (Barkley, 1998). Marital relationships may also be disrupted by parent psychiatric disorder. As these multiple examples suggest, risk factors may tend to cluster directly in families. This clustering, in turn, may be rooted in parent psychopathology in many cases. Thus, clarifying whether ADHD subtypes in DSM-IV differ in rates of parent psychiatric problems is an important first step in evaluating possible contextual differences in their development.

At the same time, transmission of psychiatric problems in families can provide clues to genetic transmission of risk for a disorder such as ADHD. Whereas the heritability of ADHD as an undifferentiated syndrome has been established in family, twin, and adoption studies (Sherman, Iacono, & McGue, 1997) whether DSM-IV subtypes transmit differently in families is largely unknown.

Overall parent psychopathological profiles can provide clues to possible etiological pathways and therefore may inform validity of subtype distinctions in ADHD. In psychiatric syndromes, construct validity reflects the extent to which the DSM-IV criteria capture etiological or developmental pathways to disturbance in adjustment. Family psychiatric history is a frequently recognized tool for obtaining clues to such pathways. Many different approaches have been proposed for illuminating the validity of medical as well as psychiatric syndromes (e.g., Cantwell & Baker, 1988; Feighner et al., 1972; for a further discussion, see Appendix A), but family psychiatric history is one reasonable component of all such efforts.

Thus a family psychiatric study can clarify whether particular disorders tend to cluster together or whether the disorder under study is common in relatives. Such data can provide clues as to possible shared etiologies. Despite all these advantages, adequate

family psychiatric studies of ADHD subtypes are in short supply. The few family studies of ADHD nearly all used earlier definitions of ADHD, and most are complicated by the failure to control for other disorders that frequently co-occur with ADHD (Tannock, 1998). Taken together, all of the omissions in past research (old definitions of ADHD, failure to control of comorbid disorders, and inadequate representation of girls) paint an incomplete picture of the families of children with ADHD. Nevertheless, a careful review of that literature is in order.

Family psychopathology studies of ADHD

Research in the area of family psychiatric history of ADHD children is relatively new; the first studies were done in the early 1970's, just after reliability was established for adult diagnoses. Since that time, only about a dozen studies have examined psychopathology in families of children with ADHD. Table I summarizes the studies to be discussed. As the table illustrates, nearly all studies used pre-DSM-IV definitions, focused more on boys, and failed to consider subtypes of ADHD. These studies are next reviewed in more detail.

ADHD in families. Early studies mainly established a pattern of family transmission of ADHD in families of children with ADHD. Cantwell (1972) found higher rate of hyperactivity in the parents of children with Hyperactive Child Syndrome than in the parents of controls. Cantwell also found higher prevalence rates of hyperactivity in second-degree relatives of hyperactive children (12 % of all male relatives, 6.3% of all relatives) compared to the relatives of controls (0.6%). In a review, Cantwell (1975) concluded hyperactivity was more prevalent in families of children with the disorder than controls; this was true for both first-degree relatives and extended

family members. Later studies of ADHD in families have replicated these findings.

Other disorders in ADHD families. In addition to increased rates of hyperactivity in the families of children with hyperactivity, early family studies also examined the rates of other disorders in the parents of children with hyperactivity. Morrison and Stewart (1971) compared the family psychiatric histories of children admitted to a hospital for behavioral problems with children who were admitted for medical procedures, such as an appendectomy. Parents of the hyperactive children had more diagnoses of alcoholism, sociopathy, and hysteria than the parents of controls. They also were more likely to report hyperactive symptoms during their own childhood, replicating Cantwell (1972). The results suggested a correlation between the presence of other psychiatric diagnoses in first-degree relatives and the presence of hyperactivity in children.

Later studies of familial transmission of ADHD and other psychiatric diagnoses have supported Morrison and Stewart's (1971) initial findings that parental psychopathology was associated with child ADHD. Biederman et al. (1995) found that chronic conflict, decreased family cohesion, and exposure to parental psychopathology, particularly maternal psychopathology, were more common in ADHD families than in control families. Children were diagnosed with ADHD using Achenbach's Child Behavior Checklist. A maternal self-report of their own history of psychiatric disorders was correlated with externalizing and internalizing behaviors in their children. Maternal psychiatric disorders were not reported in terms of individual disorders; rather only the total number of diagnoses was analyzed. This total was greater in the parents of children with ADHD than the parents of normal controls. The authors concluded that exposure to family conflict and maternal psychiatric symptoms may index important environmental

adversity indicators in ADHD families. Although that study, like earlier studies, supported a link between parental psychopathology and child ADHD using a more recent description of ADHD, it shared key weaknesses with earlier studies: comorbid disorders in children were not controlled, and ADHD subtypes were not defined.

Thus a critical question that arose from these studies concerned whether the elevated parental hyperactivity and other psychiatric diagnoses in families of ADHD children were specifically linked with child hyperactivity, or rather were linked with general maladjustment in children. To address that issue, Stewart et al. (1980) examined the rates of parental psychiatric diagnoses in families of children with hyperactivity compared to the families of children with other psychiatric diagnoses. Children who were admitted to psychiatric clinics with unsocialized, aggressive behavior, including hyperactivity and conduct disorders, were more likely to have fathers with antisocial personality disorder or alcoholism and mothers who were neurotic than children admitted for other psychological disorders such as depression. Importantly, when aggressive child behavior was statistically controlled, the difference in parental diagnoses between the families of the hyperactive children versus the children with other diagnoses was no longer significant. The rates of parental disorder in the families of children with hyperactivity without aggression were the same as in the parents of children with depression and anxiety. Hyperactivity in parents was not examined. Therefore Stewart and colleagues concluded that the association of antisocial and neurotic disorders in parents with hyperactivity in their children was not specific to child hyperactivity. Instead, increased parental psychiatric symptoms of Antisocial Personality Disorder,

alcoholism, and neuroticism were related to the presence of aggression in the children rather than the presence of hyperactivity per se.

However, Stewart et al.'s (1980) conclusions may not have been entirely warranted. From studies done in the DSM-III era, it was known that the presence of hyperactivity was associated with aggression (Hinshaw, 1987). Subsequently it has remained clear that hyperactivity commonly co-occurs with aggressive behavior disorders such as CD and ODD (Tannock, 1998). Crucially, Stewart et al.'s definition of aggression was very broad and included many hyperactive behaviors. Further, their diagnosis of "hyperactivity" was most similar to a diagnosis made with DSM-III-R: either hyperactivity or inattentiveness could be sufficient for a diagnosis. As a result, separating hyperactive boys by the presence or absence of aggression, as Stewart et al. did, may have simply created a group of hyperactive and a group of inattentive children by today's definition. Thus, it is unclear whether parental diagnoses were unrelated to child hyperactivity or rather unrelated to child inattentiveness. Therefore, it could be that children with ADHD combined type are more likely to have parents with psychiatric disorders; alternatively it could be true that parental psychopathology is simply correlated with the co-occurrence of CD/ODD in ADHD children. Fortunately, other studies have begun to address this issue more carefully.

Family studies controlling formally for the presence of CD and Aggression. Implied in the preceding section, understanding the specifically of the association between parental disorder and hyperactivity in children has been complicated by the high rates of aggressive and disruptive behavior problems in ADHD children. It therefore becomes important to consider the relationship between what are today called ODD and

CD in children and parental psychiatric disorder and whether these comorbid syndromes "explain" association of child ADHD with parental disorders other than with ADHD.

As a prelude, it has been shown that child ODD and CD also have characteristic familial psychiatric correlates. Schacher and Wachsmuth (1990) found that parent psychopathology was more prevalent in CD and ODD children compared to normal control families. Frick et al. (1992) found higher rates of paternal and maternal antisocial personality disorder and substance abuse in families of children with CD and ODD than in the control families (ADHD was not evaluated). The results point to the possibility that ODD and CD, if not controlled for, could be responsible for observed increased rates of non-ADHD psychopathology in families of ADHD children in the early studies.

Lahey, Piacentini, McBurnett, Stone, Hartdagen, and Hynd (1988) assessed psychopathology in parents of children with either ADHD or CD. All children in the study were outpatients referred to a local Psychological Clinic. Psychiatric diagnoses in the children were obtained through a semi-structured interview with one custodial biological parent, usually the mother, according to DSM-III criteria. Information for the DSM-III diagnoses of both parents was obtained from a structured diagnostic interview (SADS; Spitzer & Endicott, 1978) of the mother. Parents of children with CD alone were more likely than controls to exhibit antisocial personality disorder and substance abuse. ADHD in children was not associated with any disorder in parents. However, children with the comorbid diagnosis of ADHD+CD had fathers with elevated rates of aggression, arrest, and imprisonment compared to the fathers of children with ADHD alone or fathers of children with CD alone. This appeared to suggest that ADHD alone was not associated with <u>any</u> disorder in the parents. Rather it was the presence of CD that was

correlated with parental disorder and the combination of the CD and ADHD in children that predict the most associated non-ADHD disorders in fathers.

However that study had two crucial weaknesses. First, ADHD in the parents was not assessed. Second, child ADHD and CD were associated with the rate of disorder in the father; yet, diagnoses for the child and both parents were obtained only from the mother report. Mothers could be biased in their reporting of symptoms in the father or the mothers of children with conduct problems might be more attuned to the behaviors in their partners.

Frick and colleagues (1991) also examined how controlling for the presence of child CD affects the rate of childhood behavior problems in the relatives of children with ADHD. Children were 96 boys recruited at the Western Psychiatric Institute. Each boy, his parent, and his teacher were all interviewed using the Diagnostic Interview Schedule for Children (DISC; Costello, Edelbrock, Kalas, & Dulcan, 1984) based on DSM-III-R criteria. Diagnoses for parents and second-degree relatives were made using family history questionnaires completed by one of the biological parents, most often the mother of the boy. Boys with ADHD were significantly more likely to have a mother, father, one relative, or two relatives who exhibited ADHD symptoms during childhood. Fathers of boys with CD were more likely to have used substances (tobacco, alcohol, or drugs) before the age of 15 or to have had CD themselves. Boys with comorbid ADHD+CD had relatives with elevated levels of hyperactivity similar to the ADHD alone group. Relatives of boys with ADHD plus CD also had high rates of CD and substance use similar to the parents of boys with CD alone. Frick et al. did not find that the children ADHD plus CD had higher ratings of hyperactivity in the children than ADHD alone,

suggesting that the ADHD plus CD group was not simply a more severe ADHD group. Therefore, ADHD in parents was associated with ADHD in children and parental substance abuse was associated with CD in children. The higher number of disorders in parents of children with ADHD plus CD was due to the co-occurrence of the two disorders in the child; CD and ADHD in children each predicted separate psychiatric disorders in parents.

These studies taken together suggest two different possibilities for association between ADHD comorbidity and parental disorder. One possibility as presented by Lahey et al. (1988) is that child ADHD alone is not associated with non-ADHD disorders in the parents of these children, but when ADHD and CD co-occur, the severity of non-ADHD parent disorders is qualitatively worse than would be suggested by either the presence of ADHD or CD alone. Because Lahey et al. did not assess for ADHD in parents of the children, it remains unclear whether the combination of ADHD + CD in children would also predict a higher rate of ADHD in their parents. This differs from the results presented by Frick et al. (1991), which suggest that it is a diagnosis of ADHD and CD in children that predict distinct disorders in the parents. The associated disorders in parents of children with both ADHD and CD were merely the additive affect of what would be predicted by each of the child disorders alone. The relationship between parent psychopathology and childhood ADHD and childhood CD is not clear; the presence of ADHD and CD could represent the simple co-occurrence of 2 disorders, or ADHD plus CD could be a distinct syndrome with unique correlates. As a result of this ambiguity, it is essential to control for comorbidity with CD when interpreting the relationship between child ADHD and parent disorder.

Kuhne, Schachar, and Tannock (1997) examined the effects of comorbid child CD and ODD and severity of child ADHD symptoms and aggression, in relation to maternal reports of their own experience with psychiatric symptoms, as assessed using the Symptom Checklist. Child diagnoses were made using questionnaires completed by parents and teachers based on DSM-III-R criteria. ADHD symptoms were more severe in both ADHD+ODD and ADHD+CD children compared to ADHD only children. Mothers rated their children with ADHD+CD as more aggressive than the mothers of either children with ADHD or children with ADHD+ODD had rated them. Mothers of the ADHD+CD children had elevated levels of self-report psychopathology in areas of interpersonal sensitivity, anxiety, and hostility compared to mothers in the ADHD-only group. These findings are generally consistent with earlier findings by Lahey et al. (1988) and Frick et al. (1991).

Faraone et al. (1997) examined rates of ADHD, ODD, CD, and Antisocial Personality disorder in the parents, siblings, and other relatives of controls and of children with either ADHD alone or ADHD and comorbid externalizing disorder. They found that relatives of all three child groups (ADHD+CD, ADHD+ODD, and ADHD alone) were all at greater risk for both ADHD and ODD compared to controls. However only the relatives of the ADHD+CD group had an increased risk of CD and antisocial personality disorder. The relatives of the ADHD+ODD and ADHD only did not differ in rates of ADHD, ODD, CD, or antisocial personality disorder.

Nigg and Hinshaw (1998) looked more specifically at the presence of ADHD along with other psychiatric diagnoses and their associated features in parents of children with ADHD. The participants in the experimental groups were the parents of boys

enrolled in a summer research program; boys were diagnosed with ADHD (defined by DSM-III-R). The control group was parents of boys without ADHD. Both groups were recruited from the community. All children were also assessed for comorbid CD and ODD using parent rating scales data. Parent ADHD diagnosis was made using direct the Wender Utah questionnaire (Wender, 1985), other non-ADHD diagnoses were determined using the Quick Diagnostic Interview Schedule (QDIS). The children were then initially placed in one of three groups: comparison, ADHD, and ADHD+ODD/CD. The mothers in both ADHD groups had higher rates of recent maternal depression: 25% of the mothers of the ADHD group and 39% of mothers of the ADHD+ODD/CD group compared to 0% of mothers of the comparison group. When the parents of the boys with ADHD+CD were examined apart from ADHD+ODD, the mothers of the boys with ADHD+CD had higher rates of childhood ADHD than all other groups, including the mothers of ADHD+ODD children. Fathers of the ADHD group (41%) were more likely than control fathers (0%) to have a childhood history of ADHD, based on the Wender rating scales. Furthermore, the fathers of the boys with ADHD+CD were more likely to have antisocial personality disorder as compared to all other fathers.

DSM-IV familial subtypes? In a study to determine whether or not the subtypes of ADHD in families were the same as those found in children, Faraone, Biederman, and Friedman (2000a) examined ADHD subtypes in relatives of boys with ADHD. Children for the study were recruited from referrals to a psychiatric clinic. ADHD diagnoses were initially made using DSM-III-R criteria. The investigators estimated DSM-IV subtypes retrospectively using a proxy procedure to estimates DSM-IV subtype from DSM-III-R data. The relatives of boys with ADHD were more likely to have had ADHD themselves,

replicating previous studies. However, relatives were not more likely to have the same subtype as their child. In other words, relatives of boys with combined-type ADHD were not more likely to have combined-type themselves. The authors concluded that the child subtypes of ADHD are not related to familial or genetic differences, but rather are different manifestations of one disorder that may vary according to environmental influences. The latter reflected potentially important evidence in opposition to the conclusions of Milich et al. (2001), who argued that the inattentive type is a distinct disorder.

However the study by Faraone et al. (2000a) had key limitations. Most important, the study was not done with actual prospectively assessed DSM-IV criteria, but rather by approximating DSM-IV subtypes from the DSM-III-R criterion set. Secondly, the children were from a clinic-recruited sample, which may make results vulnerable to referral bias (Goodman et al., 1997). Third, parents were not independently examined; rather all the relatives were combined in the analyses. Finally, all diagnoses in the relatives were determined from a family history questionnaire filled out by the mother of the participants, rather than by direct structured interview of each parents.

A similar study was also completed with the families of girls. Faraone and colleagues (2000b) looked at the rates of ADHD subtypes in relatives of girls diagnosed with ADHD. The girls for this study were recruited from the same psychiatric clinic as reported in Faraone et al. (2000a). As was the case in the previous study, higher rates of ADHD were found in relatives of girls with ADHD; but the relatives of girls with ADHD were <u>not</u> more likely to be diagnosed with the same subtype as diagnosed in the child. One advantage of this study is that diagnoses in the relatives of the girls were assessed

using semi-structured clinical interviews of each relative. However, this study also suffered from the same key limitations as Faraone et al. (2000a). Specifically, (1) girls with ADHD were diagnosed using DSM-III-R, and DSM-IV subtypes were approximated, (2) the sample was clinic-referred, and (3) results of analyses were reported for all relatives, but not for mothers and fathers separately.

In addition, the authors concluded that their findings demonstrate that familial transmission of ADHD in girls is similar to the families of boys, and therefore, previous studies using male samples generalizes to girls with ADHD. This broad conclusion was probably premature for several reasons, including the methodological limitations already noted. Even by the authors' own report, this is the only known study of familial transmission of ADHD in girls with ADHD (Faraone et al., 2000b); the extent of generalizability between results of studies using male samples to female populations warrants replication and extension.

Comment on Past Studies

These studies taken together suggest an association between child ADHD and parental psychopathology. Specifically the parents of children with ADHD were more likely to exhibit ADHD themselves. Along with having ADHD, mothers of children with ADHD were also more likely to have been diagnosed with depression. Fathers of ADHD children are more likely to have antisocial personality disorder. However, the relationship between child ADHD and parental non-ADHD psychopathology was not straightforward; parental disorder was also associated with other disruptive behavior disorders. Parents of children with ADHD plus ODD or CD were more likely to exhibit more antisocial and hyperactive symptoms than the parents of children with ADHD

alone. Mothers of children with ADHD were more likely to be depressed than the mothers of control children. However mothers of children with ADHD plus CD were even more likely to be depressed than mothers of children with ADHD alone.

Taking these studies together, it appears that the co-occurrence of ADHD plus CD in children could suggest a unique, severe combination with different parental correlates greater than simply the sum of these found in either CD/ODD alone or ADHD alone. Stewart et al.'s (1980) experimental group (labeled as hyperactive and aggressive) could have represented this severe sub-group.

All of the previous studies were done using previous editions of the DSM, prior to the DSM-IV; many were done with the DSM-III-R. Only two studies even considered the subtypes found in the DSM-IV (none even examined DSM-III subtypes), yet the DSM-IV subtypes differentially co-vary with comorbid disorders. The relationship between parental psychopathology and DSM-IV ADHD subtypes therefore becomes an important question, for which insufficient data exist.

What we know about family context and validity of ADHD subtypes is incomplete. Family psychiatric studies provide one type of data that can be informative. Past studies found that children with ADHD were more likely to have parents with ADHD. In general, although raw proportions were not reported in all studies, rates of ADHD in parents were 3 to 8 times higher for ADHD than control children. Table 2 summarizes these data. It indicates that there was considerable variability. However these rates suggested guidelines for what to anticipate in the current DSM-IV study.

A crucial point is that no prior study examined subtypes of ADHD prospectively assessed using the DSM-IV. This gap in the literature leaves questions of both family

environmental effects and current subtype validity unanswered. In addition, several other limitations exist within the relatively small family psychiatric literature. In addition to outdated diagnostic criteria, many of the studies used samples that were 100% male. In addition to contributing to the under-representation of girls in ADHD studies, clinic-recruited samples have other limitations. Many times, clinic-recruited samples are not representative of youth with mental disorders. Clinic-recruited samples are more impaired, less competent, more likely to have comorbid disorders, and more likely to be non-Hispanic white (Goodman et al., 1997). Therefore, previous research needs to be balanced using community recruited samples of children.

Summary

The ADHD subtypes apparently differ in social adjustment and associated behavioral problems. It is less clear, however, whether these differences indicate different underlying etiological factors and possible different disorders (Milich et al., 2001) or rather different manifestations of the same underlying disorder of attention (Faraone et al., 2000). Finding patterns of ADHD subtype transmission in families would lend support to continued theoretical and clinical differentiation between subtypes and possible different etiologies, whereas replication of no difference (as in Faraone et al.) could counter that argument.

Children diagnosed with different subtypes of ADHD have differing rates of associated disorders, with hyperactive children being more likely than inattentive children to receive an additional externalizing disorder diagnosis, such as CD or ODD. ADHD children with comorbid CD were more likely to have parents with both ADHD and CD themselves. Paralleling that profile, exposure to other types of parental

psychopathology was also more common in ADHD families compared to control families, in several studies. Table 3 summarizes these findings across all studies. Determining whether parents of ADHD children defined by DSM-IV subtypes have distinct patterns of psychiatric disorder, other than ADHD, would clarify further whether the differentiation of the subtypes is a reflection of etiology differences. If the subtypes of ADHD are different disorders, it would be expected that children with different ADHD subtypes would have parents with different patterns of psychopathology. If the latter case is true, and in fact the two subtypes do not differ, parents of all ADHD children, regardless of subtype, would show similar patterns of psychopathology.

It is important to understand the relationship between ADHD subtypes in children and (a) parental disorder along with (b) comorbid disorders in the children. It has already been shown that child CD and ODD have their own associated parent psychopathologies. Thus it is crucial to distinguish parental disorders correlated with child ADHD from parental disorders correlated with other comorbid child disorders. There has been a sparse history of research in the area of transmission of disorder in the families of children with ADHD. There have been no studies that examine the diagnosis of prospectively defined DSM-IV ADHD subtypes in children and the correlated disorders in parents.

Current Study

Although past studies have shown a link between parent ADHD and child ADHD, no studies have been done using prospectively defined DSM-IV child diagnosis. In addition, many of the studies failed to include girls in the study and nearly all used a clinic-recruited sample. Further, many relied solely on maternal self-report of psychiatric disorder in both parents, and few used structured interviews. The present study examined the relationship between DSM-IV ADHD subtypes in children and parental psychopathology, while correcting limitations in the literature. Specifically it looked at the association between child DSM-IV ADHD subtypes and the rates of DSM-IV ADHD subtypes and other diagnoses in parents. Due to the relatively low prevalence of the hyperactive subtype, only two child DSM-IV ADHD subtypes were considered, ADHD-combined type and ADHD-inattentive type.

Thus this was the first family psychiatric study of ADHD using children prospectively diagnosed by DSM-IV criteria. The sample included both boys and girls recruited from the community. Both biological parents were asked to report their own psychological history in separate structured clinical interviews. Child symptoms were rated by parent and teacher report.

Primary Hypotheses.

Following the logic just summarized on the previous page, the following hypotheses were tested.

Hypothesis I: Comparing Parents of Children with ADHD-any type.

(I-A) Parent ADHD (any type). In order to replicate past studies (see Table 2) it was predicted that the parents of children with ADHD (any type) would have a higher

prevalence of ADHD (any type) themselves as compared to the parents of the control children.

(I-B) Parent other (non-ADHD) disorders. It was also predicted that the rates of psychiatric diagnoses would be higher in the parents of children with ADHD (any type) as compared with the parents of control children.

Hypothesis II: Comparing Parents of Children with and without comorbid ODD/CD

In order to control for the effects of child comorbidity, rates of ADHD and other non-ADHD diagnoses in parents of children with ADHD plus ODD/CD were compared with diagnoses in parents of children with ADHD alone and parents of control children.

(II-A) Parent ADHD (any type). It was hypothesized that the parents of children with ADHD alone and parents of ADHD + ODD/CD would both have a higher prevalence of ADHD than parents of control children.

(II-B) Parent ADHD subtypes. Although it was predicted that the both parents of the child ADHD groups (ADHD alone and ADHD+ODD/CD) would have higher rates of ADHD (any type), it was predicted that there would be no differences between the parents of the child ADHD groups on rates of specific ADHD subtypes.

(II-C) Parent other (non-ADHD) disorders. It was also predicted that the parents of children with ADHD alone and parents of ADHD+ODD/CD would have higher rates of both subtypes of ADHD compared with controls. However there was no predicted difference between the parents of the two child ADHD groups (ADHD alone and ADHD+ODD/CD) for prevalence of subtype.

Hypothesis III: Comparing Parents of Children with different DSM-IV ADHD subtypes

Finally and centrally, ADHD subtypes were also examined. The hypothesis that ADHD-combined subtype and ADHD-inattentive subtype are distinct disorders (as presented in Milich et al., 2001) was tested by three predictions.

(III-A) Parent ADHD (any type). As a preliminary test, it was predicted that the parents of children with ADHD-Combined type and of children with ADHD-Inattentive type would have a higher prevalence of ADHD (any type) themselves when compared with the parents of control children.

(III-B) Parent ADHD subtypes. It was also predicted that the diagnosis of ADHD subtype in the children would be the same as the ADHD subtype in the parents. Specifically [1] parents of children with ADHD inattentive type will be more likely to have ADHD inattentive type compared with parents of control children and parents of children with ADHD-combined type. Also, [2] parents of children with ADHD-combined type will be more likely to have ADHD-combined type, compared with parents of children with ADHD-inattentive type or parents of controls.

(III-C) Parent other (non-ADHD) disorders. Child ADHD subtypes will differ in rates of parent other (non-ADHD) disorders.

Secondary Analyses

I. Since there are relatively few studies of childhood ADHD and parental psychopathology, and no studies of DSM-IV ADHD subtypes and other psychopathology, additional analyses explored the relationship between child ADHD and key individual psychiatric diagnoses in parents, including Major Depression, Generalized

Anxiety Disorder, Antisocial personality disorder, and Alcohol and Drug dependence. In addition, the relationship between specific diagnoses in parents and ADHD subtypes in children was also examined.

II. Almost every previous study failed to include girls (see Table 1). The one study that specifically used an all female sample did not use prospectively diagnosed DSM-IV ADHD, nor did they look at mothers or fathers separately to explore unique gender interactions. Therefore, additional analyses separating girls and boys were conducted to explore the differences between genders. These were folded in as secondary analyses within each of the hypotheses listed earlier.

Methods

Overview

Data were gathered from children and their biological parents who participated in the Michigan State University Child Attention Study directed by Dr. Nigg. The families were community-recruited from the Lansing area. They completed a multi-stage screening procedure (detailed below) before they were invited to participate in a six-hour battery broken into two visits of three hours. The present report used data collected through 2001 with a total *N* of 144 families participating. The author has been closely involved with the project for over two years, assisting with data collection.

Participants

Two groups of children and their biological parents were included in the study. Adoptive and stepparents were excluded from the study to hold consistent the possibility of genetic transmission. The children were initially included in one of two groups: [1] children diagnosed with ADHD, any type (n = 95) and [2] control children (n =49). A total N = 245 parents were available for the study. They included n =159 parents (n= 90 mothers and n = 69 fathers) of children with ADHD and n = 86 parents (n = 49 mothers and n = 37 fathers) of control children. For additional analyses, children with ADHD were divided into several groups depending on hypothesis. For hypothesis II, ADHD children were divided into groups according to comorbidity: [1] children with ADHD only (n = 48) and [2] children with ADHD plus ODD/CD (n = 46). For hypothesis III, the original group of children with ADHD (n = 95) was then restratified according to DSM-IV ADHD subtypes: [1] children with ADHD Inattentive subtype (n = 28) and [2] children with ADHD Combined type (n = 66). Additional demographic information for

children and their parents (i.e. age, ethnicity) is included in Table 4 and further discussed in the Results section later. All participants were recruited into the study and diagnosed using the screening process described in the following section.

Procedure

Initial Recruitment. All parents of third, fourth, and fifth graders in the Lansing school district were contacted through mass mailings with information about the study. In addition, advertisements were placed in local newspapers. Families were also recruited through local support groups and advertisements in local pediatric clinics. Although the majority of the sample was community-recruited, a minority of ADHD children (n=12) was recruited from a local pediatric clinic specializing in ADHD. Control subjects for those children were recruited from a general pediatric clinic in the same facility. Those parents who responded to the mailing or newspaper advertisement completed prescreening to determine the eligibility of their child for the study. A total of $N = 836$ families either called or sent in postcards expressing interest in participating in the study.

Multi-stage recruitment process: Screen. Those ($N = 836$) were then screened in several stages. First the family was contacted by phone and had to meet certain qualifications. These included a willingness to keep the child off psychostimulant medication for 24 hours prior to the visit for short-acting medication and 48 hours prior to the visit for slow-acting medication. English was required to be the first language of both parents and the child. The initial phone screen also precluded any children with neurological impairments, seizure history, head injury, other major medical conditions, or those taking slow-acting psychotropic medication (e.g., antidepressant medication)

according to parent report. Children were also excluded if they had a prior diagnosis of Mental retardation or autism. If the families were not screened out by phone, both the child's parents and schoolteachers were invited to complete normative behavior rating scales ($N = 455$). Those children who exceeded empirically supported cutoffs on at least one normed parent rating scale and one normed teacher rating scale (described later) were eligible as possible ADHD. Children whose scores fell below empirically supported cutoffs on all normed rating scales were eligible as possible controls.

Eligible children and their mother or primary caregiver were then invited to attend an on-campus diagnostic screening visit, during which the child completed the Wechsler Intelligence Scale for Children-third edition (WISC-III, Wechsler, 1991) and the Wechsler Individual Achievement Test-Reading (WIAT, Wechsler, 1992) and the mother completed the Diagnostic Interview Schedule for Children (DISC-IV; Shaffer, Fisher, & Lucas, 1997). Children who had an IQ above 75 on the WISC-III, and either (a) met criteria for a diagnosis of ADHD (as defined below) or (b) did not meet criteria for ADHD, ODD or CD were invited, along with their parents, to participate in the stud ($N =$ 155).

Once a family was admitted into the study, the biological parents, the child, and a sibling participated in two additional visits; each visit was three hours long. The children completed a neuropsychological battery (reported elsewhere), in addition to filling out questionnaires. The parents also completed a neuropsychological battery (reported elsewhere), questionnaires (described below) and a structured diagnostic interview about their own psychiatric history. The interview, the Diagnostic Interview Schedule, is also described in detail later.

Measures

Child Diagnostic Assignment Measures.

Pre-Screen. The initial prescreen measures alluded to earlier were a DSM-IV symptom rating scale, a broadband behavior rating scale, and a disruptive behavior rating scale. For data collected from 1997-1999 these were the Parent and Teacher DSM-IV Checklist (SNAP-IV Swanson et al., 1998), the Child Behavior Checklist (CBCL; Achenbach, 1991a), the Teacher Report Form (TRF; Achenbach, 1991b) and the Parent and Teacher Conners' Rating Scales-Revised-Short Forms (Conners; Conners, 1997). Data collected from 1999-2001 used the School and Home versions of the ADHD Rating Scales (DuPaul, Power, Anastopoulos & Reid, 1998), the Parent and Teacher Behavior Assessment System for Children (BASC; Reynolds & Kamphaus, 1992), and the Parent and Teacher Conners' Rating Scales-Revised-Short Forms (Conners; Conners, 1997). The ADHD rating scales and the BASC replaced the SNAP-IV and the CBCL, respectively, because they had superior, empirically validated cut-off scores while providing comparable symptom coverage.

Psychometric characteristics of prescreen measures. The test-retest reliability for the School and Home versions of the ADHD Rating Scales were .90 and .85 respectively (DuPaul et al., 1998). The validity coefficient between the Hyperactivity-Impulsivity scale of the School version of the ADHD Rating Scale and the Hyperactivity scale of the Conners Teacher Rating Scale-48 was .79. For the same questionnaires respectively, the validity coefficient between the Inattention scale and the Daydream-Attention Problems

scale was .85. The validity coefficient between the Hyperactivity-Impulsivity scale of the Home version of the ADHD Rating Scale and the Impulsivity-Hyperactivity scale of the Conners Parent Rating Scale-Revised was .78 (DuPaul et al., 1998). Internal reliability (alpha) can be reported from our own data. Internal reliability for the parent version of the ADHD rating scales was .98. The school version's reliability was .97. Agreement between the Home and School versions was .98.

The 15-day test-retest reliability of the CBCL was .90 for the attention problems scale. With respect to validity, the correlation was .86 between the CBCL aggressive behavior and the Conners' conduct problems scales (Achenbach, 1991a). Internal reliability for our own data was acceptable with alpha equal to .87.

The 15-day test-retest reliability of the TRF was .96 for the attention problem scales. In terms of validity of the TRF, the correlations were .80 between the TRF attention and the Conners' inattention-passivity scales, .67 between the TRF aggressive behavior and the Conners' conduct problem scales, and .83 between the two measures total problems scales (Achenbach, 1991b).

With respect to the reliability of the Parent and Teacher BASC, the one-month test-retest reliabilities for the Teacher BASC attention problems, hyperactivity, and aggression scales were .92, .92, and .91 respectively (Reynolds & Kamphaus, 1992). The one month test-retest reliabilities for the Parent BASC attention problems, hyperactivity, and aggression scales were .92, .84, and .69 respectively (Reynolds & Kamphaus, 1992). In terms of the validity of the BASC, the correlations were .78 between the Parent BASC attention problems and the CBCL attention problems scales, .82 between the Parent

BASC aggression and the CBCL aggressive behavior scales, and .52 between the Parent BASC anxiety and the CBCL anxious/depressed scales (Reynolds & Kamphaus, 1992).

For the Parent CONNERS, the test-retest reliability coefficients were .62, .85, and .72 for the oppositional, hyperactive, and ADHD scales (Conners, 1997). Internal reliability from our study for the parent CONNERS had alpha equal to .98 for the ADHD scale. For the Teacher CONNERS, the test-retest reliability coefficients were .84, .72, and .80 for these scales respectively (Conners, 1997). Our study's internal reliability for the Teacher CONNERS ADHD scale was alpha = .97. With respect to the validity of the Parent CONNERS the correlations between the short and long factor-derived scales were .98 and .97 for males and .97 and .97 for females for the oppositional and hyperactive problems scales respectively (Conners, 1997). These correlations for the Teacher CONNERS were .99 and 1.00 for both males and females (Conners, 1997). In summary, the screen scales are widely used scales with more than adequate reliability and validity.

Screening Visit. After screen-in cut-offs as possible ADHD were exceeded, diagnoses of ADHD were confirmed with the help of the National Institutes of Health Diagnostic Interview Schedule for Children (DISC-IV; Shaffer, Fisher & Lucas, 1997) completed with the mother or primary caregiver. The DISC-IV is a computer-guided, structured interview that assesses onset, duration, and impairment criterion from the DSM-IV as well as assessing individual symptoms of disorder. A graduate student interviewer who first completed 10 hours of training administered the DISC-IV. The quality of the interviews was checked by having the interview video recorded and 10 % reviewed by a certified trainer (either Dr. Nigg or Dr. Fisher at Colombia University).

DISC-IV. With regard to the reliability of the DISC, test-retest reliability coefficient for ADHD diagnosis was .80. The reliability coefficients for ODD was .73; .59 for CD. The reliability for Depressive Disorders and Anxiety Disorders were .56 and .64 respectively (Schwab-Stone et al., 1996). In terms of validity, the correlations with clinician symptom ratings were .82 for ADHD, .73 for ODD, .49 for CD, .67 for depressive disorders, and .53 for anxiety disorders (Schwab-Stone et al.). In summary, the DISC-IV is a widely accepted and a cited research diagnostic interview with acceptable reliability and validity for ADHD.

Final Diagnosis. Children who scored above the 80th percentile on at least one parent ADHD rating scales and above the 90th percentile on at least one teacher ADHD rating scales were considered as possible ADHD. The diagnosis was confirmed using the DISC-IV supplemented with an "or" algorithm. If children met age of onset, duration, impairment, and cross-situational criteria, then the diagnostic assignment was determined by adding the endorsed symptoms of the DISC-IV with the teacher reported symptoms to establish the final ADHD subtype. Thus if either teacher or parent reported the symptom as present, it was counted as present. This method was chosen to approximate the DSM-IV field trial data (Lahey et al., 1994). Which indicated maximal validity for the DSM-IV symptom count cut-offs using an "or" algorithm.

Control children failed to meet cut-offs for all parent and teacher ADHD rating scales at the 80th percentile. In addition, they exhibited 4 or fewer symptoms of ADHD by the "or" algorithm. Those children exhibiting 5 symptoms of overactivity or inattention by the "or" algorithm were excluded from all groups based on field trial data indicating that borderline cases might have ADHD (Lahey et al., 1994). Controls also

had to have never been diagnosed with ADHD in the past and could not have a sibling diagnosed with ADHD. Prior parent diagnoses were not considered in the screen process.

Comorbid Child Diagnoses. The DISC-IV interview was used for establishing the presence of child Oppositional Defiant Disorder and Conduct Disorder by DSM-IV criteria.

Parental Diagnostic Assignment

ADHD in parents. Because DSM-IV does not provide adult-specific diagnostic criteria for ADHD, parents completed a structured diagnostic interview of their childhood behavior as well as current behavior. As a supplement, parents also completed a series of normative self-report questionnaires concerning adult behaviors related to problems with attention, activity, and impulsivity. The questionnaires and the interview ask participants to retroactively assess ADHD symptoms in their childhood, as well as current symptoms. Participants completed the Self-SNAP, a revised version of the Swanson, Nolan, and Pelham DSM-IV ADHD rating scale (SNAP-IV) (1998). This version was revised using the same language as the original SNAP-IV in order for the parents to retrospectively rate their own behavior. All participants were also given the ADHD module of the Diagnostic Interview Schedule-IV (DIS-IV), based on DSM-IV (1994) ADHD criteria. The DIS-IV asks parents to report on their own childhood symptoms of ADHD as well as any symptoms of the disorder during adulthood. It also assesses impairment and age of onset.

A total of $N = 160$ parents completed both the DIS-IV and the Self-SNAP. Agreement between the instruments for diagnosing the presence of ADHD (any type) was 90.0% (Kappa = .58, $p < .001$). For the diagnosis of the DSM-IV ADHD subtypes,

the instruments had 94.0% agreement on the diagnosis of ADHD-inattentive type (Kappa = .642, p = .000) and 96.0% agreement on the diagnosis of ADHD-combined type (Kappa = .480, p = .000). A final diagnosis of ADHD and DSM-IV subtype in parents was defined when criteria for that subtype in the parent's own childhood was met on the DIS-IV. Diagnoses were made for the parents were based on parents' retrospective report of their own symptoms in childhood. Individuals must report at least 5 or more symptoms of inattention or hyperactivity[2]. Impairment on the DIS-IV must be rated moderate or severe, have a reported age of onset before 7 years. Parents who were just shy of meeting diagnostic criteria (4 symptoms of either inattention or hyperactivity) were excluded from parent ADHD analyses (n = 2). DSM-IV subtypes were determined if criteria was meet for one area, but met 3 or fewer symptoms in the other area. Parents who met criteria for one symptom area, but endorsed 4 symptoms in the other were counted as positive for ADHD (any type) but were excluded from DSM-IV subtype analysis (n = 2). Due to constraints on the number of testers and total length of the testing battery, n = 28 parents did not complete the DIS-IV. For those parents, diagnoses were based on endorsed symptoms on the Self-Snap, justified by the acceptable agreement between the instruments.

Parental Diagnostic Assignment-other disorders. Other psychiatric disorders in assessed parents were: Generalized Anxiety Disorder (GAD), Major Depression Disorder (MDD), Dysthymia, Bipolar Disorder, Antisocial Personality Disorder (ASPD), Alcohol Abuse, and Drug Abuse. All were assigned on the basis of the DIS-IV interview. However some parents used a DSM-III-R version for the diagnosis, as explained next.

DIS and QDIS. In addition to the DIS-IV ADHD module, each parent also completed either the rest of Diagnostic Interview Schedule-IV (DIS-IV) or the Quick Diagnostic Interview Schedule (QDIS) for the DSM-III-R in order to assess current and lifetime occurrence of non-ADHD psychiatric disorders. The two are versions that are computer assisted, structured interviews administered by trained interviewers. The interview is divided into modules based on diagnostic categories. Each module follows a Probe Flow Chart in which each question is followed up by a series of probe questions exploring severity and alternative explanations (e.g. medical causes) of any symptom. Each question appears on the screen to be read by the interviewer exactly as written. The interviewee responds to most questions with a simple yes or no answer recorded by the interviewer, and the next question to be asked appears on the screen. The Q-DIS-III-R assessed DSM-III-R diagnoses; once a disorder was either established or ruled out, the interview moved to questions about the next disorder rather than assessing all the symptoms of a disorder. $N = 39$ parents completed the Q-DIS-III-R, which was utilized early in the project due to the lack of availability of the DIS-IV program for DSM-IV. The DIS-IV became available in 1998 and was then used.

Although for most disorders, DSM-III-R and DSM-IV diagnostic criteria are similar, analyses were conducted to evaluate whether the rates of diagnoses were significantly different based on type of interview used. Complete results of those comparisons are provided in Appendix B. In brief, 2x2 chi-square comparisons were done comparing the frequency of each disorder assessed in the DIS-IV and QDIS-III-R (GAD, Dysthymia, drug dependence, ASPD, Alcohol dependence, depression). Overall, no differences in prevalence were found between versions at less than $p = .31$. An

additional 2x2 Chi-square was done to compare the presence or absence of any disorder across the two versions. Again, no difference was found ($\chi^2(1) = .803, p = .370$). Finally, using a one-way ANOVA, the QDIS-III-R and DIS-IV were compared on the total number of disorders diagnosed for each person. The two interviews did not differ in average number of disorders diagnosed ($F(1) = 1.26, p = .26$). Therefore it was deemed appropriate to pool across both instruments in testing the hypotheses.

DIS Reliability. In initial field studies done with the DIS-IV, inter-rater reliability (Kappa) was .80 for Alcohol Dependence, .77 for Drug dependence, .63 for Mania, .63 for Antisocial personality disorder, and .63 for depression (Robins et al., 1981) The QDIS has been shown retain equally reliability as the early paper versions of the full DIS as well as having acceptable reliability to the full DIS itself (.69) (Bucholz et. al., 1996). The newer computer version of the DIS also shows acceptable reliability with previous paper versions of the DIS (.64) (Erdman et al., 1992). Overall, the DIS shows high reliability across administrations. It is widely used and accepted as a valid research and diagnostic tool designed for community samples.

Data Analysis

In order to assess the differences between the groups of children (those with ADHD (any type) and control children), a series of 2x2 chi-squares were performed. First child groups were compared in terms of presence of parent ADHD (any type). For power analysis purposes, population affect size estimates were made using rates of ADHD found in Table 2. The difference in rates of ADHD (any type) between parents of children with ADHD (any type) and parents of control children was estimated to be 14% (average in Table 2). For a control sample size of $n = 86$ parents (mothers and fathers

combined) and a ADHD (both subtypes) sample size of 159 parents, power was equal to .96 to detect a 14% difference between groups using a chi-square with 2-tailed alpha test (.99 for a 1-tailed alpha). Mothers and fathers of the two child groups were then analyzed separately. Using the same method for estimating population prevalence, a 23 percentage-points difference was assumed between fathers of children with ADHD and fathers of control children in the rate of ADHD (any type). For the available fathers, power was equal to .74 to detect this effect for 2-tailed test of significance (.90 for 1-tailed alpha). Average difference between the mothers of the two child groups was estimated to be 16 points, which yielded a power equal to .80 (2-tailed) or .88 (1-tailed alpha) for the available *n* of mothers for the two groups.

Additional 2x3 chi-squares were used to assess the differences between the parents of the child DSM-IV ADHD subtype groups. Because this was the first time ADHD subtypes as defined by the DSM-IV were examined in parents, there were few prior data on which to base an estimate of effect size in the population. However if the subtypes are etiologically distinct disorders, as proposed by Milich, Balentine, and Lynam (2001), differences comparable to those between ADHD (any type) and control groups might be expected and population estimates from Table 2 were again used. As before the differences between all parents (mothers and fathers) were examined. Sample sizes for parents of children with ADHD-combined was $n = 106$ and for parents of children with ADHD-inattentive is $n = 51$ (mothers and fathers combined). An estimate of a 14-point difference in prevalence between parents of children with ADHD-Inattentive type and parents of children with ADHD-Combined type would yield power equal to .83 for a 2-tailed alpha test (.95 for 1-tailed alpha). The power to detect a 14-

point difference between parents of control children and parents of children with ADHD-Combined type equals .93 for 2-tailed alpha (.99 for 1-tailed alpha). To detect the same difference between parents of control children and parents of children with ADHD-Inattentive type power for a 2-tailed test of significance equals .80 (.94 for 1-tailed alpha).

Mothers and fathers of the three child groups were again analyzed separately using the population estimates as described above. Power to detect a 16-point difference between mothers of children with ADHD-Combined type (n = 61) and mothers of children with ADHD-Inattentive type (n = 28) was .58 for 2-tailed alpha (.70 1-tailed alpha). A 2-tailed test would have a power of .55 (.64 for 1-tailed) to detect a 16-point difference between mothers of control children and mothers of children with ADHD-Inattentive type; power equals .73 (.83 for 1-tailed alpha) to detect the same difference between mothers of controls and mothers of children with ADHD-Combined type.

With an average estimated difference of 23 percentage points between fathers of children with ADHD-Inattentive type and fathers of children with ADHD-Combined type, power = .62 (2-tailed alpha) to detected a 23-point difference (.74 for 1-tailed alpha). Power = .75 (.84 1-tailed alpha) to detect the same difference between fathers of controls and fathers of children with ADHD-Inattentive type. Power = .75 (.84 1-tailed alpha) to detect a 23-point difference between fathers of controls and fathers of children with ADHD-Combined type. However it is recognized that the power was more limited to detect potentially important smaller differences between the subtypes; this is considered later in data interpretation.

Results

Participants

Demographic information for children and parents are included in Table 4. A total of 144 children were included in the study. The two groups of children, control children (n = 49) and children with ADHD (n = 95), did not significantly differ in ethnicity or in IQ. Despite efforts to include an equal amount of girls with ADHD, more girls were included in the control group (40.8%) than in the ADHD group (26.3%). Also control children were slightly older (mean age = 9.78 yrs.) than children with ADHD (mean age = 9.42 yrs.); however, this difference was shy of significance. Differences in behavior ratings were consistent with diagnostic groupings; children with ADHD were rated as having a greater amount of inattentive and hyperactive symptoms compared with control children.

Mothers of the control children (n = 49) and mothers of children with ADHD (any type) (n = 90) were similar in age, ethnicity, IQ, educational level, and full time employment status (measured by number of months employed full time in the last 12 months). The fathers of control children (n = 37) and fathers of children with ADHD (n = 69) also did not differ in respect to the same variables. However, mothers and fathers of control children were more likely to be married than the mothers and fathers of children with ADHD.

Hypothesis 1: Comparing ADHD (any type) with controls.

To address the first hypothesis, children were placed into two groups: [a] children with ADHD (any subtype) and [b] control children.

(I-A) Parent ADHD (any type). The two groups were first compared on the frequency of parent ADHD diagnosis (any subtype). Table 5 lists all results. Significance was determined if groups differed at $p<.05$ two-tailed, however because of directional hypotheses, 1-tailed significance is also reported. Parents of children with ADHD were more likely to have had ADHD (21.4%) compared with the parents of control children (4.7%). When examined separately, both mothers (20.0%) and fathers (23.2%) of children with ADHD were more likely to have had ADHD themselves compared with the mothers (4.1%) or fathers (5.4%) of controls. Thus, Hypothesis I-A was supported.

In order to look at the effects of gender in separate analyses, the parents of girls and parents of boys were examined independently (Table 6 list the results separately for girls and boys). The parents of girls with ADHD had a higher rate of ADHD themselves (22.5%) when compared with parents of control girls (6.1%). The same was true for parents of boys (21.0% vs. 3.8%). Mothers of girls with ADHD were also more likely to have had ADHD themselves (20.8%) when compared with the mothers of control girls (0%). However fathers of girls with ADHD (25.0%) were not more likely to have ADHD themselves compared with the fathers of control girls (15.4%). The apparently high prevalence may have been an artifact of the low n (total affected fathers, $n = 2$). The opposite was true of mothers and fathers of boys. Fathers of boys with ADHD (22.6%) were more likely to have ADHD themselves compared with the fathers of control boys (0%). There is not a significant difference between the prevalence of ADHD in mothers of boys with ADHD (19.7%) and mothers of control boys (6.9%).

(I-B) Parent other (non-ADHD) disorders. The two child groups were also compared on the prevalence of parental non-ADHD psychiatric diagnoses (Generalized

Anxiety Disorder, Major Depression Disorder, Dysthymia, Bipolar Disorder, Antisocial Personality Disorder, Alcohol Abuse, and Drug Abuse) using a 2x3 chi-square as follows. Parents were separated into three groups, a) no diagnosis, b) only one other non-ADHD diagnosis, or c) two or more other non-ADHD diagnoses. Parent ADHD was excluded from analysis. Parents of children with ADHD did not differ in rates of 0, 1, or 2 or more diagnoses when compared with the parents of Control children. Differences in the total number of disorders were assessed using a one-way ANOVA. Parents of children with ADHD had a higher number of disorders compared with parents of control children, however results were short of significance ($F(1) = 3.15$, two tailed $p = .077$).

When mothers and fathers were examined separately, the number of mothers of controls and mothers of ADHD children with only one disorder did not differ. However more mothers of children with ADHD had 2 or more diagnoses than mothers of control children. Mothers of children with ADHD also had a greater number of disorders than mothers of control children ($F(1) = 4.99$, $p = .027$). Fathers of controls and fathers of ADHD children did not differ in the chance of having 1 or 2 or more other disorders. Fathers of the two child groups also did not differ in total number of disorders ($F(1) = .18$, $p = .672$). Thus overall, hypothesis I-B was only partially supported, the only differences found were in rates of maternal non-ADHD disorder.

No significant differences were found between the parents of girls or boys with ADHD compared with the parents of control girls or boys for rates of non-ADHD disorders. (Results found in Table 6) When mothers and fathers were examined separately, mothers of girls with ADHD were not more likely than control mothers to have 1 disorder. However, mothers of ADHD girls were more likely to have 2 or more

disorders compared with the mothers of control girls. Mothers of control girls were more likely have 1 disorder compared to more than 2 disorders, whereas mothers of girls with ADHD were more likely to have 2 or more disorders compared to only one disorder ($\chi^2(1)= 4.219, p=. 040$). Fathers of girls with ADHD were more likely to have one disorder compared to fathers of control girls. They were not more likely to have two or more disorders compared with fathers of control girls. There were no differences in rates of non-ADHD disorder between the mothers and fathers of boys with ADHD compared with the mothers and fathers of control boys.

For additional analyses, the two groups of children were also compared on rates of individual parental disorders. When examining all parents, parents of controls and parents of children with ADHD did not differ in rates of any disorder (GAD, MDD, BPD, Dysthymia, ODD/CD, ASPD, Drug dependence, and Alcohol dependence). Results are found in Table 7. Fathers of the two groups also did not differ in prevalence of any disorder. Mothers of children with ADHD were more likely to have GAD (19.0%) compared with mothers of controls (6.7%), however the difference did not reach significance at two-tailed significance.

Hypothesis 2: Comparing child comorbid groups.

To address the second hypothesis, children were placed into three groups: (a) children with ADHD (any subtype) alone (b) children with ADHD plus ODD or CD and (c) control children. The three child groups were first compared on the frequency of parent ADHD (any type) diagnoses. Secondly the child groups were compared on the frequency of parent ADHD subtypes. All comparisons were made using chi-squares;

Table 8 lists results of all 3-group chi-squares. If the omnibus chi-square was significant, follow up pair-wise chi-squares were performed (represented in Table 8 by superscripts).

(A) Parent ADHD (any type). Parents of both the ADHD - alone group ($\chi^2(1)= 8.339, p= .004$) and the ADHD + ODD/CD group ($\chi^2(1)= 11.681, p= .001$) were more likely to have had ADHD compared with parents of the control children. The two ADHD groups did not significantly differ from each other ($\chi^2(1)= .449, p= .503$). This pattern remained significant when mothers and fathers were analyzed separately. Mothers of children with ADHD alone ($\chi^2(1)= 4.029, p= .045$) and mothers of children with ADHD + ODD/CD ($\chi^2(1)= 7.270, p= .007$) were more likely to have had ADHD compared with the mothers of control children. However the mothers of the ADHD-alone and ADHD +ODD/CD groups did not significantly differ from each other ($\chi^2(1)= .633, p= .426$). Fathers of children with ADHD alone ($\chi^2(1)= 4.202, p= .040$) and fathers of ADHD + ODD/CD ($\chi^2(1)= 4.454, p= .035$) were also more likely to have had ADHD. Again, there were no differences between the fathers of the ADHD groups ($\chi^2(1)= .025, p= .874$). Thus hypothesis II-A was supported.

(B) Parent ADHD subtypes. When rates of parental ADHD-subtypes were compared across child comorbid groups, parents of children with ADHD-alone (12.6%) were more likely than parents of controls (3.7%) to have ADHD-Inattentive type ($\chi^2(1)= 4.899, p= .027$). Parents of children with ADHD + ODD/CD were not more likely to have ADHD-Inattentive type compared with the parents of controls ($\chi^2(1)= 1.46, p= .23$). The parents of the two child ADHD groups (ADHD alone and ADHD+ODD/CD) did not differ in rates of ADHD-Inattentive type ($\chi^2(1)= .816, p= .366$). When parents were examined separately, the three groups of mothers and the three groups of fathers did not

significantly differ in rates of ADHD-Inattentive type, perhaps due to a reduction in power.

When the child comorbid groups were compared on rates of parental ADHD-Combined subtype, both parents of children with ADHD –alone ($\chi^2(1)= .275, p= .039$) and parents of children with ADHD + ODD/CD ($\chi^2(1)= 11.951, p= .001$) were more likely to have had ADHD-Combined type compared to parents of controls. Compared with parents of children with ADHD –alone, the parents of ADHD + ODD/CD children had higher rates of ADHD –combined type although the difference was significant by 1-tailed test ($\chi^2(1)= 3.239, p= .036$). The same analyses were repeated for mothers and fathers separately.

Mothers of children with ADHD + ODD/CD had a higher rate of ADHD-Combined type than the mothers of control children ($\chi^2(1)= 5.297, p= .021$). This difference was also significant for the same two groups of fathers ($\chi^2(1)= 6.667, p= .010$). The mothers ($\chi^2(1)= 2.957, p= .086$) of the ADHD- alone group did not differ significantly from either the mothers of controls or from the mothers ($\chi^2(1)= .134, p= .714$) of the ADHD + ODD/CD group in rates of ADHD combined type. Rates of ADHD-combined type also did not differ between the fathers of children ADHD-alone and fathers of controls ($\chi^2(1)= 2.008, p= .156$) or between the fathers of children with ADHD alone and fathers of children with ADHD+ODD/CD ($\chi^2(1)= .794, p= .373$).

Because the pattern for parent ADHD-subtypes appeared to differ between the child ADHD-alone and child ADHD+ODD/CD groups, those were compared directly. Using 2-group chi-squares, the groups were tested for an interaction between parent ADHD subtype by child comorbid subtype. Parents of children with ADHD alone were

more likely to have ADHD-Inattentive type than ADHD-Combined type whereas parents of children with comorbid ODD/CD are more likely to have ADHD-Combined type than ADHD-Inattentive type ($\chi^2(1)= 4.14, p= .042$). This interaction was not significant when comparing the mothers ($\chi^2(1)= 1.167, p= .280$) or the fathers ($\chi^2(1)= 3.233, p= .072$) of the ADHD groups by a 2-tailed test, perhaps due to loss of power. Thus hypothesis II-B was not supported, and in fact there was a significant difference in rates of parent DSM-IV subtype between the child ADHD-alone and the child ADHD+ODD/CD groups.

(C) Parent other (non-ADHD) disorders. When comparing the rates of other non-ADHD parental (all parents, and mothers and fathers separately) diagnoses (0 disorders versus 1 disorder versus 2 or more disorders), no differences were found between the three groups of children. When comparing the total number of parental non-ADHD disorders, there were again no differences between child groups ($F(2) = 1.39, p= .250$). There were also no differences between the 3 groups of paternal non-ADHD disorders ($F(2) = .57, p = .566$). There was, however, a difference between child groups in rates of maternal disorder ($F(2) = 3.07, p = .050$). Mothers of children with ADHD+ODD/CD were more likely to have a greater number of diagnoses compared with mothers of controls ($p = .036$). There were no differences between mothers of children with ADHD-alone and mothers of controls ($p= .537$) or between mothers of children with ADHD-alone and mothers of children with ADHD+ODD/CD ($p = .340$). Thus, hypothesis II-C was partially supported.

Hypothesis 3: Comparing ADHD Subtypes.

To address the third hypothesis, all children were restratified into one of three groups: (a) ADHD combined subtype, (b) ADHD inattentive subtype, and (c) control

children. An omnibus chi-square compared the three child groups for frequency of each parent ADHD subtype.

(A) Parent ADHD (any type). First, rates of ADHD (any type) in the three groups of parents were compared. Parents of children with ADHD-Combined type were more likely to have ADHD themselves than parents of the controls ($\chi^2(1)= 14.23, p= .000$). Parents of children with ADHD-Inattentive type had higher rates of ADHD compared with parents of controls, however the difference was only significant by one-tailed test ($\chi^2(1)= 3.57, p= .03$). Parents of the two child ADHD groups did not significantly differ in rates of ADHD ($\chi^2(1)= 2.42, p= .120$). Both mothers ($\chi^2(1)= 7.784, p= .005$) and fathers ($\chi^2(1)= 6.483, p= .011$) of children with ADHD-Combined type were more likely to have ADHD themselves when compared to controls. Mothers of children with ADHD-Inattentive type did not significantly differ in rates of ADHD from mothers ($\chi^2(1)= 2.58, p= .108$) of controls or mothers ($\chi^2(1)= .893, p= .345$) of children with ADHD-Combined type. The rates of ADHD did not differ between fathers of children with ADHD-Inattentive type and fathers of controls ($\chi^2(1)= 1.08, p= .298$) or between the fathers of the two groups of children with ADHD (inattentive and combined) ($\chi^2(1)= 1.64, p= .200$).

The parents of boys and girls were then examined independently. Parents of girls with ADHD combined type were more likely to have ADHD themselves compared with parents of control girls ($\chi^2(1)= 3.90$, p= .048). There were no other differences between parents of the other groups of girls. Parents of boys with ADHD-Combined type were more likely to have ADHD compared with parents of control boys ($\chi^2(1)= 10.12$, p= .001). Parents of boys with ADHD-Combined type were also more likely to have ADHD compared with parents of boys with ADHD-Inattentive type ($\chi^2(1)= 3.04$, p= .04)

although the difference was only significant by a one-tailed test of significance. There were no difference between the parents of boys with ADHD-Inattentive type and parents of control boys ($\chi^2(1)= 1.22$, p= .270).

Mothers of girls with ADHD-Combined type were also more likely to have ADHD (any type) compared with mothers of control girls ($\chi^2(1)= 5.52$, p= .019). Mothers of girls with ADHD-Inattentive type (16.7%) were more likely to have ADHD compared with the mothers of control girls (0%) however it fell short of significance at a 2-tailed alpha level ($\chi^2(1)= 3.56$, p= .059) perhaps due to lack of power. There were no differences between the fathers of the three groups of girls.

This was not the case with the groups of boys. Fathers of boys with ADHD-Combined type were more likely to have ADHD themselves compared with the fathers of control boys ($\chi^2(1)= 7.76$, p= .005). Fathers of boys with ADHD-Combined type also had a somewhat higher prevalence of ADHD compared with the fathers of boys with ADHD-Inattentive type ($\chi^2(1)= 2.65$, p= .103) however the difference fell short of 2-tailed significance. There were no differences between rates of ADHD in fathers of boys with ADHD-Inattentive type and fathers of control boys ($\chi^2(1)= 1.64$, p= .200). Also, unlike the mothers of girls, there were no differences in the rate of ADHD in the mothers of the three groups of boys. Thus, support for hypothesis III-A was mixed.

(B) Parent ADHD subtypes. The 3 groups of children were also compared for rates of parental ADHD subtypes. Compared with parents of control children, parents of children with ADHD-Inattentive type ($\chi^2(1) = 4.40$, $p= .036$) and parents of children with ADHD-Combined type ($\chi^2(1) = 9.67$, $p= .002$) were more likely to have ADHD-Inattentive type themselves. However parents of the two groups of children with ADHD

did not differ from each other in rates of parent ADHD-Inattentive type. Mothers of children with ADHD-Combined type had higher rates of ADHD-Inattentive type compared with parents of control children ($\chi^2(1) = 4.155, p = .042$). Rates of ADHD-Inattentive type in mothers of children with ADHD-Inattentive type did not differ from mothers of controls ($\chi^2(1) = .24, p = .623$) or from mothers of children with ADHD-Combined type ($\chi^2(1) = 1.45, p = .793$). Rates of ADHD-Inattentive type in fathers of the three groups of children did not differ.

Rates of ADHD-Combined type in parents of children with ADHD-Inattentive type ($\chi^2(1) = 5.41, p = .020$) and parents of children with ADHD-Combined type ($\chi^2(1) = 9.67, p = .002$) were higher than in the parents of control children. The rates of ADHD-Combined type did not differ in the parents of the two different ADHD child groups. Mothers of children with ADHD-Inattentive type were more likely to have ADHD-Combined type than mothers of controls ($\chi^2(1) = 5.54, p = .019$). There was no difference in the rate of maternal ADHD-Combined type between mothers of controls and mothers with ADHD-Combined type ($\chi^2(1) = 2.91, p = .088$) or between the mothers of the two child ADHD groups ($\chi^2(1) = .677, p = .411$). However rates of ADHD-Combined type in fathers of children with ADHD-Combined type was higher than in fathers of controls ($\chi^2(1) = 6.76, p = .009$) and higher than in fathers of children with ADHD-Inattentive type ($\chi^2(1) = 3.96, p = .047$). Furthermore, fathers of children with ADHD-Inattentive type were more likely to have ADHD-Inattentive rather than ADHD-Combined type themselves, while fathers of children with ADHD-Combined type were more likely to have ADHD-Combined type rather than ADHD-Inattentive type ($\chi^2(1) = 3.82, p = .051$). This interaction was not significant when comparing all parents of the child ADHD

groups ($\chi^2(1) = .048$, $p = .827$) or the mothers of the child ADHD groups alone ($\chi^2(1) = 2.36$, $p = .124$).

When boys and girls were examined separately, an interesting pattern emerged. There was no significant difference between the 3 groups of girls on parental rates of ADHD-Inattentive type. However, parents of girls with ADHD-Inattentive type ($\chi^2(1) = 3.81$, $p = .051$) and parents of girls with ADHD-Combined type ($\chi^2(1) = 3.81$, $p = .051$) were more likely to have ADHD-Combined type than parents of control girls. There was no significant difference between parents of girls with ADHD-Inattentive type and parents of girls with ADHD-Combined type ($\chi^2(1) = 0$, $p=1$) in rates of ADHD-Combined type. Boys with ADHD-Combined type were more likely to have a parent with ADHD-Inattentive type ($\chi^2(1) = 4.35$, $p = .037$) and more likely to have a parent with ADHD-Combined type ($\chi^2(1) = 5.95$, $p = .015$) than control boys. Parents of boys with ADHD-Inattentive type did not differ from parents of control boys in rates of ADHD-Inattentive type ($\chi^2(1) = 1.17$, $p = .279$) or in rates of ADHD-Combined type. Also, the parents of boys with ADHD-Inattentive type and parents of boys with ADHD-Combined type did not differ in rates of ADHD-Inattentive type ($\chi^2(1) = .715$, $p = .398$) or in rates of ADHD-Combined type ($\chi^2(1) = 1.50$, $p = .221$).

Further analysis separating the mothers from fathers of boys and girls, uncovers even more remarkable results. The 3 groups of girls did not differ in rates of mother or fathers with ADHD-Inattentive type. Rates of parental ADHD-Combined type were higher in mothers of girls with ADHD-Inattentive type compared with mothers of control girls ($\chi^2(1) = 3.89$, $p = .049$). Rates of parental ADHD-Inattentive type did not differ between the mothers of girls with ADHD-Combined type and mothers of control girls

($\chi^2(1) = 2.07, p = .150$) or between the mothers of girls with ADHD-Combined type and mothers of girls with ADHD-Inattentive type ($\chi^2(1) = .95, p = .330$). There were no significant differences between the rates of ADHD-Combined type in the fathers of the three groups of girls (ADHD-Combined, ADHD-Inattentive, and control).

For boys, rates of maternal ADHD-Inattentive and ADHD-Combined type did not differ between the three groups. Also rates of paternal ADHD-Inattentive type did not differ between the 3 groups of boys. However, boys with ADHD-Combined type were more likely to have a father with combined type compared with control boys ($\chi^2(1) = 4.82, p = .027$). They were also more likely to have a father with ADHD-Combined type when compared with boys with ADHD-Inattentive type ($\chi^2(1) = 2.92, p = .044$), when using a one-tailed test of significance. There was no difference between boys with ADHD-Inattentive type (0%) and Control boys (0%) for rate of paternal ADHD-Combined type. Overall, hypothesis III-B was not supported.

(C) Parent other non-ADHD disorders. The 3 groups of children were compared on the presence of parent non-ADHD Disorder. The 3 child groups did not differ in total number of non-ADHD disorders in parents ($F(2) = 2.05; p = .131$) or in fathers ($F(2) = .17; p = .841$). However, mothers of children with ADHD-Combined type were more likely to have 2 or more disorders when compared with parents of control children ($\chi^2(1) = 5.48, p = .019$). Mothers of children with ADHD-Combined type were also more likely to have a greater total of disorders compared with mothers of controls ($p = .018$). Rates of maternal non-ADHD disorders did not differ between mothers of children with ADHD-Inattentive type and mothers of controls ($\chi^2(1) = .74, p = .390$) or between the mothers of

the two child ADHD groups ($\chi^2(1) = 1.31$, $p = .253$). Thus Hypothesis III-C was supported only when mothers were examined.

When looking at individual diagnoses in parents, the parents of three child groups only differed in rates of drug dependence. Parents of children with ADHD-Combined type had higher rates of drug dependence (12.8%) compared with parents of controls ($\chi^2(1) = 4.09$, $p = .043$). Rates of drug dependence did not differ in parents of controls and parents of children with ADHD-Inattentive type ($\chi^2(1) = .466$, $p = .495$) or between parents of children with ADHD-Inattentive type and parents of children with ADHD-Combined type ($\chi^2(1) = 1.16$, $p = .281$).

Discussion

Family prevalence studies provide a key step in considering possible developmental context, etiological pathways, genetic effects, and validity of nosological distinction of psychopathological syndromes. In the case of ADHD, a promising early literature has lacked completion in terms of DSM-IV subtypes. Existing family studies have for the most part overlooked the subtypes, failed to control for comorbidity, and all but ignored possible differences in gender effects. These and other problems have left the ADHD family literature with an incomplete understanding of parent psychopathology in relation to child ADHD as now defined. The present study was the first to examine the prevalence of parent psychopathology (both DSM-IV ADHD subtypes and other non-ADHD disorders) in the families of children with prospectively defined DSM-IV ADHD subtypes.

The clearest and most easily understood result of the present study was the replication of past findings that children with ADHD were more likely than control children to have parents who also have ADHD. Confirmation of this basic fact using DSM-IV is reassuring, but not surprising. Nearly all, prior family studies have shown an elevated rate of the then-current form of ADHD in relatives of children with ADHD. However, more interesting in the present study was the new data obtained concerning comorbidity, subtypes, and gender effects. Each of these findings is discussed in detail; contribution to understanding possible etiology and the value of current diagnostic classification of ADHD is also considered.

Child Comorbidity and Parent ADHD. When the presence of comorbid child disorders was controlled, the main finding remained significant: parents of children with

ADHD, regardless of comorbid behavior disorders in the children, were more likely to have ADHD themselves. Parents of children with ADHD + ODD/CD had similar rates of ADHD as the parents of children with ADHD alone, further supporting the specific validity of ADHD. For example past studies have asserted that it is comorbid disorders that "explained" the association between parental psychopathology and child ADHD (Lahey et al. 1988), however the results of the current study found that the presence of comorbid disorders did not explain the relationship between parent and child ADHD. To this extent, the results were consistent with the findings of Frick et al. (1991). Like their data, the co-occurrence of ADHD plus ODD/CD in children in our sample did not predict higher rates of ADHD in parents than did child ADHD alone.

Results also demonstrated a significant relationship between DSM-IV ADHD subtypes in parents and child comorbidity, which was not consistent with the findings of Frick et al. (1991). Parents of children with ADHD alone were more likely to have the Inattentive subtype, whereas the parents of children with ADHD+ODD/CD were more likely to have ADHD-Combined subtype. Although there is controversy about whether the DSM-IV subtypes are best viewed as degrees of severity of ADHD, if one was to think of ADHD-Combined type as a more severe manifestation of ADHD (individuals have both inattentive and hyperactive/impulsive symptoms) compared with ADHD-Inattentive type, then perhaps our data are consistent with prior studies. Although the presence of child ODD/CD alone did not account for the presence of parental ADHD, it suggests that the child ADHD+ODD/CD group could demonstrate a more severe disorder than ADHD alone. The former is more likely to be correlated with more severe parental

psychopathology. This study also looked at non-ADHD disorders in parents in order to explore this further.

Non-ADHD Disorders in Parents. Past research was not replicated when examining rates of non-ADHD disorders in all parents of children with ADHD. When all parents of children with ADHD were examined, they did not have higher rates of non-ADHD disorders when compared with control children, nor were any specific non-ADHD disorders in parents related to ADHD in children. Also striking was that the number of non-ADHD disorders in parents was unrelated to child comorbidity status. This result is contrary to findings of either Lahey et al. (1988) or Frick et al. (1991) using earlier definitions of ADHD in samples consisting only of boys. Both studies found that a diagnosis of child ADHD plus CD predicted higher rates of non-ADHD disorders in parents when compared with parents of children with ADHD alone. Several explanations for the contrasting results are possible.

First of all, unlike prior studies, here mothers and fathers were examined separately. There were no significant differences in fathers' rates of non-ADHD disorder between fathers of children with ADHD and fathers of controls. However, mothers of children with ADHD, regardless of comorbidity, were more likely to have non-ADHD disorders than mothers of control children, replicating past studies. Unique to our study, non-ADHD parental diagnoses were examined in relation to DSM-IV subtypes. Mothers of children with ADHD-Combined type had a greater number of disorders than mothers of children with ADHD-Inattentive type and mothers of control children. Specifically mothers of children with ADHD-Combined type were more likely to have a diagnosis of Major depression and drug dependence.

The current finding that only mother's disorders were related to child ADHD could be merely a result of the different diagnostic procedures. In both Lahey et al. (1988) and Frick et al. (1991), parental disorders were assessed by asking the mother of the child to rate themselves and the child's father, whereas the present study directly interviewed both parents. Perhaps mothers had over-reported psychiatric symptoms in the child's father in previous studies, leading to over-estimating the relationship between paternal pathology and child ADHD. On the other hand we may have underestimated. Because a significant number of fathers did not participate, it could be that the fathers who were unavailable for the study had more disorders than the fathers that were still in the family. Thus the current study could be under-representing the number of non-ADHD disorders in the fathers of ADHD children. In addition, both of the aforementioned studies used clinic recruited samples in which child disorder and familial correlates could be more severe than would be found in the largely community recruited samples used here, although many of the community recruited children in our study had previously obtained clinical services.

However, non methodological interpretations warrant consideration. The higher rates of non-ADHD disorders in mothers of children with ADHD-Combined subtype could be interpreted in two contrasting ways. It could mean [1] findings could lend support the hypothesis that ADHD combined type is merely a more severe manifestation of ADHD or [2] results could also be interpreted as ADHD-Combined type having distinct parental correlates compared with ADHD-Inattentive type, thereby could validate the distinction between the subtypes. Either hypothesis, however, does suggest a possible

etiology, in that maternal non-ADHD disorder contributes to either the severity or the manifestation of ADHD.

Maternal psychopathology has been correlated with a number of psychological and behavioral outcomes in children (Frick, 1994). For example it has been suggested that maternal psychopathology may degrade parenting ability, disrupt the marital relationships, and even effect work performance and result in lower socioeconomic status. Therefore, it is not entirely surprising that maternal pathology might also contribute to the severity of behavior problems in children. If this were the case, it would be expected that more severe maternal psychopathology would result in more severe and a greater number of ADHD symptoms. Conversely, if maternal disorder is correlated with the manifestation of ADHD beyond the severity and/or number of symptoms of the subtypes, it could suggest that maternal psychopathology contributes unique risk factors for the development of ADHD-combined type independent of the risks for the Inattentive subtype and lend support to the distinction between the subtypes. However, in order to further determine the nature of familial transmission of ADHD, it is important to clarify the relationship between parental DSM-IV ADHD subtypes and DSM-IV subtypes in children.

Parent and Child DSM-IV ADHD Subtypes. When all parents were examined together, DSM-IV subtypes in parents did not directly correspond to the subtype diagnosed in their child. As was found in previous studies of DSM-IV ADHD, subtypes were not found to "breed true" per se. For instance, Smalley et al. (2000) concluded that inattention and hyperactivity seem to share the same familial underpinning, and do not have unique effects. Alternatively, it has been suggested that subtypes based on

symptom expression have been shown to cluster in families (Todd, et al., 2001). This suggests that differential expressions of ADHD could be transmitted in families and exploring possible mechanisms for symptom transmission becomes essential. One area that has been neglected in past research has been gender.

In prior studies mothers and fathers were not directly interviewed, instead all relatives were assessed using a questionnaire completed by the mother of the child. Further, neither of the previous studies reported results for mothers and fathers separately or for child boys and girls separately. Contrary to past studies, the present study assessed parental ADHD using a structured clinical interview with each parent and directly assessed gender effects for family transmission. When considering mothers and fathers, and boys and girls separately, gender differently predicted parental rates of ADHD (any type) in boys and girls with ADHD. Specifically girls with ADHD had mothers with higher rates of ADHD, whereas boys with ADHD have fathers with higher rates of ADHD themselves.

Gender and DSM-IV ADHD Subtypes. When boys and girls or mothers and fathers were combined, the subtypes did not appear to be distinct disorders. However, as was true with ADHD (any type), subtype transmission appeared to be gender specific. In general, there was a relationship between child subtype and ADHD status in the parents. Girls with ADHD combined type compared to control girls were more likely to have mothers with ADHD (any type), there was no significant relationship between girls ADHD subtypes and father ADHD status. The opposite was true for boys. Boys with ADHD-Inattentive type and boys with ADHD-Combined type were both more likely to have a father with ADHD compared with fathers of control boys. Also, boys with

combined type were more likely to have a father with ADHD, when compared with boys with ADHD-Inattentive type. There was no significant relationship between a diagnosis of ADHD subtype in boys and mother ADHD status.

Further, fathers of boys with ADHD-Combined type were more likely to have ADHD-Combined type versus both the fathers of controls and fathers of boys with ADHD-Inattentive type. Fathers of boys with ADHD-Inattentive type were more likely to have Inattentive type themselves versus fathers of the other two child groups. This was not true for mothers of boys. Thus, subtypes did appear to be validated with boys and fathers, but not girls and mothers.

Results suggested that the ADHD subtypes directly transfer only within boys. This was clear when looking at boys and fathers; results for girls and their mothers were less clear. Perhaps one reason for this was the low number of girls with ADHD-Combined type (n = 12). However, beyond the sample's limitations, the results could also hint at important gender differences in the manifestation of ADHD. Previous studies have found that girls with ADHD have a greater chance of having a parent with a diagnosis of ADHD (Smalley, et al., 2000) and other studies have suggested that girls with ADHD require a greater loading of familial influences to develop ADHD. Thus it may be that symptoms of ADHD in boys are present with genetic loading of ADHD, whereas for girls, genetic interaction with environmental factors is more crucial for symptom expression. Therefore to examine familial transmission in families of girls with ADHD would require including measures of environmental risk that may interact with symptom expression.

Additionally, differential familial transmission for boys vs. girls also may be a result of the lack of research with girls with ADHD. DSM-IV field trials were done with primarily male samples. It could be that the subtype differentiation could be more valid with boys than with girls. This could in part explain why gender-specific transmission was found with mothers and girls for ADHD-any type, but not for ADHD subtypes. As found in Todd et al., it may be beneficial to organize symptoms in alternative ways, especially when examining familial transmission in girls.

Overall, this gender-specific transmission pattern requires replication, but is provocative. Unlike past studies that have merely found correlations between parent and child ADHD, gender-specific transmission hints at possible etiological pathways. For example it could be consistent with some emerging results suggesting that molecular genetic correlates of ADHD are better understood when gender-specific transmission is considered (Fisher et al., 2002). Also, assuming non-sex linked genetic markers, differential familial transmission could be a result of social modeling; perhaps the girls are more likely to model the behavior after their mothers whereas the boys are more likely to model their behaviors after their fathers. In addition to suggesting etiological mechanisms, gender-specific familial transmission could help understand past inconsistencies in the literature. However, it is important to determine potential confounding factors in the pattern of familial transmission.

Limitations. Although one of the strengths of the present study was the use of a structured interview with each of the parents, interviews were designed to be administered using lay interviewers. Although this interview is appropriate and acceptable for the community-recruited sample we studied, a clinician-guided interview,

such as the SCID-IV-TR (First, Spitzer, Gibbon, & Williams, 2001) might provide more sensitive and valid diagnostic assignments and could alter some findings if ADHD was over or under diagnosed in the present study. Parent ADHD also relied entirely on self report; it would be ideal to have observer and childhood observer ratings of parents own behaviors to assess their ADHD status.

Another possible limitation to results was the use of the QDIS-III-R for a subset of parents. The QDIS-III-R, based on DSM-III-R criteria, was used to diagnose non-ADHD disorders in some parents who did not complete the DIS-IV. Although agreement between the two versions demonstrated reasonable agreement to warrant pooling the data for this study, the inconsistency between diagnostic interviews could have influenced interpretation of associations between child subtypes and parental non-ADHD disorders in ways that are indeterminate.

Finally, although a strength of the present study was that both boys and girls were included, when groups were stratified by both parent and child sex and ADHD subtype, the number of girls in some cells was very small, especially in the ADHD combined type group. As previously mentioned, unclear gender subtype validity with the parents of girls may have been a result of this unequal distribution between subtypes.

Conclusions. Overall, this study made several significant contributions to the ADHD family studies literature. Overall, this was the first study to do a familial transmission study of prospectively defined DSM-IV ADHD in a community sample of children. This study replicated and extended previous studies, in that this study examined mothers and fathers separately and found gender specific ADHD transmission in families. In addition, this study also corrected for short-comings of previous family studies of

ADHD. The current study used boys and girls diagnosed with ADHD subtypes while controlling for comorbid behavior disorders in children. Results showed that parental ADHD is related to ADHD in children, regardless of child comorbidity. It also revealed intriguing hints of gender specific transmission of ADHD in families. Therefore this study underscores the importance of future research that explores in more depth gender based theories of family effects on ADHD symptoms in children. Overall, specific patterns of ADHD subtype transmission within families raises further implications for both genetic and psychosocial transmission processes related to gender and ADHD subtypes.

[1] Although there are 3 ADHD subtypes as defined in the DSM-IV, recent literature has hypothesized that the hyperactive subtype is a precursor to the combined subtype (Hard, et al., 1994). By the time children reach school age, there is a relatively low prevalence of the hyperactive type. For these reasons, only two DSM-IV subtypes (Inattentive and Combined) will be considered here and in the final analyses.

[2] The use of 5 symptoms, rather than 6, has been shown to be acceptable to use with adult populations (Barkley, 1998).

Appendix A

Different models and criteria have been proposed for establishing the validity of a medical and psychiatric illness (e.g. Cronbach & Meehl, 1955; Feighner et al., 1972; Cantwell & Baker, 1988). One approach to the validation of psychiatric disorders arises from the medical model tradition in classification (Feighner et al., 1972). This approach is often traced to Emil Kraeplin's classic observations of schizophrenia. Early in the 20th century Kraeplin advocated that mental disorders be identified through the grouping of people with similar observable behaviors and symptoms. Once a clinical description of the disorders was established, he believed that the cause of the disorder could be gleaned from the study of this group of people with the particular syndrome. Kraeplin, following the medical tradition, believed the cause might be traced to a single gene or pathogen. Although that approach was out of favor for much of the next half century, it was renewed with the advent of the contemporary DSM approach. Its renewal was directed in part when Feighner et al. (1972) published an article that updated the medical model approach to include five phases of validating diagnostic categories: clinical description (observable behaviors, symptoms and other associated factors), laboratory studies, the addition of criteria excluding overlap of other syndromes, follow-up studies of syndrome course, and family prevalence studies. That article set the stage for the DSM-III, in which criteria for diagnoses were revised from the purely theory-based descriptions to the directly observable symptom descriptions that are found in the DSM today. The medical model assumes that once a homogenous group of people with the same syndrome is isolated, the cause will be more easily located; this cause was believed by some to be biogenetic and found in one gene or virus/bacteria, such as true with most physical

disorders. However most contemporary scientists have discarded this metabolic view of psychopathology. It is believed that there are several different contributing etiological factors (Depue & Monroe, 1991). Nevertheless family prevalence studies may provide clues as to whether these etiological agents are present in systematic ways within families.

Appendix B
Comparing the QDIS to the DIS-IV for Differences in Disorder Prevalence

Parent Disorder	% Parents with disorder for Q-DIS	% Parents with disorder for DIS-IV	Chi-Square	*p*
Generalized Anxiety Disorder (GAD)	10.3%	11.4%	$\chi^2(1) = .04$	.843
Dysthymia	0%	1.7%	$\chi^2(1) = .53$	.469
Drug Dependence	5.1%	8.9%	$\chi^2(1) = .59$	.441
Anti-Social Personality Disorder (ASPD)	10.3%	17.1%	$\chi^2(1) = .99$	.319
Depression	23.1%	27.6%	$\chi^2(1) = .33$	.565
Alcohol Dependence	15.4%	11.1%	$\chi^2(1) = .55$	.457
Any disorder	40%	47.8%	$\chi^2(1) = .80$	.370
Total number of disorders*	Ave = .63 (SD=.90)	Ave = .83 (1.07)	F = 1.26	.262

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Table 1
Family Psychiatric Studies of ADHD Since 1970

Study	Child disorder	Diagnostic Tool	N	% Male	Sample source *	Parent Interview	Comorbid disorders isolated	Subtype isolated	Principle Findings
1.Morrison & Stewart (1971)	Hyper-active Child Syndrome	Out-Patient diagnosis	100	96%	1	Unstructured	No	No	Hyperactivity may be related to parental alcoholism, hysteria, and sociopathy
2. Cantwell (1972)	Hyper-active Child Syndrome	Parent report	100	100%	1	Unstructured	No	No	Parents of children with hyperactivity were more likely to have hyperactive symptoms
3. Stewart et al. (1980)	Hyper-activity	In-patient diagnosis	126	100%	1	Clinician administered	No	No	Child aggression, not ADHD, was associated with psychological disorders in parents
4. Lahey et al. (1988)	ADHD & CD	DSM III-R	86	72%	1	SADS	Yes	No	Children with ADHD plus CD were more likely to have an antisocial parent than children with ADHD or CD alone.
5. Frick et al. (1991)	ADHD & CD	DSM III-R	177	100%	1	Questionnaire	Yes	No	Children with ADHD, CD, or ADHD w/CD were more likely to have parents with the same disorder as diagnosed in the children.
6. Biederman et al. (1995)	ADHD	DSM III-R	260	100%	1	SADS	No	No	The number or maternal diagnoses was greater in children with ADHD.

7. Kuhne, Schachar, & Tannock (1997)	ADHD, ODD, & CD	DSM III-R	91	78%	1	SCL-R-90	Yes	No	Mothers of children with ADHD plus CD reported having significantly more psychological problems than ADHD alone.
8. Faraone et al. (1997)	ADHD, ODD, & CD	DSM III-R	260	100%	1	SCID	Yes	No	Families of children with ADHD plus CD have significantly higher rates of both disorders than ADHD or CD alone.
9. Nigg & Hinshaw (1998)	ADHD, ODD, & CD	DSM III-R & DSM-IV	142	100%	2	WURS & DIS-3-R	Yes	No	Mothers of ADHD children had higher rates of depressions; fathers had more ADHD. Mothers of ADHD+CD were more likely to have ADHD; fathers: anxiety, cocaine dependence, and ASPD.
10. Faraone, Biederman, & Friedman (2000a)	ADHD	DSM-IV Approx-imated	260	100%	1	SCID-III-R	No	Yes	Relatives of ADHD children were more likely to have ADHD, but it was not correlated with same subtype as diagnosed in children.
11. Faraone et al. (2000b)	ADHD	DSM-IV Approx-imated	262	0%	1	SCID-III-R	No	Yes	Relatives of girls with ADHD have higher rates of ADHD themselves, but it is not related to subtype as diagnosed in girls.

* 1= Clinic recruited
2= Community recruited

Table 2
Prevalence of ADHD in Parents and Relatives of Controls compared to Parents of Children with ADHD, with ODD/CD.

Study	Child classification								Significant P < .05	
	Control [a]		ADHD [b]		ADHD plus CD [c]		Rates of ADHD in Parents of ADHD:Control			
	Fathers	Mothers	Fathers	Mothers	Fathers	Mothers	Fathers	Mothers	Fathers	Mothers
Cantwell (1972)	2%	0%	16%	4%	-	-	8:1	4:0	b>a	NS
Frick et al. (1991)	22%	11%	40%	48%	48%	36%	1.8:1	4.4:1	b,c> a,d	b,c> a,d
Nigg & Hinshaw (1998)	0%	4%	41%	7%	13%	13%	41:1	1.75:1	b>a, c	NS
Faraone et al. (1997) *	All Relatives	Parents	All Relatives	Parents	All Relatives	Parents	All Relatives	Parents	All Relatives	Parents
	5%	2%	15%	9%	29%	23%	3:1	4.5:1	c>b>a	c>a
Faraone et al. (2000) **	All Relatives		C[+]	H-I[+]	I[+]					
	6%		24%	18%	19%	-	-	3.4:1		C, H-I, I[+] > a

* Faraone et al. (1997) did not examine mothers and fathers independently. They also included all relatives of the probands in the analyses.

**Faraone et al (2000) included all relatives in a single analysis.

++ C = Combined Subtype; H-I = Hyperactive Impulsive Subtype; I = Inattentive Subtype.

Table 3
Rates of Psychiatric Disorders in Parents of Controls compared to Parents of Children with ADHD and Parents of Children with ADHD+CD

Parent Disorder	Study	Control		ADHD		ADHD+CD	
		Father	Mother	Father	Mother	Father	Mother
Depression	4.	16.7	16.7*	11.1	22.2*	4.8	30.43*
	9.	9	18*	15	25*	11	39*
Conduct Disorder	8.	4*	4*	8*	8*	17*	17*
Antisocial Personality Disorder	1.+	0	0	8.5	0	-	-
	2.+	2	0	8*	0	-	-
	4.	16.7	0	5.6	0	52.4*	8.7*
	8.	6*	6*	9*	9*	21*	21*
	9.	14	5	4	0	6	4
Alcohol Abuse	1.	24.4	0	33.9	8.5	-	-
	2.	7	0	15	4*	-	-
	4.	10	0	5.6	5.6	28.6*	4.4
	9.	23	20	19	14	37	11
Drug Abuse	4.	6.7	0	0	5.6	23.8*	4.4
	9.	17*	5	0*	4	21*	9
Total Number of Disorders	1.	41.5	36.6	62.7	49.2	-	-
	2.	12	6	27*	18*	-	-

* = significant difference ($p<.05$) between groups

+ = In study 1. and 2. the sociopathy was the diagnosis used.

Table 4
Demographics

	Child Diagnosis (N = 144)						2-Group P-test[1]
	Control	ADHD(any)[2]	ADHD-alone	ADHD+ODD/CD	ADHD- I	ADHD- C	
Children							
N	49	95	48	46	28	66	-
Ave. age in years	9.78	9.42	9.35	9.47	9.8	9.26	.055
%boys	59.2	73.7	70.8	76.1	57.1	80.3	.019*
%white	65.6	88.5 .	86.4	89.7	80.0	91.7	.199
Ave. IQ	107.29	105.02	105.04	105.23	105.59	104.52	.582
Ave. # of Inattentive Sx.	.76	7.12	6.94	7.31	7.00	7.28	.000*
Ave. # of Hyperactive Sx.	.56	5.04	3.56	6.62	1.29	6.67	.000*
Mothers							
N	49	90	47	42	28	61	-
Ave. age	38.24	37.57	38.02	37.27	39.31	36.74	.569
%white	77.3	89.5	87.0	92.5	92.3	88.1	.428
Ave. IQ	109.09	110.55	111.80	109.80	113.23	109.30	.569
% married	93.9	71.6	71.7	70.7	76.9	68.9	.029*
Ave. # yrs of education	13.05	12.30	12.26	12.47	12.77	12.10	.650
Ave. # mo. Employed[3]	5.63	5.98	5.73	6.33	4.11	6.60	.529
Fathers							
N	37	69	40	29	23	45	-
Ave. age	40.44	40.18	40.03	40.39	40.8	39.53	.842
%white	71.0	89.5	85.3	95.7	91.7	91.7	.209
Ave. IQ	108.92	112.93	112.39	113.70	110.49	110.49	.208
% married	100.0	86.0	85.3	87.0	83.3	83.3	.029*
Ave. # yrs of education	13.57	10.59	9.90	12.25	10.76	10.76	.731
Ave. # mo. Employed [2]	9.89	7.47	6.50	10.0	7.36	7.36	.938

[1] Two group P-test compared Controls with ADHD-any type.
[2] Children with ADHD were divided into two different sets of groups for additional analysis depending on hypothesis: (1) divided children with ADHD based on comorbid diagnoses (2) divided children with ADHD based on DSM-IV subtypes. See text for further elaboration.
[3] Ave. # mo. Employed=the average number of months that the Parent was employed for full time (equal to or greater than 35 hours per week) in one year.

Table 5

Hypothesis 1: Comparing ADHD (any type) with Controls

Parent Diagnosis	Control Children ($n = 49$)	Children w/ADHD (any type) ($n = 95$)	2 (child) group Chi-Square [1]	*P* 2-tailed	*P* 1-tailed
ADHD-Any type					
All Parents:	($N = 86$)	($N = 159$)			
	4.7%	21.4%	$\chi^2(1) = 11.92$	.001*	.0005*
Mothers:	($N = 49$)	($N = 90$)			
	4.1%	20.0%	$\chi^2(1) = 6.53$	.011*	.005*
Fathers:	($N = 37$)	($N = 69$)			
	5.4%	23.2%	$\chi^2(1) = 2.10$	.020*	.010*
Non-ADHD disorders					
All Parents:	($N = 81$)	($N = 141$)			
0 disorders	60.5%	49.6%			
1disorder	22.2%	26.2%	$\chi^2(2) = 2.43$	.28	.14
2 disorders	17.3%	24.1%			
Mothers:	(N=45)	(N=84)			
0disorders	64.4%	52.4%			
1disorder	26.7%	23.8%	$\chi^2(2) = 4.36$	.11	.06
2 disorders	8.9%	23.8%			
Fathers:	($N = 36$)	($N = 57$)			
0 disorders	55.6%	45.6%			
1disorders	16.7%	29.8%	$\chi^2(2) = 2.07$	.36	.18
2 disorders	27.8%	24.6%			

[1] 2x3 Chi-Squares for Non-ADHD disorders compare 0 disorders, 1 disorder, or 2 or more disorders in parents by child group.

Table 6
Hypothesis 1: Comparing ADHD (any type) with Controls (Girls and Boys)

Parent Disorder	Girls		2-group (girls) Chi-Square		Boys		2-group (boys) Chi-Square	
	Control	ADHD	$\chi^2(1) =$	$p=$[1]	Control	ADHD	$\chi^2(1) =$	$p=$[1]
ADHD (any type)								
All Parents:	(*N* = 33)	(*N* =40)			(*N* =53)	(*N* =119)		
	6.1%	22.5%	3.82	.051	3.8%	21.0%	8.23	.004*
Mothers:	(*N* = 20)	(*N* = 24)			(*N* = 29)	(*N* = 66)		
	0%	20.8%	4.70	.030*	6.9%	19.7%	2.48	.115
Fathers:	(*N* = 13)	(*N* = 16)			(*N* = 24)	(*N* = 53)		
	15.4%	25.0%	.40	.525	0%	22.6%	6.44	.011*
			$\chi^2(2) =$[2]	$p=$[1]			$\chi^2(2) =$[2]	$p=$[1]
Non-ADHD disorders								
All Parents:	(*N* =33)	(*N* =36)			(*N* =48)	(*N* =105)		
0 disorders	51.5%	38.9%			66.7%	53.3%		
1disorder	21.2%	30.6%	1.25	.54	22.9%	24.8%	3.44	.18
2 disorders	27.3%	30.6%			10.4%	21.9%		
Mothers:	(*N* = 19)	(*N* = 22)			(*N* =26)	(*N* =62)		
0 disorders	63.2%	50.0%			65.4%	53.2%		
1disorder	31.6%	18:2%	4.75	.09	23.1%	25.8%	1.43	.49
2 disorders	5.3%	31.8%			11.5%	21.0%		
Fathers:	(*N* = 14)	(*N* = 14)			(*N* = 22)	(*N* = 43)		
0 disorders	35.7%	21.4%			68.2%	53.5%		
1disorder	7.1%	50.0%	6.33	.04*	22.7%	23.3%	2.12	.35
2 disorders	57.1%	28.6%			9.1%	23.3%		

[1] two-tailed.
[2] 2x3 Chi-Squares for Non-ADHD disorders compare 0 disorders, 1 disorder, or 2 or more disorders in parents by child group.

Table 7

Prevalence of Parent Non-ADHD Disorder in Children with ADHD compared with Controls

Parent diagnosis	Control Children	Children w/ADHD (any type)	$\chi^2(1) =$	Two-tailed *p*
All Parents –	(N =77)	(N =138)		
GAD	6.5%	13.8%	2.637	.104
MDD	23.4%	28.7%	.705	.401
Bipolar	7.8%	10.6%	.293	.589
Dysthymia	0%	2.3%	1.35	.245
ODD/CD	22.5%	27.6%	601	.438
Antisocial PD	5.2%	7.5%	1.19	.275
Drug dependence	3.9%	10.7%	2.98	.084
Alcohol dependence	10.5%	12.7%	.216	.642
Mothers –	(N = 45)	(N = 84)		
GAD	6.7%	19.0%	3.576	.059
MDD	22.2%	34.5%	2.102	.147
Bipolar	6.1%	13.6%	1.278	.258
Dysthymia	0%	3.8%	1.01	.193
ODD/CD	22.5%	30.4%	.962	.327
Antisocial PD	2.2%	7.3%	1.448	.229
Drug dependence	2.2%	10.0%	2.607	.106
Alcohol dependence	6.7%	11.0%	.634	.427
Fathers -	(N = 32)	(N = 54)		
GAD	6.3%	5.6%	.018	.894
MDD	25.0%	19.2%	.392	.531
Bipolar	11.1%	5.3%	.63	.427
Dysthymia	0%	0%	-	-
ODD/CD	23.3%	22.9%	.002	.966
Antisocial PD	9.4%	7.8%	.060	.807
Drug dependence	6.3%	11.8%	.407	.407
Alcohol dependence	16.1%	15.4%	.008	.928

Table 8

Hypothesis 2: Comparing ADHD with ADHD + ODD/CD

Parent Disorder	Control Children	Children w/ADHD (alone)	Children w/ADHD+ODD/CD	3 child-group Chi-Square[1]	*P* 2-tailed	*P* 1-tailed
ADHD						
All Parents –	(*N* =82)	(*N* =87)	(*N* =71)			
ADHD (any type)	4.9%[a]	19.5%[b]	23.9%[b]	$\chi^2(2) = 12.7$	.05*	.025*
ADHD-inattentive	3.7%[a]	12.6%[b]	7.0%	$\chi^2(2) = 4.91$	.086	.043*
ADHD-combined	0%[a]	4.6%[b]	12.7%[c]	$\chi^2(2) = 12.73$	.002*	.001*
Mothers –	(*N* =47)	(*N* =47)	(*N* =42)			
ADHD (any type)	4.3%[a]	17.0%[b]	23.8%[b]	$\chi^2(2) = 7.50$	.058	.029
ADHD-inattentive	2.1%	10.6%	7.1%	$\chi^2(2) = 2.90$	.235	.118
ADHD-combined	0%[a]	4.3%	9.5%[b]	$\chi^2(2) = 5.36$	.068	.034*
Fathers -	(*N* = 35)	(*N* = 40)	(*N* = 29)			
ADHD (any type)	5.7%[a]	22.5%[b]	23.8%[b]	$\chi^2(2) = 5.48$	.140	.070
ADHD-inattentive	5.7%	15.0%	6.9%	$\chi^2(2) = 2.28$	.321	.161
ADHD-combined	0%[a]	5.0%	17.2%[b]	$\chi^2(2) = 1.21$	.024*	.012*
Non-ADHD disorders						
All Parents:	(*N* =78)	(*N* = 76)	(*N* =64)			
0 disorders	61.5%	50.0%	50.0%			
1disorder	20.5%	25.0%	28.1%	$\chi^2(3) = 6.00$	.42	.21
2 disorders	17.9%	25.0%	21.9%			
Mothers:	(*N* =44)	(*N* =42)	(*N* =41)			
0 disorders	63.6%	57.1%	48.8%			
1disorder	27.3%	21.4%	26.8%	$\chi^2(3) = 5.09$	.53	.265
2 disorders	9.1%[a]	21.4%	24.4%[b]			
Fathers:	(*N* = 34)	(*N* = 34)	(*N* = 23)			
0 disorders	58.8%	41.2%	52.2%			
1disorder	11.8%	29.4%	30.4%	$\chi^2(3) = 11.08$	.08	.04*
2 disorders	29.4%	29.4%	17.4%			

If columns have different superscripts, they differed at p>.05.

[1] 3x3 Chi-Squares for Non-ADHD disorders compare 0 disorders, 1 disorder, or 2 or more disorders in parents by child group.

Table 9

Hypothesis 3: Comparing DSM-IV ADHD Subtypes

Parent disorders	Control Children	Children w/ADHD – Inattentive type	Children w/ ADHD – Combined type	3 (child) group Chi-Square[1]	*p* 2-tail	*p* 1-tail
ADHD						
All Parents –	(*N* =86)	(*N* =51)	(*N* = 106)			
ADHD (any type)	4.7%[a]	13.7%	24.5%[b]	$\chi^2(2) = 16.26$	.001*	.0005*
ADHD-inattentive	3.5%[a]	7.8%	10.4%[b]	$\chi^2(2) = 4.36$	.113	.062
ADHD-combined	0%[a]	5.9%[b]	9.4%[b]	$\chi^2(2) = 9.49$	.009*	.005*
Mothers –	(*N* = 49)	(*N* = 28)	(*N* = 61)			
ADHD (any type)	4.1%[a]	14.3%	23.0%[b]	$\chi^2(2) = 8.03$	.046*	.023*
ADHD-inattentive	2.0%[a]	10.7%	11.5%[b]	$\chi^2(2) = 4.96$	.084	.042*
ADHD-combined	0%[a]	3.6%	4.9%[b]	$\chi^2(2) = 4.93$	.085	.023*
Fathers -	(*N* = 37)	(*N* = 23)	(*N* = 45)			
ADHD (any type)	5.4%[a]	13.0%	23.0%[b]	$\chi^2(2) = 11.65$	.009*	.005*
ADHD-inattentive	5.4%	13.0%	8.9%	$\chi^2(2) = 1.15$	.563	.232
ADHD-combined	0%[a]	0%[a]	15.6%[b]	$\chi^2(2) = 10.39$	.006*	.003*
Non-ADHD disorders						
All Parents:	(*N* = 81)	(*N* = 45)	(*N* = 95)			
0 disorders	60.5%	53.3%	47.4%			
1disorder	22.2%	28.9%	25.3%	$\chi^2(3) = 5.27$	.51	.255
2 disorders	17.3%	17.8%	27.4%			
Mothers:	(*N* = 45)	(*N* = 25)	(*N* = 59)			
0 disorder	64.4%	60.0%	49.2%			
1disorder	26.7%	24.0%	23.7%	$\chi^2(3) = 5.89$	.21	.105
2 disorders	8.9%	16.0%	27.1%			
Fathers:	(*N* = 36)	(*N* = 20)	(*N* = 36)			
0 disorders	55.6%	45.0%	44.4%			
1disorder	16.7%	35.0%	27.8%	$\chi^2(3) = 3.86$	.70	.35
2 disorders	27.8%	20.0%	27.8%			

If columns have different superscripts, they differed at p>.05.

[1] 3x3 Chi-Squares for Non-ADHD disorders compare 0 disorders, 1 disorder, or 2 or more disorders in parents by child group.

Table 10
Hypothesis 3: Comparing ADHD Subtypes
(Girls and Boys)

Parent Disorder	Girls			3-group		Boys			3-group	
	Control	ADHD-I	ADHD-C	$\chi^2(2) =$	p^1	Control	ADHD-I	ADHD-C	$\chi^2(2) =$	p^1
ADHD										
All Parents –	($N = 30$)	($N = 20$)	($N = 20$)			($N = 53$)	($N = 31$)	($N = 86$)		
ADHD (any type)	6.1%[a]	20.0%	25.0%[b]	12.70	.05*	3.8%	9.7%	24.4%	11.71	.003*
ADHD-inattentive	6.1%	10.0%	10.0%	.67	.716	1.9%	6.5%	10.5%	4.56	.102
ADHD-combined	0%	10.0%	10.0%	3.89	.143	0%	3.2%	9.3%	6.90	.032*
Mothers –	($N = 20$)	($N = 12$)	($N = 12$)			($N = 29$)	($N = 15$)	($N = 49$)		
ADHD (any type)	0%[a]	16.7%	25.0%[b]	7.50	.058	6.9%	12.5%	22.4%	3.68	.299
ADHD-inattentive	0%	0.0%	8.3%	2.98	.226	0%	6.3%	12.2%	2.20	.332
ADHD-combined	0%	16.7%	8.3%	3.60	.165	0%	6.3%	4.1%	1.57	.456
Fathers -	($N = 13$)	($N = 8$)	($N = 8$)			($N = 24$)	($N = 15$)	($N = 37$)		
ADHD (any type)	15.4%	25.0%	25.0%	.40	.817	0%[a]	6.7%a	27.0%[b]	14.44	.002*
ADHD-inattentive	15.4%	12.5%	25.0%	.39	.821	0%	6.7%	8.1%	2.47	.291
ADHD-combined	0%	12.5%	0%	2.53	.282	0%[a]	0%	16.2%[b]	7.55	.023*

If columns have different superscripts, they differed at p>.05.
[1] two-tailed.

Table 11

Prevalence of Parent non-ADHD disorder by Child Subtype

	Control Children	Children w/ADHD-I	Children w/ ADHD-C	$\chi^2(2) =$	*P*
Parents –	(N = 77)	(N = 44)	(N = 94)		
GAD	6.5%	9.1%	16.0%	4.06	.131
MDD	23.4%	22.7%	31.5%	1.819	.391
Bipolar	7.8%	8.6%	11.6%	.535	.765
Dysthymia	0%	0%	9.4%	2.763	.251
ODD/CD	22.5%	21.1%	31.0%	2.786	.426
Antisocial PD	5.2%	4.5%	9.0%	1.338	.507
Drug dependence	3.9%[a]	6.7%	12.8%[b]	4.456	.108
Alcohol dependence	10.5%	15.6%	11.2%	.748	.680
Mothers –	(N = 45)	(N = 25)	(N = 59)		
GAD	6.7%	16.0%	20.3%	3.84	.147
MDD	22.2%[a]	16.0%[a]	42.4%[b]	7.89	.019*
Bipolar	6.1%	14.3%	13.3%	1.29	.524
Dysthymia	0%	0%	10.7%	1.62	.446
ODD/CD	22.0%	23.8%	33.3%	2.13	.546
Antisocial PD	2.2%	4.0%	8.8%	2.20	.332
Drug dependence	2.2%[a]	0%[a]	14.5%[b]	8.05	.018*
Alcohol dependence	6.7%	8.0%	12.3%	1.0	.606
Fathers -	(N = 32)	(N = 19)	(N = 35)		
GAD	6.3%	0%	8.6%	1.67	.434
MDD	25.0%	31.6%	12.1%	3.10	.212
Bipolar	11.1%	0%	8.3%	1.56	.459
Dysthymia	0%	0%	0%	-	-
ODD/CD	23.3%	17.6%	26.7%	.80	.849
Antisocial PD	9.4%	5.3%	9.4%	1.07	.585
Drug dependence	6.3%	15.0%	9.7%	1.08	.582
Alcohol dependence	16.1%	25.0%	9.4%	2.28	.319